November 2025 Volume 80 Supplement 2

# Thorax

AN INTERNATIONAL JOURNAL OF RESPIRATORY MEDICINE

## British Thoracic Society Winter Meeting 2025

QEII Centre
Broad Sanctuary
Westminster
London SW1P 3EE

26 to 28 November 2025

Programme and Abstracts

thorax.bmj.com

BMJ Group

TRIXEO AEROSPHERE®

(formoterol fumarate, glycopyrronium, and budesonide) Inhalation Aerosol

Intended for UK HCPs only

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TRIXEO AEROSPHERE: The **world's first next-generation propellant** containing inhaler[1]




With **a near-zero** global warming potential **(GWP)** that is **99.9% lower** than current propellants[2-4]

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*TRIXEO with the next-generation propellant will continue to phase in as previous pack stock levels deplete.

**TRIXEO** AEROSPHERE® (formoterol fumarate dihydrate/glycopyrronium/budesonide) 5/7.2/160 µg is indicated as a maintenance treatment in adult patients with moderate to severe chronic obstructive pulmonary disease (COPD) who are not adequately treated by a combination of an inhaled corticosteroid and a long-acting $\beta_2$-agonist or combination of a long-acting $\beta_2$-agonist and a long-acting muscarinic antagonist.[5] Limitation of use: This medicinal product is not indicated for the treatment of acute episodes of bronchospasm, i.e. as a rescue therapy[5]

**Click here for more information**

TRIXEO and AEROSPHERE are registered trademarks of the AstraZeneca group of companies.

**References: 1.** MHRA. MHRA approves world's first low-carbon version of COPD inhaler, Trixeo Aerosphere. May 2025. Available at: https://www.gov.uk/government/news/mhra-approves-worlds-first-low- carbon-version-of-copd-inhaler-trixeo-aerosphere [Accessed October 2025]; **2.** Smith C, et al. The Earth's energy budget, climate feedbacks and climate sensitivity supplemental material. In: Masson-Delmotte V, et al. Climate change 2021: the physical science basis. 2021; **3.** Hargreaves C, et al. Abstract presented at BTS 2022 (Abstract S60); **4.** Hargreaves C, et al. Oral presentation at BTS 2022 (Abstract S60) **5.** TRIXEO AEROSPHERE 5 micrograms/7.2 micrograms/160 micrograms pressurised inhalation, suspension. Summary of Product Characteristics.

**Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App store. Adverse events should also be reported to AstraZeneca by visiting https://contactazmedical.astrazeneca.com or by calling 0800 783 0033.**

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GB-71054 | October 2025

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PROGRAMME

AND

ABSTRACTS

Thorax

# British Thoracic Society
# **Winter Meeting 2025**

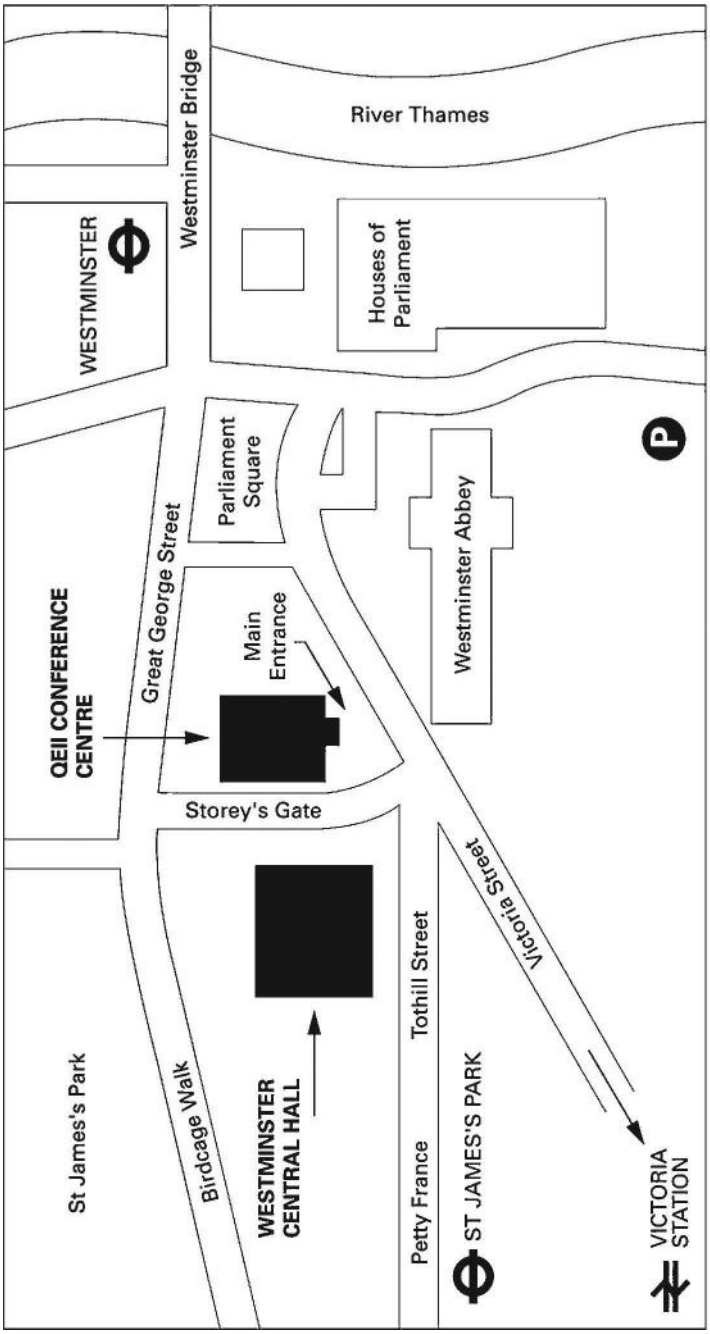
QEII Centre
Broad Sanctuary
Westminster
London SW1P 3EE

**Wednesday 26 to 28 Friday**
**November 2025**
**Programme and Abstracts**

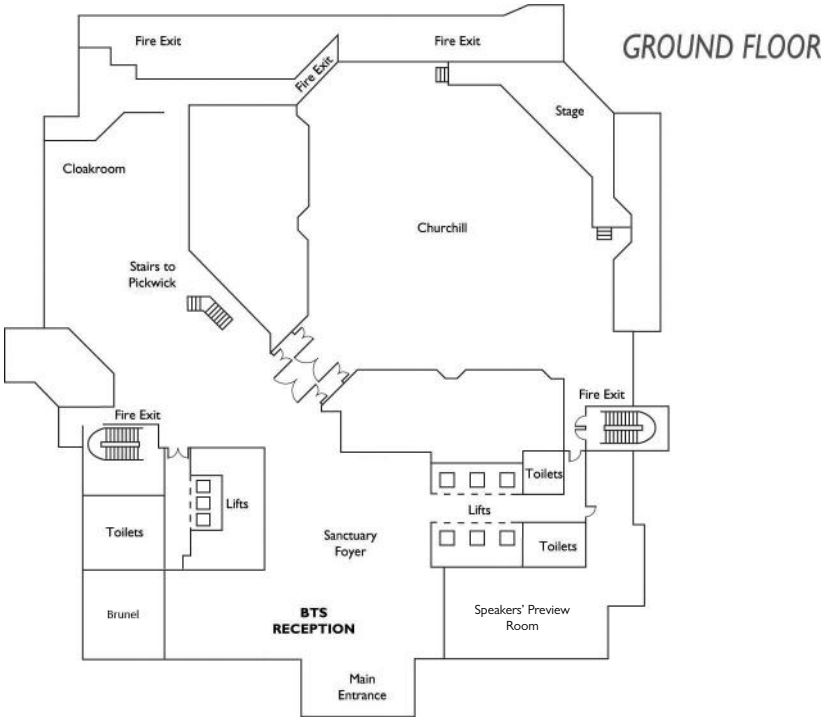
Approved by the Federation of the
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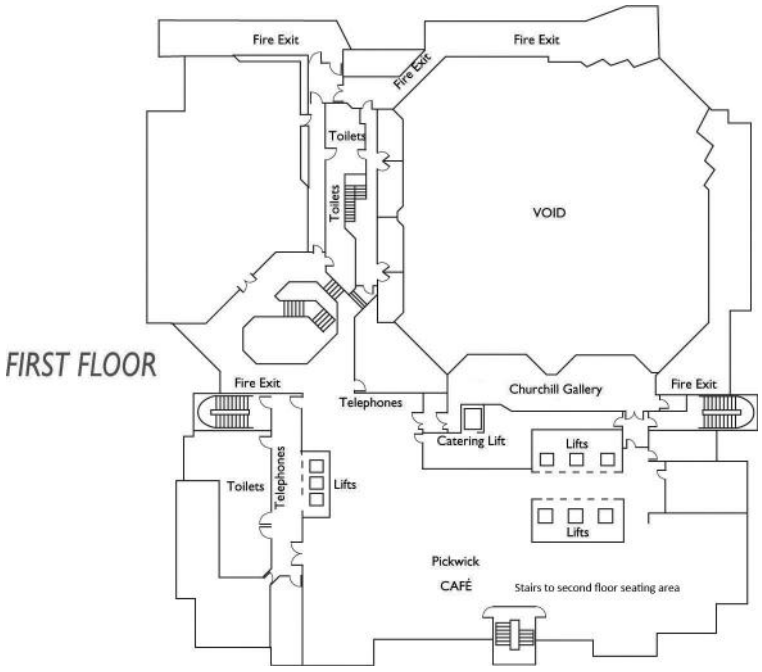
# Map to the QEII Centre

PowerPoint preview facilities will be available throughout the three days of the Meeting on the ground floor of the Centre. All presenters and chairs should report to the Speakers' section of the Registration Desks on arrival.



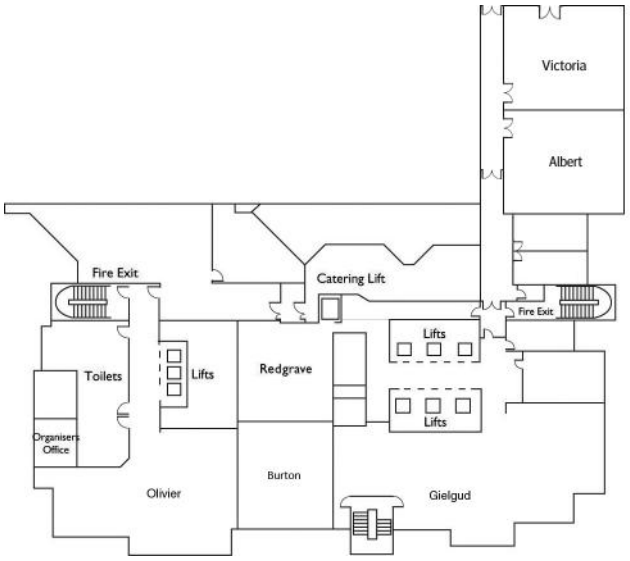
# The QEII Centre - Ground and First Floors



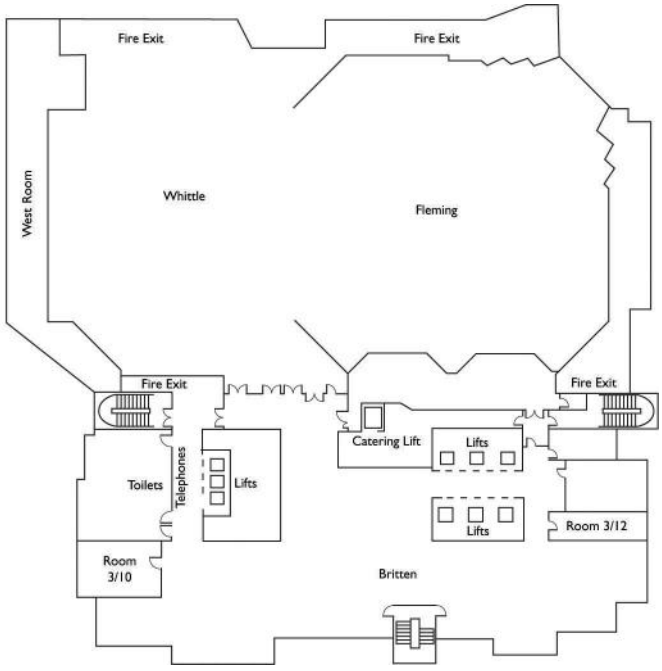




**Full café facilities will be open in the Pickwick on the 1st floor from 8.00am to 4.00pm on Wednesday 26 and Thursday 27 November and from 8.00am to 2.30pm on Friday 28 November. A snack bar, serving hot and cold drinks, sandwiches and confectionery only, will be open at the same times in the Whittle & Fleming on the 3rd floor.**

# The QEII Centre - Second and Third Floors




SECOND FLOOR




THIRD FLOOR

**Full café facilities will be open in the Pickwick on the 1st floor from 8.00am to 4.00pm on Wednesday 26 and Thursday 27 November and from 8.00am to 2.30pm on Friday 28 November. A snack bar, serving hot and cold drinks, sandwiches and confectionery only, will be open at the same times in the Whittle & Fleming on the 3rd floor.**

This promotional advert is developed and funded by AstraZeneca for UK HCPs only.

TEZSPIRE (tezepelumab) Subcutaneous Injection

EVIDENCE Experience

# So what's your early experience with **TEZSPIRE**?

Real-world evidence and usage of TEZSPIRE is fast growing across the UK.

Explore the TEZSPIRE video library to learn more about the experience so far.

Learn about the 12-month outcomes of the first 175 patients on TEZSPIRE at Guys Hospital.




**Professor David Jackson** Respiratory Consultant and Professor of Respiratory Medicine, Guy's and St Thomas' Hospitals, King's College London

**CLICK HERE TO WATCH**
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Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App store. Adverse events should also be reported to AstraZeneca by visiting https://contactazmedical.astrazeneca.com or by calling 0800 783 0033.

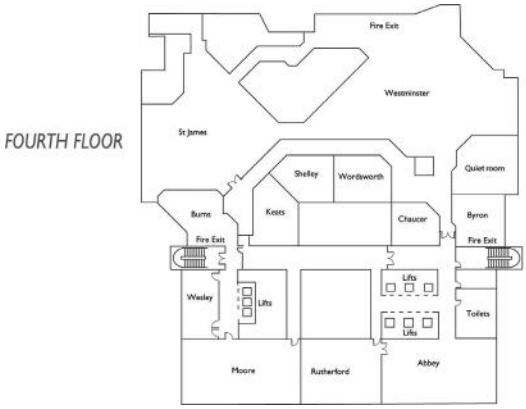
TEZSPIRE (tezepelumab) is indicated as an add-on maintenance treatment in adults and adolescents 12 years and older with severe asthma who are inadequately controlled despite high dose inhaled corticosteroids plus another medicinal product for maintenance treatment.

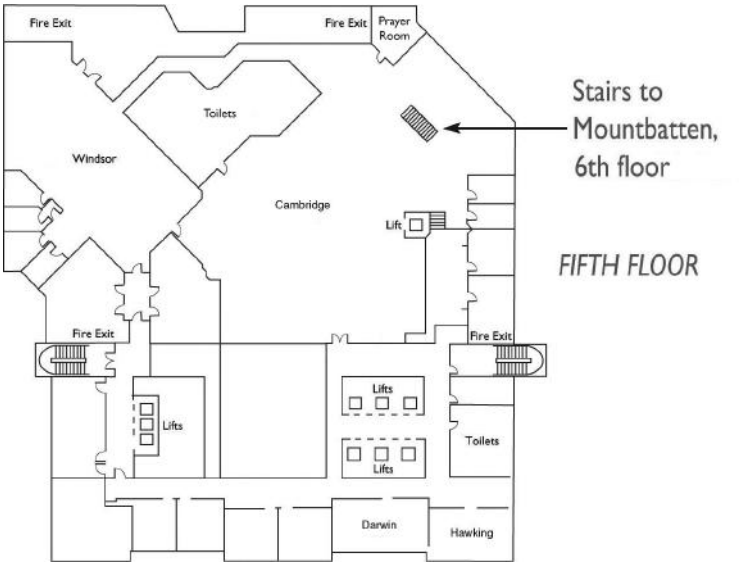
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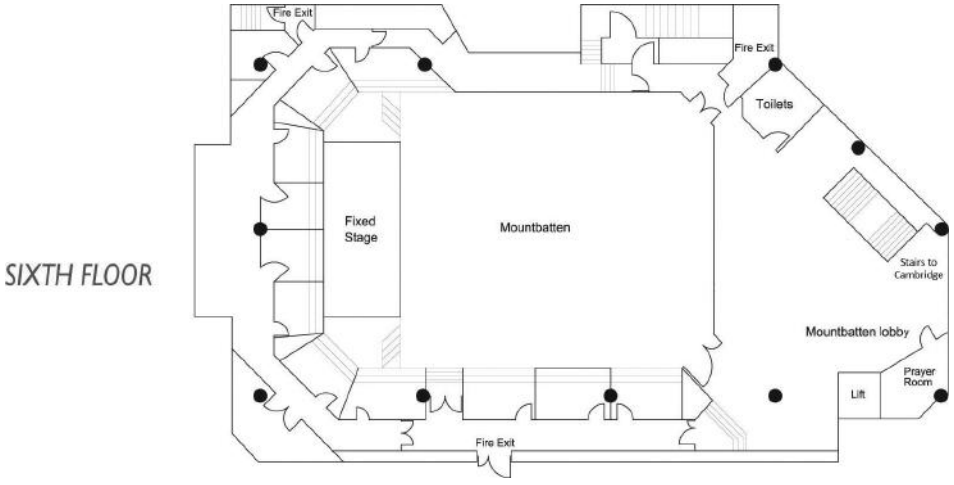
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# The QEII Centre - Fourth, Fifth and Sixth Floors




**Full café facilities will be open in the Pickwick on the 1st floor from 8.00am to 4.00pm on Wednesday 26 and Thursday 27 November and from 8.00am to 2.30pm on Friday 28 November. A snack bar, serving hot and cold drinks, sandwiches and confectionery only, will be open at the same times in the Whittle & Fleming on the 3rd floor.**

# DAILY PROGRAMME

## WEDNESDAY 26 NOVEMBER 2025

| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 8.00am-9.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming/3rd |
| 8.45am-4.00pm<br><br>10.00am-11.00am | Poster viewing<br><br>Authors present | P1-P15 | From primary to secondary care: evolving management of COPD | Whittle & Fleming/3rd |
| | | P16-P30 | Monitoring, managing and treating ILD to improve outcomes | |
| | | P31-P45 | Enhancing outcomes in chronic respiratory disease: the role of rehabilitation and physiotherapy | |
| | | P46-P60 | Advances in sleep medicine | |
| | | P61-P76 | Holistically improving asthma care | |
| 8.45am-4.00pm | Moderated poster viewing | M1-M16 | Impact of treatment on COPD outcomes: from smoking to biologics | Cambridge/5th |
| 8.00am-8.30am | BTS Journal Club | | Critical care | Moore/4th |
| 8.30am-10.30am | Joint BTS/BALR symposium (part I) | | At the cutting edge of models for lung disease | Westminster/4th |
| 8.45am-10.15am | Symposium | | Lung cancer screening: update and optimisation | Churchill/Ground |
| 8.45am-10.15am | Symposium | | The unhappy host: infection and immune-compromise | Mountbatten/6th |
| 8.45am-10.20am | Spoken session | S1-S6 | Emerging clinical trial data | Windsor/5th |
| 8.45am-10.20am | Spoken session | S7-S12 | Addressing inequalities in COPD | St James/4th |
| 8.45am-10.20am | Spoken session | S13-S18 | Approaches to predict survival and increased mortality risk in ILD | Moore/4th |
| 8.45am-10.20am | Spoken session | S19-S24 | Sleep matters | Rutherford/4th |
| 8.45am-10.20am | Spoken session | S25-S30 | Physiological approaches to decoding breathlessness | Albert/2nd |
| 9.15am-10.15am | Open session | | Supporting women in respiratory medicine | Abbey/4th |
| 10.00am-11.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming and Britten/3rd |
| 10.45am-12.15pm | Symposium | | Optimising sleep disordered breathing: smart approaches for better outcomes | St James/4th |
| 10.45am-12.15pm | Symposium | | Piecing together the personalisation puzzle – is PR the missing piece? | Churchill/Ground |
| 10.45am-12.15pm | Symposium | | Chronic cough as a neuronal disorder | Mountbatten/6th |
| 10.45am-12.20pm | Spoken session | S31-S36 | Optimising diagnostics in thoracic malignancy | Moore/4th |
| 10.45am-12.20pm | Spoken session | S37-S42 | Bronchiectasis: clinical trials and real world impact | Abbey/4th |
| 10.45am-12.20pm | Spoken session | S43-S48 | Paediatric asthma: are we getting it right? | Rutherford/4th |
| 10.45am-12.35pm | Spoken session | S49-S55 | Tracking the science: smarter asthma strategies | Windsor/5th |
| 11.00am-12.00pm | SAG open meeting | | Pulmonary Embolism and other Pulmonary Vascular Diseases | Albert/2nd |

**Please see page Axii for a complete list of the days and times of BTS Specialist Advisory Group open meetings.**

Coffee and tea are complimentary ONLY during the coffee/tea break times shown. Outside these times, drinks may be purchased from the café in the Pickwick (1st floor), or the snack bar in the Whittle & Fleming (3rd floor).

# DAILY PROGRAMME (cont.) WEDNESDAY 26 NOVEMBER 2025

| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 11.00am-12.00pm | SAG open meeting | | Tobacco Dependency | Victoria/2nd |
| 11.00am-1.00pm | Joint BTS/BALR symposium (part 2) | | At the cutting edge of models for lung disease | Westminster/4th |
| 12.00pm-2.15pm | LUNCH and visit the exhibition | *Not included in the delegate fee, but available to purchase on site. Card payments only* | | Pickwick / 1st and Whittle & Fleming/3rd |
| 12.30pm-1.30pm | SAG open meeting | | Pulmonary Rehabilitation | Albert/2nd |
| 12.45pm-1.45pm | SAG open meeting | | Pleural Disease | Victoria/2nd |
| 1.15pm-2.00pm | BTS Scientific Guest Lecture | | Understanding immune regulation of the lung and what it means for lung health | Churchill/Ground |
| 2.00pm-3.00pm | SAG open meeting | | Bronchiectasis | Albert/2nd |
| 2.00pm-3.00pm | SAG open meeting | | Cough | Victoria/2nd |
| 2.05pm-4.05pm | Moderated Poster discussion | M1-M16 | Impact of treatment on COPD outcomes: from smoking to biologics | Cambridge/5th |
| 2.15pm-3.45pm | Symposium | | Updates and advances in pleural effusions | Churchill/Ground |
| 2.15pm-3.45pm | Symposium | T1-T6 | BTS/BALR/A+LUK Early Career Investigator of the Year Award Symposium | Windsor/5th |
| 2.15pm-4.05pm | Poster discussion | P1-P15 | From primary to secondary care: evolving management of COPD | St James/4th |
| 2.15pm-4.05pm | Poster discussion | P16-P30 | Monitoring, managing and treating ILD to improve outcomes | Moore/4th |
| 2.15pm-4.05pm | Poster discussion | P31-P45 | Enhancing outcomes in chronic respiratory disease: the role of rehabilitation and physiotherapy | Abbey/4th |
| 2.15pm-4.05pm | Poster discussion | P46-P60 | Advances in sleep medicine | Rutherford/4th |
| 2.15pm-4.15pm | Poster discussion | P61-P76 | Holistically improving asthma care | Westminster/4th |
| 2.30pm-4.00pm | Joint BTS/BPRS symposium | | Sustainable respiratory care for future generations | Mountbatten/6th |
| 3.00pm-4.30pm | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming and Britten/3rd |
| 3.15pm-4.10pm | SAG open meeting | | Acute and Complex Pulmonary Infections | Albert/2nd |
| 3.15pm-4.10pm | SAG open meeting | | Global Lung Health | Victoria/2nd |
| 4.15pm-4.45pm | Award presentations | | | Churchill/Ground |
| 4.45pm-5.30pm | The BTS President's Address | | "Breathtaking!" | Churchill/Ground |
| 5.35pm-6.05pm | BTS AGM | | BTS Annual General Meeting (BTS members only) | Churchill/Ground |

**Please see page Axii for a complete list of the days and times of BTS Specialist Advisory Group open meetings.**

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# DAILY PROGRAMME

## THURSDAY 27 NOVEMBER 2025

| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 8.00am-9.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming/3rd |
| 8.45am-4.00pm | Poster viewing | P77-P87 | Best practice in pleural procedures and pneumothorax | Whittle & Fleming/3rd |
| 10.00am-11.00am | Authors present | P88-P102 | Insights and advances in ILD care | |
| | | P103-P117 | The road to change in chronic cough | |
| | | P118-P132 | Respiratory realities: the patient perspective | |
| | | P133-P147 | Paediatric infection, inflammation and physiology | |
| | | P148-P162 | Risk predictors and diagnostics in COPD | |
| | | P163-P177 | Cancer: from screening to therapy | |
| | | P178-P192 | Respiratory infections: pathogens to patients | |
| 8.45am-4.00pm | Moderated poster viewing | M17-M32 | The multi-morbidity of asthma | Cambridge/5th |
| 8.00am-8.30am | BTS Journal Club | | Tobacco dependency / smoking / vaping | Moore/4th |
| 8.45am-10.15am | Symposium | | Key determinants of outcome in asthma: from onset to remission | Churchill/Ground |
| 8.45am-10.15am | Symposium | | Highlights from *Thorax* and *Lancet Respiratory Medicine* | Mountbatten/6th |
| 8.45am-10.20am | Spoken session | S56-S61 | Impact of infection on disease mechanisms | St James/4th |
| 8.45am-10.20am | Spoken session | S62-S67 | Biology and clinical insights in cancer | Westminster/4th |
| 8.45am-10.20am | Spoken session | S68-S73 | Cystic fibrosis: impact of CFTR modulators | Moore/4th |
| 8.45am-10.20am | Spoken session | S74-S79 | Sleep apnoea and beyond | Abbey/4th |
| 8.45am-10.20am | Spoken session | S80-S85 | Pulmonary hypertension and CTEPH: classification and treatment | Rutherford/4th |
| 8.45am-10.20am | Spoken session | S86-S91 | Mechanisms of IPF pathogenesis | Albert/2nd |
| 9.00am-10.30am | Symposium | | Respiratory workforce of the future | Windsor/5th |
| 10.00am-11.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming and Britten/3rd |
| 10.45am-11.45am | SAG open meeting | | Asthma | Abbey/4th |
| 10.45am-11.45am | SAG open meeting | | Cystic Fibrosis | Rutherford/4th |
| 10.45am-11.45am | SAG open meeting | | Pharmacist | Albert/2nd |
| 10.45am-11.45am | SAG open meeting | | Lung Cancer and Mesothelioma | Victoria/2nd |
| 10.45am-12.20pm | Spoken session | S92-S97 | Enhancing outcomes in respiratory failure: from trials to real world practice | Westminster/4th |
| 10.45am-12.30pm | Symposium | | Plenary scientific symposium | Churchill/Ground |
| 12.00pm-2.15pm | LUNCH and visit the exhibition | *Not included in the delegate fee, but available to purchase on site. Card payments only* | | Pickwick /1st and Whittle & Fleming/3rd |
| 1.00pm-1.45pm | Morriston Davies Guest Lecture | | Vaccine preventable respiratory illness | Churchill/Ground |
| 2.00pm-3.00pm | SAG open meeting | | Sleep | Victoria/2nd |
| 2.00pm-3.25pm | Poster discussion | P77-P87 | Best practice in pleural procedures and pneumothorax | Abbey/4th |

**Please see page Axii for a complete list of the days and times of BTS Specialist Advisory Group open meetings.**

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| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 2.00pm-3.50pm | Poster discussion | P88-P102 | Insights and advances in ILD care | Westminster/4th |
| 2.00pm-3.50pm | Poster discussion | P103-P117 | The road to change in chronic cough | Moore/4th |
| 2.00pm-3.50pm | Poster discussion | P118-P132 | Respiratory realities: the patient perspective | Rutherford/4th |
| 2.00pm-3.50pm | Poster discussion | P133-P147 | Paediatric infection, inflammation and physiology | Albert/2nd |
| 2.00pm-4.00pm | Moderated Poster discussion | M17-M32 | The multi-morbidity of asthma | Cambridge/5th |
| 2.15pm-3.15pm | Symposium | | Joint BTS/A+LUK/BALR Mid-Career Lectures | Churchill/Ground |
| 2.15pm-3.45pm | Symposium | | Lung cancer staging: TNM9 and recent developments in oncological therapy – implications for practice | Mountbatten/6th |
| 2.15pm-3.45pm | Symposium | | The story of three rare lung diseases | Windsor/5th |
| 2.15pm-3.50pm | Spoken session | S98-S103 | Co-morbidity and novel treatments in COPD | St James/4th |
| 3.15pm-4.15pm | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming and Britten/3rd |
| 4.15pm-5.15pm | SAG open meeting | | Nurse | Victoria/2nd |
| 4.15pm-5.45pm | Symposium | | Transforming respiratory across the current landscape | Churchill/Ground |
| 4.15pm-5.45pm | Symposium | | Perspectives in pulmonary vascular disease: mechanisms, monitoring and management | Mountbatten/6th |
| 4.15pm-5.45pm | Joint BTS/BPRS symposium | | Abnormalities of lung development-looking towards a brighter future | Windsor/5th |
| 4.15pm-5.45pm | Open session | | Translational rare lung disease research (LifeArc) | Albert/2nd |
| 4.15pm-5.50pm | Spoken session | S104-S109 | Deep dive into the asthma airways | St James/4th |
| 4.15pm-6.05pm | Spoken session | S110-S116 | Occupational hazards of exposure to particulates | Rutherford/4th |
| 4.15pm-6.05pm | Poster discussion | P148-P162 | Risk predictors and diagnostics in COPD | Westminster/4th |
| 4.15pm-6.05pm | Poster discussion | P163-P177 | Cancer: from screening to therapy | Moore/4th |
| 4.15pm-6.05pm | Poster discussion | P178-P192 | Respiratory infections: pathogens to patients | Abbey/4th |
| 5.45pm-7.00pm | The President's Reception – All welcome! | | | Britten/3rd |

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# DAILY PROGRAMME

## FRIDAY 28 NOVEMBER 2025

| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 8.00am-9.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming/3rd |
| 8.45am-2.00pm | Poster viewing | P193-P207 | The logic of biologics | Whittle & Fleming/3rd |
| 10.00am-11.00am | Authors present | P208-P222 | The interventionalist's toolkit: bronchoscopy to thoracoscopy | |
| | | P223-P237 | Ventilatory support in focus | |
| | | P238-P252 | New insights from the fundamental sciences | |
| | | P253-P268 | Updates in lung disease diagnostics (and PFTs) | |
| | | P269-P278 | Real world care of pleural fluid and infection | |
| | | P279-P288 | Advances in pulmonary vascular medicine | |
| | | P289-P303 | More than just inhalers in asthma | |
| 8.45am-5.15pm | Moderated poster viewing | M33-M48 | TB: from bench to bedside | Cambridge/5th |
| 8.00am-8.30am | BTS Journal Club | | Respiratory pharmacology | Moore/4th |
| 8.30am-10.00am | Symposium | | Predicting the future in ILD | Churchill/Ground |
| 8.30am-10.00am | Symposium | | Breathing room: confronting lung diseases from modern workplaces | Windsor/5th |
| 8.30am-10.05am | Spoken session | S117-S122 | New insights in COPD pathogenesis | Westminster/4th |
| 8.30am-10.05am | Spoken session | S123-S128 | Patient centred research in respiratory infection: from design to outcomes | Abbey/4th |
| 8.30am-10.20am | Spoken session | S129-S135 | Asthma remission and beyond | St James/4th |
| 8.45am-10.15am | Symposium | | Removing barriers to better tuberculosis care | Mountbatten/6th |
| 8.45am-10.20am | Spoken session | S136-S141 | Pulmonary rehabilitation: what works and why | Moore/4th |
| 9.00am-10.00am | SAG open meeting | | Critical Care, Respiratory Failure and Mechanical Ventilation | Rutherford/4th |
| 9.00am-10.00am | SAG open meeting | | BTS/ARTP – Physiology | Victoria/2nd |
| 9.00am-10.00am | Open session | | From data to impact: using NRAP | Albert/2nd |
| 10.00am-11.00am | COFFEE/TEA and visit the exhibition | | | Whittle & Fleming and Britten/3rd |
| 10.30am-11.30am | SAG open meeting | | Occupational and Environmental Lung Disease | Albert/2nd |
| 10.30am-11.30am | SAG open meeting | | COPD | Victoria/2nd |
| 10.30am-11.50am | Spoken session | S142-S146 | Translational pulmonary vascular science | Rutherford/4th |
| 10.30am-12.00pm | Symposium | | Approaching respiratory physiology with a different perspective | Churchill/Ground |
| 10.30am-12.00pm | Symposium | | Ex-vivo lung perfusion: lessons in pathophysiology and therapeutics from lung transplantation | Windsor/5th |
| 10.30am-12.05pm | Spoken session | S147-S152 | Pleural disease – basic science, new trials | Westminster/4th |
| 10.30am-12.05pm | Spoken session | S153-S158 | The importance of early diagnosis in ILD: novel approaches | Abbey/4th |

**Please see page Axii for a complete list of the days and times of BTS Specialist Advisory Group open meetings.**

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# DAILY PROGRAMME (cont.)

## FRIDAY 28 NOVEMBER 2025

| Time | Details | | | Location/Floor |
|---|---|---|---|---|
| 10.35am-12.10pm | Spoken session | S159-S164 | Thinking outside the (asthma) biologic box | St James/4th |
| 10.45am-12.15pm | Symposium | | BTS audit and quality improvement | Mountbatten/6th |
| 10.45am-12.20pm | Spoken session | S165-S170 | The role of neutrophils in respiratory disease | Moore/4th |
| 11.45am-12.45pm | SAG open meeting | | Specialty Trainee | Albert/2nd |
| 11.45am-12.45pm | SAG open meeting | | TB and Non-Tuberculous Mycobacteria | Victoria/2nd |
| 12.00pm-2.00pm | LUNCH and visit the exhibition | *Not included in the delegate fee but available to purchase on site. Card payments only. Exhibition closes at 2.00pm* | | Pickwick / 1st and Whittle & Fleming/3rd |
| 12.30pm-1.15pm | BTS Grand Challenge Guest Lecture | | From target to treatment: shaping drugs for respiratory patients | Churchill/Ground |
| 1.30pm-2.30pm | SAG open meeting | | Interstitial and Rare Lung Disease | Albert/2nd |
| 1.30pm-3.00pm | Symposium | | Transforming the COPD landscape: from diagnosis to digital innovation | Churchill/Ground |
| 1.30pm-3.00pm | Symposium | | "The usual suspects": bad bugs and what is being done about them | Mountbatten/6th |
| 1.30pm-3.00pm | Symposium | | Inspiring collaborative research in respiratory medicine | Windsor/5th |
| 1.30pm-3.20pm | Poster discussion | P193-P207 | The logic of biologics | St James/4th |
| 1.30pm-3.20pm | Poster discussion | P208-P222 | The interventionalist's toolkit: bronchoscopy to thoracoscopy | Westminster/4th |
| 1.30pm-3.20pm | Poster discussion | P223-P237 | Ventilatory support in focus | Moore/4th |
| 1.30pm-3.20pm | Poster discussion | P238-P252 | New insights from the fundamental sciences | Abbey/4th |
| 1.30pm-3.30pm | Poster discussion | P253-P268 | Updates in lung disease diagnostics (and PFTs) | Rutherford/4th |
| 3.00pm-3.30pm | COFFEE/TEA (Exhibition closed) | | | Britten/3rd |
| 3.15pm-4.45pm | Symposium | | A lot of yap about CAP, HAP and VAP | Churchill/Ground |
| 3.15pm-4.45pm | Symposium | | Bronchiectasis – recent advances and clinical trials | Mountbatten/6th |
| 3.15pm-4.50pm | Spoken session | S171-S176 | How the eosinophil and T2 biomarkers guide decisions in COPD | Windsor/5th |
| 3.15pm-5.15pm | Moderated Poster discussion | M33-M48 | TB: from bench to bedside | Cambridge/5th |
| 3.30pm-4.45pm | Poster discussion | P269-P278 | Real world care of pleural fluid and infection | Westminster/4th |
| 3.30pm-4.45pm | Poster discussion | P279-P288 | Advances in pulmonary vascular medicine | Moore/4th |
| 3.30pm-5.20pm | Poster discussion | P289-P303 | More than just inhalers in asthma | St James/4th |

**Please see page Axii for a complete list of the days and times of BTS Specialist Advisory Group open meetings.**

Coffee and tea are complimentary ONLY during the coffee/tea break times shown. Outside these times, drinks may be purchased from the café in the Pickwick (1st floor), or the snack bar in the Whittle & Fleming (3rd floor).

# OPEN MEETINGS OF THE BTS SPECIALIST ADVISORY GROUPS

Open meetings of the British Thoracic Society Specialist Advisory Groups (SAGs) will take place during the Winter Meeting. All participants are welcome to attend and hear more about the work of the SAGs and be involved in planning symposia for future Summer and Winter Meeting programmes.

## WEDNESDAY 26 NOVEMBER

| Time | SAG | Location |
|---|---|---|
| 11.00am-12.00pm | Pulmonary Embolism and other Pulmonary Vascular Diseases | Albert, 2nd floor |
| 11.00am-12.00pm | Tobacco Dependency | Victoria, 2nd floor |
| 12.30pm-1.30pm | Pulmonary Rehabilitation | Albert, 2nd floor |
| 12.45pm-1.45pm | Pleural Disease | Victoria, 2nd floor |
| 2.00pm-3.00pm | Bronchiectasis | Albert, 2nd floor |
| 2.00pm-3.00pm | Cough | Victoria, 2nd floor |
| 3.15pm-4.10pm | Acute and Complex Pulmonary Infections | Albert, 2nd floor |
| 3.15pm-4.10pm | Global Lung Health | Victoria, 2nd floor |

## THURSDAY 27 NOVEMBER

| Time | SAG | Location |
|---|---|---|
| 10.45am-11.45am | Asthma | Abbey, 4th floor |
| 10.45am-11.45am | Cystic Fibrosis | Rutherford, 4th floor |
| 10.45am-11.45am | Pharmacist | Albert, 2nd floor |
| 10.45am-11.45am | Lung Cancer and Mesothelioma | Victoria, 2nd floor |
| 2.00pm-3.00pm | Sleep | Victoria, 2nd floor |
| 4.15pm-5.15pm | Nurse | Victoria, 2nd floor |

## FRIDAY 28 NOVEMBER

| Time | SAG | Location |
|---|---|---|
| 9.00am-10.00am | Critical Care, Respiratory Failure and Mechanical Ventilation | Rutherford, 4th floor |
| 9.00am-10.00am | BTS/ARTP – Physiology | Victoria, 2nd floor |
| 10.30am-11.30am | Occupational and Environmental Lung Disease | Albert, 2nd floor |
| 10.30am-11.30am | COPD | Victoria, 2nd floor |
| 11.45am-12.45pm | Specialty Trainee | Albert, 2nd floor |
| 11.45am-12.45pm | TB and Non-Tuberculous Mycobacteria | Victoria, 2nd floor |
| 1.30pm-2.30pm | Interstitial and Rare Lung Disease | Albert, 2nd floor |

## BTS AWARD PRESENTATIONS

**Wednesday 26 November at 4.15pm in the Churchill, Ground floor**

The presentations will be made just before the BTS President's Address, for the annual BTS Medal, BTS President's Award, BTS Award for Meritorious Service, BTS/BALR/A+LUK Early Career Investigator of the Year, BTS Medical Student and BTS/A+LUK/BALR Mid-Career Lecture Awards.
Please come along to this session to congratulate the winners.

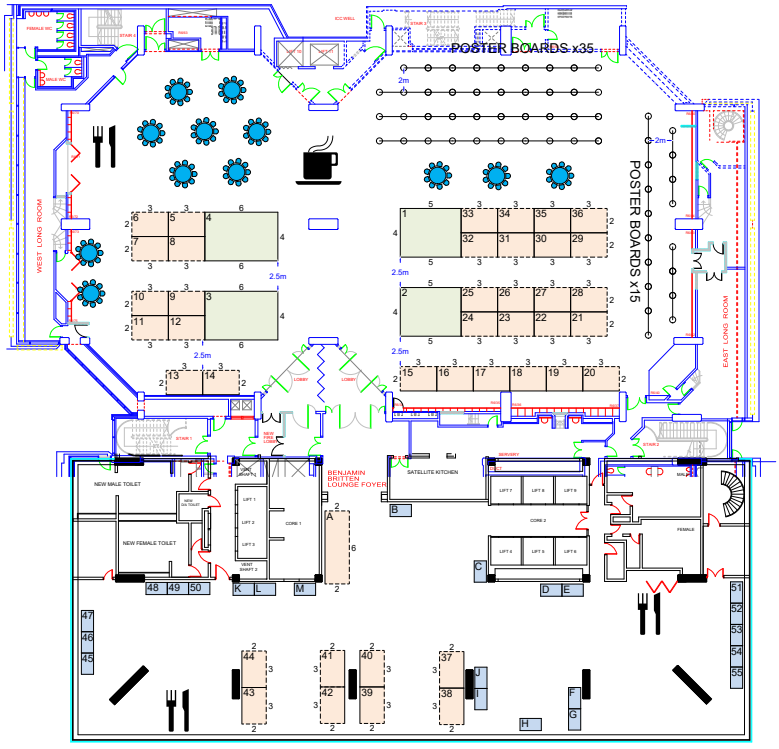
## THE BTS PRESIDENT'S RECEPTION

**Thursday 27 November from 5.45pm to 7.00pm in the Britten, 3rd floor**

All participants are warmly invited to join us for this social occasion.


*Thorax* 2025;**80**(Suppl 2):Ai–Alxxxix

# FLOOR PLAN OF THE EXHIBITION STANDS




## Exhibitors and stand numbers

| | |
|---|---|
| 15 | APR Medtech |
| 16 | Aquilant Endoscopy |
| 2, 45 & 46 | AstraZeneca |
| 34 | BD |
| 21 | Broncus Medical Inc |
| 9 | Celltrion Healthcare UK Ltd |
| 4 | Chiesi |
| 5 | Cipla |
| 12 | Creo Medical |
| 6 | Erbe Medical Ltd |
| 10 | Essential Pharma Ltd |
| 18 | Fannin |
| 24 | Fisher & Paykel Healthcare |
| 3, 37 & 38 | GSK |
| 26, 30 & 31 | Insmed |
| 17 | Inspire Medical Systems |
| 32 | Its Interventional |
| 25 | MSD |
| 22 | NeilMed Pharmaceuticals |
| 43 & 44 | NIOX Group plc |
| 13 | Olympus |
| 19 | OmniaMed Communications |
| 14 | Orion Pharma (UK) Ltd |
| 28 | Pfizer Ltd |
| 27 | Pharming |
| 20 | Rocket Medical |
| 1, 35, 36, 39 & 40 | Sanofi / Regeneron |
| 7 & 8 | STADA Group, Thornton & Ross Ltd |
| 33 | Stirling Anglian Pharmaceuticals |
| 11 | Sunvou |
| 29 | Vapotherm |
| 41 | Vitalograph |

## Charity/non-commercial organisation

| | |
|---|---|
| D | Action for Pulmonary Fibrosis |
| F | Association of Chartered Physiotherapists in Respiratory Care (ACPRC) |
| I | Association of Respiratory Nurses (ARNS) |
| J | Association for Respiratory Technology & Physiology (ARTP) |
| 47 | Asthma + Lung UK |
| G | British Association for Lung Research (BALR) |
| A | British Thoracic Society (BTS) |
| 49 | ILD IN |
| 42 | LifeArc |
| K | Mesothelioma UK |
| B | National Respiratory Audit Programme (NRAP) |
| M | NTM Patient Care UK & NTM Network |
| 50 | PCD Support Group |
| C | Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS) |
| H | Royal College of Speech & Language Therapists |
| L | SarcoidosisUK |

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**8.00am-9.00am**
**Whittle & Fleming, 3rd floor**
**COFFEE/TEA and visit the exhibition stands**

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**8.45am-4.00pm**
**Whittle & Fleming, 3rd floor**
**POSTER VIEWING**
*Authors present: 10.00am-11.00am*

**P1-P15**
**From primary to secondary care: evolving management of COPD**
*Discussion of abstracts will take place from 2.15pm to 4.05pm in the St James, 4th floor*

**P16-P30**
**Monitoring, managing and treating ILD to improve outcomes**
*Discussion of abstracts will take place from 2.15pm to 4.05pm in the Moore, 4th floor*

**P31-P45**
**Enhancing outcomes in chronic respiratory disease: the role of rehabilitation and physiotherapy**
*Discussion of abstracts will take place from 2.15pm to 4.05pm in the Abbey, 4th floor*

**P46-P60**
**Advances in sleep medicine**
*Discussion of abstracts will take place from 2.15pm to 4.05pm in the Rutherford, 4th floor*

**P61-P76**
**Holistically improving asthma care**
*Discussion of abstracts will take place from 2.15pm to 4.15pm in the Westminster, 4th floor*

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**8.45am-4.00pm**
**Cambridge, 5th floor**
**MODERATED POSTER VIEWING**

**M1-M16**
**Impact of treatment on COPD outcomes: from smoking to biologics**
*Discussion of abstracts will take place from 2.05pm to 4.05pm in the Cambridge, 5th floor*

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**8.00am-8.30am**
**Moore, 4th floor**
**JOURNAL CLUB**
**CRITICAL CARE**

*Dr Helen Newman (London)*

*Learning objectives*

*To review the latest publications and evidence in the field of critical care.*

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**8.30am-10.30am**
**Westminster, 4th floor**
**JOINT BTS/BALR SYMPOSIUM PART 1**
**AT THE CUTTING EDGE OF MODELS FOR LUNG DISEASE**

*Chaired by: Dr Alison John (London) and Dr Julie Worrell (Glasgow & Dublin)*

| | |
|---|---|
| **8.30am** | Insights into lung diseases from foetal organoids<br>Dr Jennifer Dickens (Cambridge) |
| **9.10am** | Modelling genetic airway diseases using adult stem cell cultures<br>Dr Robert Hynds (London) |
| **9.50am** | Precision cut lung slices for investigation of mechanisms and therapeutic targets in lung diseases<br>Professor Jane Bourke (Melbourne) |

*Learning objectives*

*1) To highlight the understanding of ILD and how to treat this disease emerging from novel organoid culture systems.*

*2) To demonstrate the use of 3D epithelial cultures to explore the mechanisms that allow the lung to differentiate and repair.*

*3) To give an overview of how precision cut lung slices are being used to investigate disease mechanisms and therapeutic targets.*

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**8.45am-10.15am**
**Churchill, ground floor**
**SYMPOSIUM**
**LUNG CANCER SCREENING: UPDATE AND OPTIMISATION**

*Chaired by: Dr Anna Bibby (Bristol) and Professor Matthew Callister (Leeds)*

| | |
|---|---|
| **8.45am** | Optimising the screening process – insights from the SUMMIT study<br>Professor Sam Janes (London) |
| **9.15am** | Who should (and shouldn't) we invite for screening?<br>Professor Gerard Silvestri (Charleston, South Carolina) |

**9.45am** Incidental findings: reporting and downstream impact
Dr Emily Bartlett (London)

*Learning objectives*

*1) To update on results from the SUMMIT trial of lung cancer screening, including nodule management and other outputs.*

*2) To consider whether we should expand screening to lower risk populations and whether we can optimise selection to ensure it is offered to those who will benefit the most.*

*3) To cover reporting and management of incidental findings in lung cancer screening – what should and shouldn't be reported and how should these be managed.*

**8.45am-10.15am**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## THE UNHAPPY HOST: INFECTION AND IMMUNE-COMPROMISE

*Chaired by: Dr David Connell (Dundee) and Dr Vanessa Sancho-Shimizu (London)*

**8.45am** Clinical immunology: tackling the underlying causes of infection
Dr Anita Chandra (Cambridge)

**9.15am** Interferon autoantibodies: newly discovered drivers of respiratory viral susceptibility
Dr Paul Bastard (Paris)

**9.45am** Phage therapy for atypical infections in the immuno-compromised host: hype or hope?
Dr Ghady Haidar (Pittsburgh)

*Learning objectives*

*1) Summarise the immunological aspects of respiratory infection susceptibility in immunosuppressed individuals.*

*2) Understanding the prevalence and importance of anti-interferon antibodies as new discovered susceptibility factors for respiratory viral infection.*

*3) Explore the concepts and evidence underpinning the use of phage therapy to treat atypical infection in the immunocompromised host.*

**8.45am-10.20am**
**Windsor, 5th floor**
**SPOKEN SESSION: S1-S6**

## Emerging clinical trial data

*Chaired by: Professor Judy Bradley (Belfast) and Dr Alexander Mathioudakis (Manchester)*

**8.50am** **S1**
Randomised, placebo-controlled trial of astegolimab for COPD with frequent exacerbations: ALIENTO
N Greening, CE Brightling, R Tal-Singer, B Celli, A Agusti, S Korn, P Nair, P Bremner, NA Hanania, W Janssens, DS Cheung, MA Grimbaldeston, J Ng, X Yang, D Mohan, CF Vogelmeier

**9.05am** **S2**
What is the clinical effectiveness and cost-effectiveness of nebulised 7% saline in patients with chronic obstructive pulmonary disease (COPD) – (MucAct COPD Trial)
AT Hill, S Lewis, S Tuck, A Skeikh, A Stoddart, M Gibbons, L Gibbons, F Harris, H Bayes, M Patel, A Smith, L Murray, D Saralaya, J Suntharalingam, A de Soyza, A Turner, A Usher, D Dosanjh, A Dev Vellore, M Crooks, L Linkson, P Bedi, S Finch, R Mustafa, T Nagarajan, K Ibrahim, D Miller, R Aul, D Anderson, E Ward, B Selby, K Oatey, J Miller, S Hart, J Wildgoose, S Ramalingam, S Donaldson, J Crowe, A Clarke, R Milroy, P Hodkinson, R Frangulyan

**9.20am** **S3**
Standard versus accelerated weaning from non-invasive ventilation (NIV) in chronic obstructive pulmonary disease directed by the NIV outcomes score: a randomised controlled trial
K Brewin, MH Naseer, R Arries, N Lane, A Scadding, D Ghosh, B Prudon, M Sovani, T Hartley, S McCarthy, J Gray, E Pakpahan, S Bourke

**9.35am** **S4**
Effects of Hyperoxia on Pulmonary Inflammation and organ injury in a human in vivo model (HIPI): a randomised, double-blind, placebo-controlled trial

DA Linden, D Dorrian, M Glenn, K Welzenbach, MJ Rowland, M Yang, L Klein, E Randazzo, M Sindelar, J Schlotterbeck, A Cattini, S Tandel, A Dushianthan, MC McKelvey, M Bailey, J Conlon, D Moore, S Carr, JM Bradley, D Simpson, CC Taggart, JC Kidney, CM O'Kane, DF McAuley

**9.50am** **S5**

HAP-FAST: a pilot sequential multiple assignment randomised trial (SMART) comparing chest x-ray to low-dose CT scan and empirical antibiotics to antibiotics guided by the BIOFIRE® FILM ARRAY® pneumonia plus panel in adults with suspected non-ventilator-associated hospital-acquired pneumonia

D Wootton, I Powell, S Willshaw, A Tao, N Shafiqa, E Adegbenro, K Cook, A Cross, D Mclaughlan, H Singh, K Kalapurakal, N Senior, L Turtle, S Aston, AL Howard, L Pilling, R Bazaz, S Trewick, I Poku, A Alvarez-Nishio, E Lam, S Abrams, A Collins, F Sherratt, B Young, A Jones

**10.05am** **S6**

Conservative Management in Traumatic Pneumothoraces in the Emergency Department (CoMiTED): a randomised controlled trial

A Annaw, J Banks, JR Benger, N Blythe, C Clement, K Coates, A Edwards, D Gaunt, R Kandiyali, A Lane, F Lecky, NA Maskell, C Metcalfe, S Rees, J Thompson, S Walker, D West, E Carlton, on behalf of the CoMiTED Investigators

**8.45am-10.20am**
**St James, 4th floor**
**SPOKEN SESSION: S7-S12**

## Addressing inequalities in COPD

*Chaired by: Professor John Hurst (London) and Ms Joanne King (Frimley)*

**8.50am** **S7**

National respiratory audit programme (NRAP) highlights variation in delivery of discharge bundles of evidence-based interventions in patients admitted to hospital with COPD exacerbation

H Burke, AJ Adamson, J Henderson, JK Quint, JW Dodd, TMA Wilkinson, I Patel

**9.05am** **S8**

Addressing inequalities in respiratory emergencies through data-driven interventions

V Bellamy, H Wilcock, R Crabtree, M Sutcliffe

**9.20am** **S9**

A matter of life and breath: understanding the needs of people with chronic obstructive pulmonary disease

S Thomas, REK Russell, M Bafadhel

**9.35am** **S10**

Care closer to home: the role of ambulance service specialist practitioners in urgent care in managing category 2 breathing difficulties

H Wilcock, C Marsh, L-M Hargreaves, R Crabtree, T Millington, M Sutcliffe

**9.50am** **S11**

SPIRO-AID: a randomized controlled trial of AI-Assisted Spirometry Interpretation in primary care

G Doe, W Banya, G Edwards, M Topalovic, E El-Emir, RA Evans, R Russell, E Smets, K Van Orshoven, KP Sylvester, AP Sunjaya, D Scott, M De Vos, A Elmahy, SJC Taylor, A Hutchinson, NS Hopkinson, M Morgan, SS Kon, S Patel, I Jarrold, E Kennington, S Sehdev, N Spain, AT Prevost, WD-C Man

**10.05am** **S12**

Improving safety and TReatment precision for Acute COPD Exacerbations (TRACE): feasibility of point-of-care testing in community pharmacies. A qualitative study

TH Harries, R MacKenzie, M Bafadhel, R Russell, A Wright, H Patel, D Joshi, L Hamilton, PT White

**8.45am-10.20am**
**Moore, 4th floor**
**SPOKEN SESSION: S13-S18**

## Approaches to predict survival and increased mortality risk in ILD

*Chaired by: Dr Fasihul Khan (Leicester) and Dr Gauri Saini (Cambridge)*

**8.50am** **S13**
Quantitative CT is associated with progression free survival in idiopathic pulmonary fibrosis and fibrotic hypersensitivity pneumonitis
G Dixon, HI Adamali, J Hardy, M Wells, S Mulholland, G Ruffino, P Croxford, N Kennedy, H Emms, T Hartley, W Bou-Zeid, C Rennison-Jones, A Devaraj, PM George, P Rivera-Ortega, S Stanel, JCL Rodrigues, CJ Scotton, K Tsaneva-Atanasova, SL Barratt, MA Gibbons

**9.05am** **S14**
Absence of CT imaging in AEILD admissions associated with higher mortality risk
L White, J Shaw, B Powell, NM Kyi, R Huang, E Hardy, G Hughes, D Tilakaratne, C Hayton, T Raj, V Truong, N Ismail, N Khanijoun, A Sou, G Ng Man Kwong, A Gadoud, T Gatheral

**9.20am** **S15**
The role of quantitative CT in the prognostication and monitoring of systemic autoimmune rheumatic disease related interstitial lung disease
G Dixon, HI Adamali, J Hardy, M Wells, H Gunarwardena, J Pauling, S Mulholland, G Ruffino, P Croxford, A Heaps, N Kennedy, H Emms, T Hartley, E Bussell, R Keegan, S Bishop, M Thillai, S Walsh, P Rivera-Ortega, S Stanel, MA Gibbons, JCL Rodrigues, CJ Scotton, K Tsaneva-Atanasova, SL Barratt

**9.35am** **S16**
Impact of hospitalisation in ILD subtypes and predictors of outcome
E Moran, M Ashfaq, P Gorman, L Macpherson, E Murtagh, P Minnis

**9.50am** **S17**
Low serum testosterone predicts survival in patients in fibrotic interstitial lung disease: longitudinal follow-up of the STARSHIP cohort
SE Goodrum, C Crook, S Lines, P Rivera-Ortega, MA Gibbons, CJ Scotton, A Duckworth

**10.05am** **S18**
Streptococcus is enriched in the gut microbiome in idiopathic pulmonary fibrosis and associated with worse survival
DJF Smith, NMY Teng, Z Wu, L Yazbeck, F Chua, V Domi, W Han Liu, C McCann, A Nelson, K Nguyen-Phuoc, SA Walker, DL Smith, CM Lloyd, PL Molyneaux

**8.45am-10.20am**
**Rutherford, 4th floor**
**SPOKEN SESSION: S19-S24**

## Sleep matters

*Chaired by: Dr Alanna Hare (London) and Dr Lizzie Hill (Bristol)*

**8.50am** **S19**
Impact of virtual CPAP set-up on long-term compliance
N Haq, S Imaan, M Latham, D Ghosh

**9.05am** **S20**
From clinic to click – digital obstructive sleep apnoea identifier pathway
L Piggott, R Murray, E McNally, O Gavin, A Brennan, P Coss, B Kennedy, BD Kent

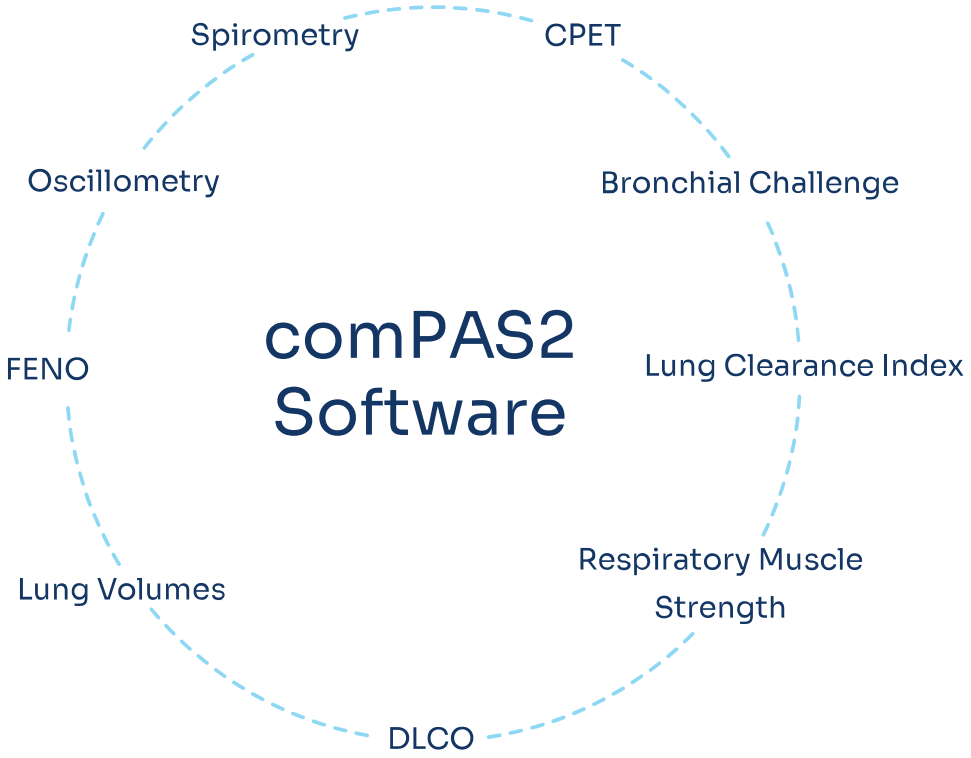
**9.20am** **S21**
Postoperative outcomes in patients with obstructive sleep apnoea: a retrospective study comparing outcomes of bariatric surgery in black and white patients
A Sadoun, R Brown, J Steier

**9.35am** **S22**
Interdisciplinary discussion of non-continuous positive airway pressure therapies for obstructive sleep apnoea: a retrospective review of difficult-to-treat cases
OSH Ho, R Brown, J Steier

**9.50am** **S23**
Obstructive sleep apnoea and metabolic syndrome in adults: evidence from case-control studies of a systematic review and meta-analysis
S Pant, I Kyrou, L Lagojda, C Kite, A Dallaway, A Dhungana, H Singh Randeva, O Prakash Kurmi

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**10.05am** **S24**
Predicting driver sleepiness in OSAHS: towards a screening tool for road safety
A Kumar, S Craig, B Chakrabarti, A Manuel, G Phillips

**8.45am-10.20am**
**Albert, 2nd floor**
**SPOKEN SESSION: S25-S30**

## Physiological approaches to decoding breathlessness

*Chaired by: Dr Emily Fraser (Oxford) and Dr Sanja Stanojevic (Halifax, Canada)*

**8.50am** **S25**
Is ageing associated with exertional ventilatory constraint?
G Cenerini, Z Williams, F Schwendinger, R Knaier, J Wagner, JH Hull, A Schmidt-Trucksäss

**9.05am** **S26**
Evaluation of an automated software application versus clinician interpretation of cardiopulmonary exercise tests
CJ Harding, KP Sylvester, F Clarkson, JP Fuld, BK Knox-Brown, CB Cooper, S Mobus

**9.20am** **S27**
The use of cardiopulmonary exercise tests in objectively diagnosing dysfunctional breathing
VC Moore, A Fisk, J Shakespeare, ED Parkes

**9.35am** **S28**
Factors associated with breathlessness and dysregulated breathing post-COVID-19 amongst referrals to a respiratory clinic
M Hamrouni, S Prosper, S Middleton, T Harvey-Dunstan, J Porte, A Gupta, IP Hall, TM McKeever, CE Bolton

**9.50am** **S29**
Peripheral muscle oxygen extraction is compromised during submaximal exercise in previously hospitalized COVID-19 survivors
S Chakraverty, D Megaritis, E Hume, E Neto, G Cucato, E Daynes, R Evans, S Singh, C Echevarria, I Vogiatzis

**10.05am** **S30**
Oxygen requirement rises earlier than the total National Early Warning Score 2 (NEWS2) score prior to deterioration
CH Harrison, S Gerry, OC Redfern, C Subbe, JP Bedford, PJ Watkinson

**9.15am-10.15am**
**Abbey, 4th floor**
**OPEN SESSION**

## Supporting women in respiratory medicine

*Chaired by: Professor Robina Coker (London) and Dr Hitasha Rupani (Southampton)*

*Speakers: Dr Sundari Ampikaipakan (Norwich), Dr Rachael Moses OBE (Newcastle upon Tyne) and Dr Hilary Tedd (Newcastle upon Tyne)*
*This is an inclusive session – all delegates are welcome. The purpose of this workstream is to support equity of opportunity across the full multi-professional respiratory team.*
*We will host an interactive discussion about routes into leadership roles, what resources are available, and the potential barriers and how to overcome them. No matter where you are in your career, please join us to share your thoughts and experiences.*

**10.00am-11.00am**
**Whittle & Fleming and Britten, 3rd floor**

## COFFEE/TEA BREAK and visit the exhibition stands

**10.45am-12.15pm**
**St James, 4th floor**
**SYMPOSIUM**

## OPTIMISING SLEEP DISORDERED BREATHING: SMART APPROACHES FOR BETTER OUTCOMES

*Chaired by: Dr Sriram Iyer (Sheffield) and Dr Karl Sylvester (Cambridge)*

**10.45am** Hypoxic burden: is it ready for clinical risk prediction?
Professor Manuel Sánchez de la Torre (Lleida, Spain)

**11.15am** Could drugs really make CPAP redundant?
Professor Jan Hedner (Gothenburg)

**11.45am** Sleep disordered breathing in people with intellectual disability
Dr Lizzie Hill (Bristol)

*Learning objectives*

*1) To explore the role of hypoxic burden in cardiovascular risk prediction in OSA.*

*2) To be briefed on the latest evidence for newly developed drugs in OSA, as a basis for considering the potential for drugs to play a significant role in managing this condition.*

*3) To discuss the challenges and benefits of using CPAP to treat OSA in patients with intellectual disability.*

**10.45am-12.15pm**
**Churchill, ground floor**
**SYMPOSIUM**

## PIECING TOGETHER THE PERSONALISATION PUZZLE – IS PR THE MISSING PIECE?

*Chaired by: Dr Gillian Doe (Leicester) and Professor Nicola Roberts (Edinburgh)*

**10.45am** How can we personalise PR? What components are important?
Dr Tom Ward (Leicester)

**11.15am** Personalisation of asthma treatment and management: where does the PR come in?
Mrs Lizzie Grillo (London)

**11.45am** How can PR contribute to the personalisation of treatment for ILD?
Dr Kerri Aronson (New York)

*Learning objectives*

*1) Will highlight the importance and impact of pulmonary rehabilitation in the management of respiratory conditions and the role of personalisation.*

*2) Illustrate the use of PR as part of personalising asthma treatment and management.*

*3) Illustrate the use of PR as part of personalising ILD treatment and management.*

**10.45am-12.15pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## CHRONIC COUGH AS A NEURONAL DISORDER

*Chaired by: Dr Paul Marsden (Manchester) and Ms Katie Rhatigan (London)*

**10.45am** Chronic cough and the role of airway nerves
Professor Matthew Drake (Oregon)

**11.15am** Triggers of cough and airway nerves: new targets and treatments
Dr Sean Parker (Northumbria)

**11.45am** Sensations in cough and the larynx
Mrs Jemma Haines MBE (Manchester)

*Learning objectives*

*1) To describe the role and importance of airway nerves in chronic cough. With focus on genetics and nerves, interaction with airway inflammation, viruses and nerve hypersensitivity.*

*2) To highlight and summarise current research. Translating neural molecular targets into antitussive therapy.*

*3) To highlight and summarise the role of the upper airway, laryngeal anatomy and neural innervation.*

**10.45am-12.20pm**
**Moore, 4th floor**
**SPOKEN SESSION: S31-S36**

## Optimising diagnostics in thoracic malignancy

*Chaired by: Professor Gerard Silvestri (Charlestown) and Dr Alexandra Teagle (Edinburgh)*

**10.50am** **S31**
Early insights into the data from the Artificial Intelligence and Big Data for Early Lung Cancer Diagnosis Prospective Study (Phase 2) (IDEAL study)
KL Ng, P Dhiman, JA Moreland, W Hickes, ML Szkonter, N Rahman, T Barton, M Callister, D Baldwin, FV Gleeson

**11.05am** **S32**
Clinical readiness of AI tumour volume quantification in mesothelioma: results of the Meso-Q study
JJ Roche, A Thu, A MacPherson, AC Kidd, M Neilly, J Pearson, H Stubbs, S Tsim, V Panou, K Atefeh, A Beattie, G Cowell, J Davies, D Fennel, A Gill, D Kim, A Laws, N Maskell, S Padley, N Screaton, R Insall, C Miller, F Gleeson, K Blyth

**11.20am** **S33**
Harnessing exhaled breath for lung cancer early detection. The ExPeL study results
SB Knight, D Patel, L D'Cruz, A Chauhan, S Grundy, N Diar-Bakerly, W Ahmed, D Trivedi

**11.35am** **S34**
Does profiling pleural fluid circulating tumour DNA (ctDNA) have a role in improving diagnostic yield in malignant pleural effusion?
A Baguneid, R Allsopp, K Page, R Sudhir, D Fennell, J Shaw, R Panchal

**11.50am** **S35**
REducing repeat PLeural biopsies In suspeCted mesotheliomA (REPLICA)
GA Lynch, S Walker, N Bhatt, J Symonds, J Potts, C Wragg, K Blyth, H Steer, A Ionescu, E Mishra, N Rahman, S Khan, DT Arnold, D De Fonseka, P White

**12.05pm** **S36**
The STREAMLINE feasibility randomised clinical trial (early pleural biopsy + indwelling pleural catheter in suspected malignant pleural effusion)
DN Addala, C Daneshvar, S Khan, E Bedawi, J Baxter, D de Fonseka, E Daly, B Iqbal, A Elsheikh, A Sundaralingam, J Sadler-Ladell, R Miller, R Hallifax, NM Rahman

**10.45am-12.20pm**
**Abbey, 4th floor**
**SPOKEN SESSION: S37-S42**

## Bronchiectasis: clinical trials and real world impact

*Chaired by: Dr Emma Johnson (Dundee) and Dr Harm Tiddens (Rotterdam)*

**10.50am** **S37**
Molecular detection and quantification of Pseudomonas aeruginosa in bronchiectasis: data from the GREAT-2 randomized controlled trial
D Alferes de Lima Headley, AJ Bell, R Hull, MB Long, J Stobo, P Emanuel, H Buchanan, E Mountjoy, D Gordon, C Hennayake, Q Du, R Galloway, E McIntosh, Z Eke, M Tunney, K Cartwright, R Hughes, A Di Giandomenico, MG Belvisi, W Brailsford, R Dakin, R Holmes, C Clarke, C Haworth, JD Chalmers

**11.05am** **S38**
Effects of Brensocatib on neutrophil serine protease levels in patients with non-cystic fibrosis bronchiectasis: a pharmacodynamic analysis of the ASPEN Trial
ML Metersky, A De Soyza, P-R Burgel, CL Daley, D Mauger, CS Haworth, A Teper, C Fernandez, K Mange, C Fan, X Zhang, JD Chalmers

**11.20am** **S39**
Polypharmacy in patients with non-cystic fibrosis bronchiectasis: the BRONCHUK observational study
H Upadhyay, A De Soyza, R Watson, P Mawson, T Shield, M Kelly, S Elborn, A Hill, T Gatheral, A Sullivan, C Haworth, J Hurst, J Brown, M Carroll, MR Loebinger, J Bradley, P Walker, J Steer, J Duckers, J Chalmers, R McNally

**11.35am** **S40**
Bronchiectasis and multimorbidity: a data analysis of the BronchUK Registry to determine the associations between multimorbidity and patient outcomes
A De Soyza

**11.50am** **S41**
Morbidity in patients with NCFBE and exacerbations in the THIN UK database
M Loebinger, J Quint, D Gor, J Feliciano, I Ghout, D Acosta-Mena, A Callet, C Renaudat

**12.05pm** **S42**
The resource impact of bronchiectasis and associated exacerbations: based on a 5-year prospective observational cohort study (BronchUK)
C Gallego-Moll, L Maynou, P Mawson, T Shield, M Kelly, S Elborn, A Hill, T Gatheral, A Sullivan, C Haworth, J Hurst, J Brown, M Carroll, M Loebinger, J Bradley, P Walker, J Steer, J Duckers, J Chalmers, R McNally, A De Soyza, A McGuire

# Wednesday 26 November 2025

**10.45am-12.20pm**
**Rutherford, 4th floor**
**SPOKEN SESSION: S43-S48**

## Paediatric asthma: are we getting it right?

*Chaired by: Professor Louise Fleming (London) and Dr Prasad Nagakumar (Birmingham)*

**10.50am** **S43**
Longitudinal trends in asthma mortality and life-threatening asthma in children in England
A Gawlik-Lipinski, S Shabnam, JK Quint, CL Gillies, DKH Lo

**11.05am** **S44**
Are children admitted with near-fatal asthma receiving the core elements of care at discharge? Results from NRAP, the National Respiratory Audit Programme
A Gawlik-Lipinski, A Adamson, R O'Beirne, P van Geffen, JK Quint, JW Dodd, T Wilkinson, I Sinha

**11.20am** **S45**
MART vs conventional therapy: impact on biomarkers, lung function and symptom control in children and young people with asthma
S Hay, K Hillson, H Almeida, B Pavlou, I Testa, Y Bingham, K Mayoral Oritz, S Lacbay, M Gore, E Paraskakis, H Tibble, S Irving, S Sonnappa, L Fleming, A Bush, S Saglani

**11.35am** **S46**
Evaluating recent changes to paediatric asthma diagnostic guidance from GINA and NICE
L Healy, AJ Simpson, R Wang, M Bennett, L Whipday, L Willmore, SJ Fowler, A Simpson, CS Murray

**11.50am** **S47**
The utility of blood eosinophils, IgE and skin prick testing in paediatric asthma diagnosis
CS Murray, L Healy, A Simpson, R Wang, M Bennett, S Fowler, A Simpson

**12.05pm** **S48**
Measurement of children and young people's asthma medication adherence by patient/family report, electronic monitoring devices and prescription refill rate
R Dobra, S Makhecha, R Moore-Crouch, C Jackman, P Hall, L Fleming

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**10.45am-12.35pm**
**Windsor, 5th floor**
**SPOKEN SESSION: S49-S55**

## Tracking the science: smarter asthma strategies

*Chaired by: Dr Martin Knolle (Cambridge) and Dr Ran Wang (Manchester)*

**10.50am** **S49**
GINA Strategy Report (2024) updates improve the sensitivity of asthma diagnosis by removing airflow limitation criterion in adults
AJ Simpson, L Healy , S Drake, R Wang, M Bennett, H Durrington, H Wardman, CS Marray, SJ Fowler, A Simpson

**11.05am** **S50**
Asthma untangled: standardising expert clinician judgement with a structured multi-modal approach
CM Gill, C Ottewill, PJ Kerr, V Brennan, O Smith, H Doherty, E MacHale, G Greene, D Ryan, B Cushen, RW Costello

**11.20am** **S51**
Multidimensional assessment in severe asthma: early findings from the Bristol Evaluation of Novel Airways Diagnostics, Therapies and Healthcare Outcomes (BREATHE) study
K Bansal, C Morgan, GW Nava, J Grenville, C Ryan, V Cobham, D Higbee, R Shrimanker, JW Dodd

**11.35am** **S52**
Agreement between frequent home-based or clinic-based monitoring of FeNO in asthma: a secondary analysis of the INFORM ASTHMA Trial
JH Noble, O Bean, R Sayers, R Cullen, ID Pavord, R Beasley, M Weatherall

**11.50am** **S53**
Hiding in plain sight: using BTS guidance to identify and optimise management of "asthma-ectasis" and ABPA through clinical phenotyping of a bronchiectasis cohort
E Nobes, J Ayling-Smith, C Addy

**12.05pm** **S54**
Predictors of response to biologic therapy in a UK severe asthma population
PA Shah, K Ntotsis, F Ardesi, MF Liew, R Tan, C Boddy, A Murphy, D Shaw, P Bradding, R Green, S Naveed, S Diver, CE Brightling

**12.20pm** **S55**
Bacterial immunotherapy for the prevention of exacerbations in patients with chronic respiratory diseases
EJ Soto Hurtado, Y Rodríguez Gallego, E Salcedo Lobera

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**11.00am-12.00pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Pulmonary Embolism and other Pulmonary Vascular Diseases

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**11.00am-12.00pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Tobacco Dependency

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**11.00am-1.00pm**
**Westminster, 4th floor**
**JOINT BTS/BALR SYMPOSIUM PART 2**

## AT THE CUTTING EDGE OF MODELS FOR LUNG DISEASE

*Chaired by: Ms Emily Cash (Nottingham) and Professor Karl Staples (Southampton)*

**11.00am** What human lung tissue tells us about lung disease
Dr Tillie-Louise Hackett (Vancouver)

**11.40am** The use of ex vivo lung models of fibrosis to evaluate novel treatments
Dr Katy Roach (Leicester)

**12.20pm** Using air-liquid interface cultures to understand respiratory viral infections
Dr Claire Smith (London)

*Learning objectives*

*1) To give an overview of how whole human lung tissue has transformed our understanding of the pathobiology of lung disease.*

*2) To highlight the understanding of pulmonary fibrosis and how to treat this disease emerging from ex vivo human lung cultures.*

*3) To demonstrate the use of ALI culture models to explore the mechanisms of respiratory viral infection of the human airway.*

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**12.00pm-2.15pm**
**Pickwick, 1st floor and Whittle & Fleming, 3rd floor**

## LUNCH and visit the exhibition stands

*(Lunch is not included in the delegate fee but is available to purchase on site. Card payments only)*

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**12.30pm-1.30pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Pulmonary Rehabilitation

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**12.45pm-1.45pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Pleural Disease

*All participants are welcome to attend and hear more about the work of the SAGs.*

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# Wednesday 26 November 2025

**1.15pm-2.00pm**
**Churchill, ground floor**
**BTS SCIENTIFIC GUEST LECTURE**

## UNDERSTANDING IMMUNE REGULATION OF THE LUNG AND WHAT IT MEANS FOR LUNG HEALTH

*Introduced by: Professor Richard Russell (London)*

*Guest Lecturer: Professor Tracy Hussell (Manchester)*

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**2.00pm-3.00pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Bronchiectasis

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**2.00pm-3.00pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Cough

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**2.05pm-4.05pm**
**Cambridge, 5th floor**
**MODERATED POSTER DISCUSSION: M1-M16**

## Impact of treatment on COPD outcomes: from smoking to biologics

*Chaired by: Dr Amy Dewar (London) and Dr Thomas Ward (Leicester)*

**M1** How vaping chemicals compromise lung surfactant
K Lutchman Singh, Z Zhou, J Bernardino de la Serna

**M2** Is the messaging about staff vaping consistent in NHS organisations across the UK?
E Blowers, M Nuttall, NJ Roberts

**M3** Supporting 'Swap to Stop' smoking cessation: a pilot trial of vape information provision within lung cancer screening
O Maynard, J Atkinson, J De Oliveira, E Buckley, J Frost, V Carlisle, T Stone, A Attwood, A Bibby

**M4** Multimodal evaluation of AI-assisted approaches to smoking cessation
JP Dexter

**M5** Smoking cessation provision and outcomes following initiation of a new smoking cessation service alongside an urgent suspect lung cancer clinic
KY Goh, T Wilson, S Butcher, N Clarkson-Sheahan, N Henderson, C Facer, K Gracie, MPT Kennedy, E Paramasivam, J Robson, K Rodger, MEJ Callister

**M6** Make every contact count – opportunistic lung cancer screening and smoking cessation at a community diagnostic centre
L Stoodley, P De Sousa, AH Nickol, C Turnbull, JE Park

**M7** 5-year outcomes of LungCast: "Smoking Cessation in Lung Cancer Study"
M Dunning, M Smith, A Bawuagh, J Edefo, KE Lewis

**M8** Tailoring cystisine's dosage in smokers with comorbidities and unsuccessful previous efforts
V Petta, G Poupouzas, N Athanasiou, K Thomas, V Ntoumou, K Dede, A Korkontzelou, Z Barbaressou, A Osarogue, T Georgiadis, P Katsaounou

**M9** Do smokers requiring secondary care services engage with community-based smoking cessation support?
N Hutchinson, HW Poole

**M10** Evaluation of participant experience in the NIHR HTA OPACE (Optimising Azithromycin prevention treatment in COPD to reduce Exacerbations) trial – a pragmatic trial of continued vs discontinued azithromycin prophylaxis in COPD
M Fisk, G Adesoye, Z Yilmaz, G Barrett, A Watson, F Early, J Fuld, F Frost, D Anderson, S Hughes, R Singh, E Jacobs, L Kabalan, G Davies, A Ionita, E Beranova, M Anwar, P Nabayego, A de-Soyza, C Bolton, D Price, IB Wilkinson

**M11** Real-life outcomes of patients with COPD initiating budesonide/glycopyrronium/formoterol after a previous dual therapy in Spain. ORESTES study
C Gotera, J González, A Herrejón, A Pallarés, S Pascual, A Saura, C Corregidor, E Fitas, J Sánchez-Covisa, B Alcázar-Navarrete, JM Figueira-Gonçalves

After 3 years, 47.7% of CPAP patients discontinued treatment[1]

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# Inspire, breath-synchronised hypoglossal nerve stimulation therapy - Designed to improve your OSA patients' long term quality of life[2,3,4]




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|---|---|---|---|
| 80% success rate[6].* | > 350 publications. | > 100,000 patients. | 91% patient satisfaction[7]. |

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**1** Database analysis; Pépin JL, Bailly S, Rinder P et al. J Clin Med. 2021;10(5):936. **2** Strollo et al. NEJM 2015. **3** Woodson BT et al, Otolaryngol Head Neck Surg. 2016. **4** Woodson et al, OtoHNS 2018. **5** Woodson, BT, Strohl, K P, Soose, R J et al. Upper Airway Stimulation for Obstructive Sleep Apnea: 5-Year Outcomes. Otolaryngology–Head and Neck Surgery 2018; 159(1):194–202. **6** Kim et al, OtoHNS 2023. **7** Suurna et al. Laryngoscope 2021.

* success rate = 80 % met the Sher criteria (AHI reduction by 50 % and < 20 events per hour) within 12 months. In addition, approximately 82 % of patients achieved an AHI < 15 at 12 months.

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**M12** Real-world disease control and stability in patients with COPD treated with Extrafine Triple Therapy with Beclometasone Dipropionate, Formoterol, and Glycopyrronium
G Brusselle, P Bakakos, D Stolz, L Di Palma, R Pizzoccheri, U Scaffidi Argentina, A Piraino, L Franzini, P Rogliani

**M13** Antibiotic and systemic corticosteroid use in patients with chronic obstructive pulmonary disease and type 2 inflammation receiving dupilumab
M Bafadhel, A Anzueto, A Papi, SP Bhatt, S Korn, I Satia, G Deslée, R Thomas, C Xia, J Heble, M Soliman

**M14** Association between early lung function improvement with dupilumab and sustained clinical outcomes in patients with chronic obstructive pulmonary disease: a clinical perspective
FME Franssen, NA Hanania, N Roche, R Chen, WW Labaki, NN Hansel, G Deslée, R Wysoczanski, C Xia, B Ferey, M Soliman

**M15** Dupilumab efficacy across baseline eosinophil counts in patients with chronic obstructive pulmonary disease with type 2 inflammation
SA Christenson, FC Sciurba, CF Vogelmeier, M Miravitlles, JR Hurst, F Wen, FJ Martinez, P Saha-Chaudhuri, M Soliman, J Heble

**M16** Treatment failures during an eosinophilic exacerbation following treatment with benralizumab or prednisolone: data from ABRA
N Smallcombe, M Zheng, S Ramakrishnan, S Cass, C Mwasuku, K Krassowska, L Bermejo-Sanchez, REK Russell, M Bafadhel

**2.15pm-3.45pm**
**Churchill, ground floor**
**SYMPOSIUM**

## UPDATES AND ADVANCES IN PLEURAL EFFUSIONS

*Dr Helen Davies (Cardiff) and*
*Dr Beth Sage (Highland and Aberdeen)*

**2.15pm** Epigenomics in pleural effusions
Dr David Arnold (Bristol)

**2.45pm** A deep dive into non-specific pleuritis
Dr Katie Ferguson (Dundee)

**3.15pm** Do Light's Criteria stand the test of time?
Dr Samira Shojaee (Nashville)

*Learning objectives*

*1) Elucidate how basic science approaches may help us better understand the biology of malignant pleural disease and therefore identify new therapeutic targets.*

*2) Provide an early update on ongoing large-scale trials which aim to understand the significance of so-called benign pleural disease, and explain how these studies may help identify time points for further intervention and allow improved prognostication.*

*3) Provide an insight on new developments on the sensitivity, specificity and application of Light's Criteria and its use in modern medicine.*

**2.15pm-3.45pm**
**Windsor, 5th floor**
**SYMPOSIUM: T1-T6**

## JOINT BTS/BALR/A+LUK EARLY CAREER INVESTIGATOR OF THE YEAR SYMPOSIUM

*Chaired by: Professor Nick Maskell (Bristol)*

*Judged by: Professor Mona Bafadhel (London), Professor Philip Molyneaux (London) and Professor Karl Staples (Southampton)*

**2.15pm** **T1**
Retrospective evaluation of Artificial Intelligence (AI) software for the detection of acute Pulmonary Embolism (PE) in CT Pulmonary Angiograms (CTPA): a preliminary ground-truth analysis from the AID-PE study
JW Page, SGS Gunning, PFP Charters, S Lyen, B Hudson, J Rossdale, A Seatter, R Mackenzie-Ross, J Suntharalingam, JCL Rodrigues

**2.30pm** **T2**
Haemophilus commensals within the airway microbiome modulate immune responses and dictate viral susceptibility in COPD
MM Jackson, O Katsoulis, M Kusumoto, C Chen, K Sanghavi, J Aniscenko, P Mallia, KT Mincham, A Decout, SL Johnston, TB Clarke, RJ Snelgrove, A Singanayagam

**2.45pm** **T3**
Lung eosinophils in eosinophilic COPD patients differ functionally but not phenotypically

SP Cass, JR Baker, REK Russell, M Bafadhel

**3.00pm** **T4**

Sex differences in asthma: a large-scale multi-omics study

J Shen, E Marchi, N Zounemat-Kermani, S-E Dahlen, I Adcock, T Hinks

**3.15pm** **T5**

Reduced airway remodelling and increased epithelial barrier integrity in bronchial biopsies from patients with T2 high severe asthma following mepolizumab treatment

I Drummond, J Phillips, R Dhillon, A Eminton, J Naftel, W Soe, P Dennison, RT Martinez-Nunez, L Lau, J Ward, C Blume, EJ Swindle, H Rupani

**3.30pm** **T6**

Genetic characterisation of Pseudomonas aeruginosa isolates from bronchiectasis patients in a randomized, double-blind placebo-controlled trial of a bispecific monoclonal antibody targeting Psl and PcrV (GREAT-2)

RC Hull, MB Long, A Gilmour, I Petrou, R Galloway, K Viligorska, H Richardson, JM New, C Hennayake, E Johnson, E McIntosh, Z Eke, M Band, H Bandara, G Martin, S Aliberti, M Vendrell, E Polverino, G Suarez-Cuartin, MR Loebinger, M Tunney, K Cartwright, R Hughes, A DiGiandomenico, MG Belvisi, W Brailsford, C Haworth, O Sibila, J Stobo, JD Chalmers

**2.15pm-4.05pm**
**St James, 4th floor**
**POSTER DISCUSSION: P1-P15**

## From primary to secondary care: evolving management of COPD

*Chaired by: Dr Cara Flynn (Leicester) and Dr Rama Vancheeswaran (Oxford)*

**P1** Current practices, perceptions and future challenges in COPD management

J Lim, H O'Brien, J McCarthy, C McCarthy, A Franciosi

**P2** Disease stability, management and treatment goals in chronic obstructive pulmonary disease among newly diagnosed vs long-term diagnosed patients: a patient survey

D Halpin, A Papi, T Mohan, A Majumdar, M Al-Ameri

**P3** Integrating AI-assisted spirometry: validation and user experience in a COPD diagnostic pathway

E Giffen, D Carroll, E Smets, K Van Orshoven, M Topalovic, E Walker, A Taylor, DJ Lowe, C Carlin

**P4** Multidimensional classification for severity of exacerbations of COPD: the COPD exacerbation BAT (Baseline, Acuity, and Trigger) conceptual model

H Aung, A Beersaerts, K Vermeersch, HJ Mcauley, C Flynn, S Ramakrishnan, M Abdo, SP Cass, I Gyselinck, H Pott, TJ Ward, A Wright, CE Brightling, PR Burgel, M Contoli, FM Franssen, A Huerta, JK Quint, B Schmeck, LE Vanfleteren, B Waschki, H Watz, M Bafadhel, W Janssens, NJ Greening

**P5** Impact of pharmacotherapy with inhaled corticosteroids and long-acting beta-2 agonists on symptom control and oscillometry parameters among Indian patients with early COPD due to smoking and non-smoking causes: Indian Pre COPD Assessment Network (IeCAN) study

R Dhar, R Swarnakar, T Kulkarni, D Christopher, S Madas, C Singh, S Singh, A Mohan, S Moitra, P Koul, R Chawla, SN Gupta, A Singh, V Das, A Nayak, A Sabale, R Kadam, S Salvi, M Sovani

**P6** Relative lung availability and total systemic exposure after inhalation of medium or high strength doses of beclometasone dipropionate/formoterol fumarate, formulated as pMDI with a next generation propellant

M Almeida, M Salvadori, M Corradi, T De Ridder, N Praet, I Viaud, G Varoli, L Girardello, F Sandri, G Cappellini, LV Viganò, KS Mathews, G Poli

**P7** Sustainable care: what people with COPD are offered a lower carbon inhaler device and who is more likely to succeed in switching?

L Piggott, A Francis, A Cumella, S Walker, BD Kent, G d'Ancona

**P8** Real-world study on causes of death in people with Chronic Obstructive Pulmonary Disease (COPD) in England

H Mullerova, N Roche, M Cheng, NC Hazra, K Gallant, S Dods, H Chen, M Cunnington, C Xu, A Wolf, CSP Lam, MBBafadhel, M Pollack

**P9** Advanced Clinical Practitioner (ACP) hot airways clinic: proof of concept to assess demand and outcomes of Eosinophilic Airways Disease (EAD)
ER Bradley, R Baron, SO Brij, WA Khan

**P10** Breathless systems: COPD pressures in hospitals across England
REK Russell, M Bafadhel, C Lupton, M Watt, C Thomas

**P11** COPD and respiratory viral infection in hospitalised adults: longer symptom duration, blunted perception, and poor early outcomes
T Morelli, A Freeman, A Jones, T Wilkinson

**P12** Characteristics and outcomes of patients with COPD and acute hypercapnic respiratory failure (AHRF) admitted to respiratory support unit (RSU)
T Htwe, A Hooi Tee, S Lutchegadoo, K Anigboro, D Sagar

**P13** Seasonal variation in exacerbation risk following severe AECOPD
CA Flynn, HJC McAuley, O Elneima, HWW Aung, W Ibrahim, TJC Ward, M Bourne, T Thornton, RA Evans, MC Steiner, AKA Wright, CE Brightling, NJ Greening

**P14** Under pressure: patient characteristics associated with primary care respiratory treatment reviews before respiratory winter pressures in the UK
PE Pfeffer, MJ Hale, M Watt, K Shah, JG Munday, A Murphy

**P15** Understanding increased hospitalisations in INTEGR-COPD: a secondary analysis of admission data
K Varma, LA Boast, K Patel, AM Turner

**2.15pm-4.05pm**
**Moore, 4th floor**
**POSTER DISCUSSION: P16-P30**

## Monitoring, managing and treating ILD to improve outcomes

*Chaired by: Professor Bibek Gooptu (Leicester) and Dr Amanda Tatler (Nottingham)*

**P16** A national survey mapping the provision of supportive and palliative care services for interstitial lung disease patients across the United Kingdom
LE Crowley, S Quarton, J Tomas, M Bird, S Bajwah, A Boland, A Crawshaw

**P17** Impact of virtual nurse/pharmacist-led monitoring in patients with interstitial lung disease (ILD) on antifibrotic treatment in a UK ILD specialist centre
SJ Lines, C Crook, B Carballido-Romero, V Elworthy, J Mandizha, B Mitchell, AR Paiva, L Rowland, H Serjeant, R Davies, J Moss, K Taylor, A Duckworth, A Stockley, SC Stanel, MA Gibbons, P Rivera-Ortega

**P18** Implementation of regional interstitial lung disease multidisciplinary meetings between tertiary and secondary care
N Chen, K Brignall, R Boorsma, K Osborne, K Myall, A West

**P19** Seasonal patterns of interstitial lung disease hospitalisations in a UK tertiary care centre
A Vapiwala, A Manga, F Khan

**P20** How dietetic interventions arrest weight loss in malnourished patients with fibrotic Interstitial Lung Disease (ILD)
R Kahai, G Castelli, F Danzo, L Ferreira, F Mirabelli, M Morviducci, A Srirangan, C Roberts, C Stock, S Bax, R Hewitt, M Kokosi, P George, F Chua, V Kouranos, P Molyneux, R Jenkins, W Banya, G Korff, A Wells, A Duncan, P Sestini, E Renzoni

**P21** Impact of treatment on quality-of-life measures in sarcoidosis
R Doherty, I Bhalwel, A Mahmood, P Minnis

**P22** Progression patterns in nintedanib-treated individuals with progressive pulmonary fibrosis (PPF)
S Bax, K Raveendran, B Vitri, S Boreland, C Hogben, E Renzoni, R Hewitt, A Devaraj , V Kouranos, P George, M Kokosi, G Jenkins , S Desai, AU Wells, PL Molyneaux, F Chua

**P23** Nintedanib in systemic sclerosis-associated interstitial lung disease: real-world multicenter cohort study on tolerability and discontinuation
P Kaenmuang, NR Goldman, A Srirangan, F Danzo, W-H Yip, A Taki, S Boreland, B Vitri, CJW Stock, CP Denton, AU Wells, VH Ong, P Sestini, EA Renzoni

**P24** Comparison of Nintedanib outcomes in Rheumatoid Arthritis associated Interstitial Lung Disease (RA-ILD) and Idiopathic Pulmonary Fibrosis (IPF)
VJF de Braal, CJ Gracie, S Poole, P Saunders, KEN Clark

**P25** Impact of tiered service delivery model on antifibrotic adherence and mortality among patients with interstitial lung disease
F Fyles, A McKinnon, S Agnew, J Sutcliffe, S Brooks, J Barnett, S McGarvie, A Simons, A Kwok, V Yioe, LG Spencer

**P26** Real-world evaluation of patient characteristics impacting tolerability of antifibrotic treatment in interstitial lung diseases
H Saqib, S Rizvi, Z Rashid, Z Khan, AC Murphy, F Khan

**P27** Can antifibrotic side-effect decision aids improve patient tolerance and continuation of antifibrotic medication?
J Bradley, J Slough, T Capstick, A Boland, TJT Sutherland, P Beirne

**P28** LITTLE WOMEN Looking Into anTifibrotic ToLerancE in relation to Weight, dOse, feMalE gender and EthNicity
S Marguerie, A Alkhdour, I Adhikari, K Amcha, L Elton-Walters, MG Jones, RM Limbrey, T Wallis, BG Marshall, KM Spinks, SV Fletcher

**P29** Efficacy of pulsed Cyclophosphamide in interstitial lung disease
W Soe, M Adil Irshad, F Thompson, A Donaldson , M Ball, S Ramachandraiah, V Titmuss, L Newman, A Wallis, S Babu

**P30** Effect of Dietary Nitrate Supplementation on Exercise Performance in Hypoxic IPF: the EDEN-Ox 3 Study
M Majrshi, A Alzahrani, P George, K Philip, A Alsulayyim, L Price, M Polkey, N Hopkinson

**2.15pm-4.05pm**
**Abbey, 4th floor**
**POSTER DISCUSSION: P31-P45**

## Enhancing outcomes in chronic respiratory disease: the role of rehabilitation and physiotherapy

*Chaired by: Ms Maria Buxton (London) and Dr Lorna Latimer (Leicester)*

**P31** A qualitative exploration of the perception of maintenance needs three months after rehabilitation in patients with long COVID
K Kontou, A Hong, H Drover, A Barradell, E Chaplin, E Daynes, NA Bateman, J Creese, L Houchen-Wolloff

**P32** Predicting enrolment to pulmonary rehabilitation in the UK
H Drover, L Wang, MW Orme, M Richardson, JK Quint, R Evans, SJ Singh, E Daynes

**P33** The use of lay health workers in supporting patients with COPD to attend pulmonary rehabilitation: a qualitative study exploring the patient experience
R Mackenzie, G Gilworth, E Godfrey, A Spinou, T Morgan, K Harris, J Kawalek, N King, B Odin, S Lewin, SJC Taylor, NS Hopkinson, J Fox-Rushby, S Ayis, PT White

**P34** Reflections from pulmonary rehabilitation (PR) services who have achieved PRSAS accreditation
NJ Roberts, C Yim, E Daynes, M Buxton

**P35** The impact of pulmonary rehabilitation services accreditation scheme on key performance indicators
E Daynes, C Yim, M Buxton

**P36** Improving the uptake and completion of pulmonary rehabilitation (PR): development and evaluation of a train-the-trainer programme to establish a lay health worker service
A Spinou, R Daryanani, G Gilworth, E Godfrey, K Harris, N King, J Kawalek, T Morgan, SA Lewin, P White

**P37** Hypertonic saline in chronic lung disease: a local experience to suggest usefulness?
J Johnson, N Ahmad

**P38** Patient, family member, and clinician experiences of physiotherapy airway clearance techniques during hospital admission: a scoping review
K Jerrard, R Dowding, B O'Neill, JM Bradley, B Connolly

**P39** Survey of North Cumbria GP practices management of bronchiectasis patients with hypersecretion and perceived barriers to referral for respiratory physiotherapy
P McCallion, A Burgess

**P40** The use and perceptions of behaviour change interventions among people with chronic respiratory disease and those who deliver physical activity programmes in The Republic of Ireland
C Hanrahan, J Broderick, TMO Connor, T Troosters, JG McVeigh

**P41** The number and location of atelectatic changes on chest CT are associated with increased mortality in patients with COPD using long-term NIV

M Injety, N Mostapha, B Csoma, A Chai, E Croft, A Bentley, A Bikov

**P42** An experimental study to analyse the effect of respiratory proprioceptive neuromuscular facilitation techniques along with conventional physiotherapy in improving oxygen saturation, respiratory rate and heart rate in COVID-19 patients admitted in ICU
B Poozhikunnath, S Raju

**P43** Impact of integrated pulmonary and cardiac rehabilitation: a holistic approach to patient care in a 10-week pilot study
S Murphy, K Howkins, R Holehouse, S Marshall, L Clarke, M Melville

**P44** Evaluate the impact of pulmonary rehabilitation on static knee extensor muscle strength through a handheld dynamometer in respiratory patients – a community approach
M Tortorella, A Grant, C Caneja

**P45** Does pulmonary rehabilitation (PR) lead to improvement in estimated 1 repetition (1RM) of a loaded squat pattern, and correlate with improvements in quadriceps strength measured on an estimated 1RM knee extension and incremental shuttle walk test distance? A small cohort review
LM Graham, T Sweetser, J John, A Kenward

**2.15pm-4.05pm**
**Rutherford, 4th floor**
**POSTER DISCUSSION: P46-P60**

## Advances in sleep medicine

*Chaired by: Dr Akshay Dwarakanath (Wakefield) and Dr Swapna Mandal (London)*

**P46** A remote digital pathway for the management of mild obstructive sleep apnoea (OSA)
J Beverstock, M Dadej, S Mandal, N Devani

**P47** Comparing CPAP delivery methods: face to face versus locker collection
AL Craven, K Suganthavel, J Bates, MI Perera, MP Unstead, B Swaffield

**P48** Assessing the sustainability of the continuous positive airway pressure treatment pathway for obstructive sleep apnoea patients
H Mehta, F Yaqoob, H Singh, C Wood, LJ Smith, KK Lee

**P49** Evaluating driving risk assessment in untreated Obstructive Sleep Apnoea Syndrome (OSAS) patients and in controls by using the Leeds Driving Risk Score
A Dwarakanath, D Ghosh, MW Elliott

**P50** A pilot digital pathway for remote detection of sleep disturbance in cardiometabolic disease
V Handoo, J Newman, E de Bie, M Davies, M Wilson, E Davies, M Toshner

**P51** Mild but not minor: investigating the clinical burden of MAA use in mild obstructive sleep apnoea
S Hussain, MT Lam, M Jahangir, R Syed, KK Lee

**P52** Prevalence of sleep apnoea among patients who required bilevel positive airway pressure (BiPAP) therapy for acute hypercapnic respiratory failure – A single-centre retrospective study
ML Chan, T Bongers

**P53** Obstructive Sleep Apnea (OSA) in sarcoidosis – A neglected aspect of the disease
A Zahid, M Sayo, A Gulati, A Fahim, A Mowat

**P54** More than a near miss: the importance of multi-question screening for driving impairment in obstructive sleep apnoea
R Syed, M Jahangir, S Hussain, MT Lam, K Lee

**P55** Personalized maxillo-mandibular advancement in obstructive sleep apnoea: a millimetre-based individualized treatment approach
MZ Nasim, D Srinivasan, S Adlakha, R Singh, J Jones, MP Sovani

**P56** Fatigue, function and fear: what matters most to patients with obstructive sleep apnoea?
HY Yap, MR Frankl, KL Lee

**P57** Accuracy of STOPBANG in identification of OSA in difficult and severe asthma
L Piggott, S Shelley, O Gavin, A Brennan, P Coss, B Kennedy, BD Kent

**P58** Impact of virtual CPAP set-up on sleep service interactions
S Imaan, N Haq, M Latham, D Ghosh

**P59** Use of multichannel sleep studies to triage CPAP treatment in symptomatic patients with normal overnight oximetry
KM Townson, EJ Garden, GV Robinson, MP Unstead

**P60** On the right Trach yet? National survey of respiratory registrar experience with tracheotomy training
C Aday, A El-Nayal, E Abugassa, D Sagar

**2.15pm-4.15pm**
**Westminster, 4th floor**
**POSTER DISCUSSION: P61-P76**

## Holistically improving asthma care

*Chaired by: Dr Luke Daines (Edinburgh) and Dr Llinos Jones (Dewsbury)*

**P61** Barrier and facilitators to primary care practitioner prescribing and patient adoption of anti-inflammatory, maintenance and reliever therapy (MART) for adults with asthma
J Dyson, H Cummings, G Williams, V Raja Gopal, M Watt, T Morris, MG Crooks

**P62** Patient reported effects of vaping on asthma control in severe asthma
Z Aslam, C Butler, N McCollough, K McCabe, L Wright, R Stone, LG Heaney, PJ McDowell

**P63** Bridging inequality through innovation: systems-based proactive approach to reduce asthma exacerbations in a novel nurse-led model in areas of social deprivation
S Ryder, K Roy, R Basra, K Pindolia, E Davies, L Parsons

**P64** Assessing the effect of ethnicity and socioeconomic status on outcomes in asthma: a systematic review
I Adejumo, S Chandrasekharan, E Barber, A De, CV Chalitsios, J Kaur, W Ekezie, TM McKeever, DE Shaw

**P65** Exploration of the perceptions of South Asian patients with asthma: South Asian Respiratory Interventions (SARIs) study
N Lakhani, A Mahmood, D Wensley, P Rivers, AC Murphy

**P66** Improving asthma outcomes in Lancashire and South Cumbria (L&SC) through SABA reduction and MART implementation with SENTINEL Plus
M Preston, H Beaumont-Kellner, A O'Reilly-Smyth, J Lawson, M Rooney

**P67** What proportion of patients attending A&E with acute asthma could be discharged on MART/AIR?
J Cartledge, A Dhara, J Pott, B Bloom, L Steed, I Skene, PE Pfeffer

**P68** Are hospital organisational factors associated with the delivery of the adult asthma discharge bundle? An analysis of data from the National Respiratory Audit Programme
GW Nava, MM Harris, A Adamson, R O'Beirne, P Van-Geffen, JK Quint, TMA Wilkinson, JW Dodd

**P69** Intensive care admissions for asthma are characterized by frequent non-attendance to outpatient appointments
C Ottewill, S Walsh, C Gill, J Mullvihill, R Dwyer, RA Leary, RW Costello

**P70** Asthma biologic case-finding, an evaluation of a pragmatic physician-led community screening programme within Bristol Complex Airways service
GW Nava, H Sakota, CR Morgan, JS Grenville, DH Higbee, JW Dodd, R Shrimanker, PW Creber

**P71** Living well with difficult asthma: beliefs about breathlessness management through lifestyle changes in a dietitian-, physiotherapy-, and psychology-led multidisciplinary group
L Rose, B Kemp, V Rose, P Pfeffer

**P72** Reduction in healthcare utilisation and inappropriate pharmacological therapy following management of refractory breathlessness
R Daly, S Ludlow, G Tavernier, S Fowler

**P73** Changing trends in occupational asthma cases identified at a single specialist centre over 30 years
T Kabir, J Szram, P Howlett, J Feary

**P74** Improving outcomes in difficult asthma: clinical impact of physiotherapy and speech and language therapy in multidisciplinary care
Y Durmus, N Kennedy, F Cicognini, N Nixon, R Salman, I Clifton

**P75** Development of the UK Severe Asthma Registry as a performance-monitoring quality improvement platform
PE Pfeffer, T Brown, A Kramer, M Patel, S Gonem, P Patel, E Idris, D Shrikrishna, K Prior, AH Mansur, H Burhan, R Chaudhuri, R Gore, S Naveed, I Clifton, T Pantin, S Faruqi, R Shrimanker, M Doherty, DJ Jackson, S Doe, S Message, L Heaney, J Busby, H Rupani

**P76** Equity and access in severe asthma care: patterns of biologic therapy initiation across England
H Rupani, R Birch, A Saleem Ullah, K Blacker, J Fuld

**2.30pm-4.00pm**
**Mountbatten, 6th floor**
**JOINT BTS/BPRS SYMPOSIUM**

## SUSTAINABLE RESPIRATORY CARE FOR FUTURE GENERATIONS

*Chaired by: Dr Harry Apperley (London) and Dr Christopher Grime (Liverpool)*

| | |
|---|---|
| **2.30pm** | Environmentally friendly inhalers – ensuring accessibility for all<br>Professor Refiloe Masekela (Durban) |
| **3.00pm** | Climate change and impact on lung health<br>Professor Jonathan Grigg (London & Munich) |
| **3.30pm** | The future of sustainable healthcare: a global perspective on lung health<br>Dr Laura-Jane Smith (London) |

*Learning objectives*

*1) To understand the current regulations relating to propellants in asthma inhalers and their potential impact on devices for children and those in low- and middle-income countries.*

*2) To understand the impact of climate change, including wildfires, extreme heat and rising $CO_2$ levels on respiratory disease.*

*3) To advocate for change at the policy and personal level to improve lung health for children and future generations.*

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**3.00pm-4.30pm**
**Whittle & Fleming and Britten, 3rd floor**

## COFFEE/TEA BREAK and visit the exhibition stands

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**3.15pm-4.10pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Acute and Complex Respiratory Infections

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**3.15pm-4.10pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Global Lung Health

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**4.15pm-4.45pm**
**Churchill, ground floor**

## AWARD PRESENTATIONS

*Presentation of the BTS Medal, BTS President's Award, BTS Award for Meritorious Service, BTS/BALR/A+LUK Early Career Investigator of the Year, BTS/A+LUK/BALR Mid-Career Lecture and BTS Medical Student Awards.*

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**4.45pm-5.30pm**
**Churchill, ground floor**
**BTS PRESIDENT'S ADDRESS**

## "BREATHTAKING!"

*Professor Robina Coker (London)*

*Introduced by: Professor Nicholas Maskell (Bristol)*

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**5.35pm-6.05pm**
**Churchill, ground floor**
**BTS ANNUAL GENERAL MEETING**

## British Thoracic Society members only

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# Thursday 27 November 2025

**8.00am-9.00am**
**Whittle & Fleming, 3rd floor**

## COFFEE/TEA and visit the exhibition stands

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**8.45am-4.00pm**
**Whittle & Fleming, 3rd floor**

## POSTER VIEWING

*Authors present: 10.00am-11.00am*

### P77-P87

### Best practice in pleural procedures and pneumothorax

*Discussion of abstracts will take place from 2.00pm to 3.25pm in the Abbey, 4th floor*

### P88-P102

### Insights and advances in ILD care

*Discussion of abstracts will take place from 2.00pm to 3.50pm in the Westminster, 4th floor*

### P103-P117

### The road to change in chronic cough

*Discussion of abstracts will take place from 2.00pm to 3.50pm in the Moore, 4th floor*

### P118-P132

### Respiratory realities: the patient perspective

*Discussion of abstracts will take place from 2.00pm to 3.50pm in the Rutherford, 4th floor*

### P133-P147

### Paediatric infection, inflammation and physiology

*Discussion of abstracts will take place from 2.00pm to 3.50pm in the Albert, 2nd floor*

### P148-P162

### Risk predictors and diagnostics I COPD

*Discussion of abstracts will take place from 4.15pm to 6.05pm in the Westminster, 4th floor*

### P163-P177

### Cancer: from screening to therapy

*Discussion of abstracts will take place from 4.15pm to 6.05pm in the Moore, 4th floor*

### P178-P192

### Respiratory infections: pathogens to patients

*Discussion of abstracts will take place from 4.15pm to 6.05pm in the Abbey, 4th floor*

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**8.45am-4.00pm**
**Cambridge, 5th floor**
**MODERATED POSTER VIEWING**

### M17-M32

### The multi-morbidity of asthma

*Discussion of abstracts will take place from 2.00pm to 4.00pm in the Cambridge, 5th floor*

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**8.00am-8.30am**
**Moore, 4th floor**
**JOURNAL CLUB**

## TOBACCO DEPENDENCY / SMOKING / VAPING

*Professor Sanjay Agrawal (Leicester)*

*Learning objectives*

*To review the latest publications and evidence in the field of tobacco dependency, smoking and vaping.*

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**8.45am-10.15am**
**Churchill, ground floor**
**SYMPOSIUM**

## KEY DETERMINANTS OF OUTCOME IN ASTHMA: FROM ONSET TO REMISSION

*Chaired by: Dr Hassan Burhan (Liverpool) and Dr Freda Yang (London)*

| | |
|---|---|
| **8.45am** | Child-onset versus adult-onset asthma<br>Professor J Christian Virchow (Rostock, Germany) |
| **9.15am** | Symptom versus biomarker-driven care<br>Professor Ian Pavord (Oxford) |
| **9.45am** | Clinical versus biological remission<br>Dr Jane McDowell (Belfast) |

*Learning objectives*

*1) To understand how age of onset of asthma influences treatment responses and what this teaches us about the different mechanisms of child- and adult-onset asthma.*

*2) To review the evidence of T2 biomarkers in chronic asthma management, the potential risks of symptom-based treatment approaches, and how routine use of biomarkers can support steroid stewardship and reduce risk for patients with asthma.*

*3) To review the concepts of clinical and biological remission in asthma, understand the barriers to achieving remission, and explore whether clinical remission in the absence of biological remission is sufficient.*

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**8.45am-10.15am**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## HIGHLIGHTS FROM *THORAX AND LANCET RESPIRATORY MEDICINE*

*Chaired by: Dr Emma Grainger (Lancet Respiratory Medicine) and Professor Cecilia O'Kane (Thorax)*

**8.45am** Welcome and introduction
Editors-in-Chief from *Thorax* and *Lancet Respiratory Medicine*

**8.50am** Performance of volume and diameter thresholds in malignancy prediction of solid nodules in lung cancer screening
https://doi.org/10.1136/thorax-2024-222086
Dr Andrew Creamer (London)

**9.20am** Oral corticosteroid reduction and discontinuation in corticosteroid-dependent adults with severe, uncontrolled asthma treated with tezepelumab (WAYFINDER): a multicentre, single-arm, phase 3b study (*Lancet Respiratory Medicine*)
Professor David Jackson (London)

**9.50am** Editors' discussion
In this session the *Thorax* and *LRM* editors will discuss challenges and opportunities in scientific publishing, for both authors and editors. Topics will include artificial intelligence and the use of scientific pre-prints.

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**8.45am-10.20am**
**St James, 4th floor**
**SPOKEN SESSION: S56-S61**

## Impact of infection on disease mechanisms

*Chaired by: Dr Martha Purcell (Southampton) and Dr Aran Singanayagam (London)*

**8.50am** **S56**
Machine learning and enrichment analysis reveal persistent protein signatures in longitudinal post-COVID symptom trajectories
K Ntotsis, C Efstathiou, M Richardson, RS Thwaites, F Liew, D Lozano-Rojas, M Sereno, RA Evans, LV Wain, PJM Openshaw, CE Brightling

**9.05am** **S57**
EP395, a novel non-antibiotic macrolide, enhances antiviral responses in bronchial epithelial cells from asthma patients
S Malm Tillgren, M Menzel, J Kricker, MJ Parnham, V Norris, M Bech, L Uller

**9.20am** **S58**
Viral infection perturbs the respiratory mycobiome in COPD
T Jakobi, M Kusumoto, T Jaggi, T Shahridan Faiez, O Pitts, P Mallia, A Shah, SL Johnston, S Chotirmall, A Singanayagam

**9.35am** **S59**
Effects of bacteria and heme on neutrophil-endothelial interactions in COPD
M Wilcox, J Baker, H Deka, D Singh, S Lea

**9.50am** **S60**
Resolvin D1 ameliorates the inflammatory response to Haemophilus Influenzae infection in COPD monocyte-derived macrophages
J Mah, P Fenwick, R Lopez, Y Guo, F Shrosbery, SWJ Bartlett-Pestell, A Braddy-Green, J Devulder, RJ Snelgrove, L Donnelly, JA Wedzicha, LJ Finney

**10.05am** **S61**
Protective early innate immune activation by mitochondrial antiviral signalling protein (MAVS) in bacterial pneumonia
Y Long, SB Morgan, Y Mai, P Klenerman, TSC Hinks

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**8.45am-10.20am**
**Westminster, 4th floor**
**SPOKEN SESSION: S62-S67**

## Biology and clinical insights in cancer

*Chaired by: Professor Sam Janes (London) and Dr Laura Succony (Cambridge)*

**8.50am** **S62**
High dimensional immune analysis reveals immune subsets associated with lung cancer
SB Knight, R Hodgetts, C Andrews, A Blake, T Hussell, P Crosbie, N Diar-Bakerly, L Diar-Bakerly, S Grundy, S Kirkham

**9.05am** **S63**
Metabolic profiling of peripheral blood and tissue specific immune alterations in Diffuse

Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia (DIPNECH)
JC Worrell, L Joshi, K Duvall, H O'Brien, D O'Malley, N Boyle, J McCormack, A Batool, M McKean, SH Forde, M Hovel, AJ Byrne, C McCarthy

**9.20am** **S64**
Integrative immune and genomic profiling of thymic epithelial tumours: a translational study of biomarkers and therapeutic potential
R Bensabai, G Gerrard, S Gennatas

**9.35am** **S65**
Evaluation of protein X-targeting bispecific T cell engager therapy in patient-derived mesothelioma cell cultures
N Manoharan, P Kok-Ting Wan, T Dong, NM Rahman, N Kanellakis

**9.50am** **S66**
A binary prognostic score, incorporating Fibulin-3, predicts 1-year survival in pleural mesothelioma
HD Stubbs, S Tsim, L Alexander, C Kelly, M Tate, KG Blyth

**10.05am** **S67**
Association of airflow obstruction identified at lung cancer screening with major adverse cardiovascular events
T Patrick, A Bhamani, SB Naidu, ML Mullin, J McCabe, R Prendecki, P Verghese, A Creamer, CR Khaw, H Hall, J Dickson, C Horst, S Tisi, K Gyertson, A Hacker, L Farrelly, A Hingorani, R Patel, A Hackshaw, A Nair, J Jacob, S Doffman, JR Hurst, SM Janes

**8.45am-10.20am**
**Moore, 4th floor**
**SPOKEN SESSION: S68-S73**

## Cystic fibrosis: impact of CFTR modulators

*Chaired by: Dr Charlotte Addy (Cardiff) and Dr Jordan Busby (Nottingham)*

**8.50am** **S68**
Long-term effects of elexacaftor/tezacaftor/ivacaftor on clinical outcomes and quality of life among adolescents and adults aged 12+ with cystic fibrosis: interim results from the TRAJECTORY Study
F Ratjen, C Schwarz, V Daccò, J Duckers, A Solé, P Azevedo, MI Ahmed, F Gamboa, M Cipolli, BS Quon, QN Do, E Muller, M Jennings, S Sutharsan, TNS the TRAJECTORY Study Team

**9.05am** **S69**
Reduction in serum markers of inflammation in people with cystic fibrosis who are chronically infected with Pseudomonas aeruginosa is seen after treatment with Elexacaftor/Tezacaftor/Ivacaftor
Y Rajeev, TS Fuchs, JA King, JC Davies

**9.20am** **S70**
Oxygen enhanced (OE-)MRI and multiple breath washout with Short extension (MBWShX) reveal lung disease progression in people with cystic fibrosis despite triple CFTR modulator therapy
C Short, T Semple, M Abkir, M Tibiletti, M Rosenthal, S Padley, GJM Parker, JC Davies

**9.35am** **S71**
Pre-conception lung function and the pregnancy outcomes among females with cystic fibrosis (fwCF) in the CF-transmembrane conductance regulator modulator (CFTRm) era
A Downes, L Weitnauer, R Robinson, E Diable, R Scott, I Felton

**9.50am** **S72**
Use of cardiovascular risk scores in adult populations with cystic fibrosis
J Lim, C Landers, MT Cooney, C Corish, O Griffin, C Gallagher, T Nicholson, E McKone, S Carter

**10.05am** **S73**
Epidemiology of cystic fibrosis-related deaths in the United States, 1999-2023: a CDC WONDER-Based Study
I Mushtaq, MR Sarfraz, MF Hemida, A Ali, AA Ibrahim, K Patel, M Saghir, Z Sharhiar, HM Ahmad, ZJ Chaudhry

**8.45am-10.20am**
**Abbey, 4th floor**
**SPOKEN SESSION: S74-S79**

## Sleep apnoea and beyond

*Chaired by: Dr Tim Quinnell (Cambridge) and Dr Anita Saigal (London)*

**8.50am** **S74**
Usefulness of OSLER for patient and clinician: 5-year data from a tertiary sleep service
L Searle, F Kelting, M Austin, L Mattock, C Best, S Iyer

**9.05am** **S75**
An exploratory study of a novel, low-cost, simple sleep apnoea screening device, ApneScan DC1, to identify apnoeas and hypopneas
A Thomas, N Bryan, M Assaduzzaman, J Walsh, A Moss

**9.20am** **S76**
The clinical impact of 3D printed positive airway pressure (PAP) therapy interfaces (3DPiPPIn): a randomised controlled trial
SK Mansell, F Gowing, D Ridout, C Kilbride, O Olsen, ST Hilton, E Main, S Schievano, S Mandal

**9.35am** **S77**
Transcutaneous electrical stimulation-induced fatigue in the tibialis anterior: a surrogate model for upper airway muscle function
M Alkhathami, A Almaghrabi, A Madkhali, C Zhang, A Alsharifi, G Kaltsakas, G Rafferty, J Steier

**9.50am** **S78**
Exploring phenotype-specific response to home non-invasive ventilation in chronic hypercapnic respiratory failure
HY Yap, K Abernethy, N Shah, KA Arif, S Bokhari, R D'Cruz, N Hart, G Kaltsakas, M Ramsay, S Srivastava, J Steier, P Murphy, ES Suh

**10.05am** **S79**
Adherence to a Non-Invasive Ventilation (NIV) trust guideline targeting tidal volumes and its correlation with clinical outcomes
CJ Westley, K Chatfield, J Hodson, A Harriman, K Filby

**8.45am-10.20am**
**Rutherford, 4th floor**
**SPOKEN SESSION: S80-S85**

## Pulmonary hypertension and CTEPH – classification and treatment

*Chaired by: Professor Stavros Konstantinides (Mainz) and Dr Alison MacKenzie (Glasgow)*

**8.50am** **S80**
Exploring machine learning techniques for classifying pulmonary hypertension using UK Biobank electronic health records
N Bhamji

**9.05am** **S81**
Epidemiology, prognosis, and healthcare resource use of patients with pulmonary hypertension associated with lung disease and/or hypoxia in the UK
D Kiely, SJ Wort, D Funes, N Petrica, C Proenca, M Pini, M Fernández Delgado, G Bacchini Jeanneret

**9.20am** **S82**
Predictors of outcome in pulmonary hypertension associated with lung disease
E Cerrone, ZM Goh, F Hitchcock, A Gajanan, M Senkathir, S Alabed, K Dwivedi, RA Condliffe, A Hameed, A Charalampopoulos, JA Hurdman, CA Elliot, AMK Rothman, AJ Swift, AAR Thompson, DG Kiely

**9.35am** **S83**
Comparing bridging therapies prior to balloon pulmonary angioplasty in chronic thromboembolic pulmonary hypertension
O Li, H Ghani, J Pepke-Zaba, S Hoole

**9.50am** **S84**
Balloon pulmonary angioplasty versus Riociguat in inoperable chronic thromboembolic pulmonary hypertension: a systematic review and meta-analysis of randomised controlled trials
A Gaur, G Ishaque, I Khan, AA Khan, F Shafiq, M Ishaque, S Prashntkumar, R Riaz, N Ehsan, V Qureshi

**10.05am** **S85**
Pulmonary haemodynamics after mechanical therapy with pulmonary endarterectomy (PEA) and balloon pulmonary angioplasty (BPA) predict survival in patients with chronic thromboembolic pulmonary hypertension (CTEPH)
E Turner-Frick, S Hoole, H Ghani

# Thursday 27 November 2025

**8.45am-10.20am**
**Albert, 2nd floor**
**SPOKEN SESSION: S86-S91**

## Mechanisms of IPF pathogenesis

*Chaired by: Dr Paul Minnis (Antrim) and Dr Pilar Rivera-Ortega (Exeter)*

**8.50am** **S86**
Integration of IPF-Associated SNPs with differential DNA methylation and gene expression shows cell-type specific mechanisms in airway and parenchymal fibroblasts
A Valand, P Rajasekar, AL Lagan, J Patel, B Barksby, RA Burgoyne, JL MacIsaac, D Lin, ST May, M Castellanos-Uribe, MS Kobor, T Hackett, AJ Halayko, AJ Knox, A Fisher, L Borthwick, LV Wain, R Clifford

**9.05am** **S87**
Targeting AKAP13 RhoGEF activity ameliorates pro-fibrotic phenotypes driven by the IPF associated AKAP13 risk variant
B Liu, S Gangi, C Wong, J May, S Wang, E Pyman, A Vairon, M Zarcone, EL Lopez Jimenez, AE John, RG Jenkins

**9.20am** **S88**
Exploring the effect of PKN2 knockdown on idiopathic pulmonary fibrosis
CE McMullan, CB Maxwell, P Rajasekar, P Stylianou, R Clifford, DJL Jones, RJ Allen, LV Wain, KM Roach

**9.35am** **S89**
The role of TRPM7 in human lung myofibroblast profibrotic function
M Ratnasingham, S Narayan, AJ Kelly, P Rajasekar, P Bradding, SM Duffy, FD Horgen, C Feghali-Bostwick, R Clifford, KM Roach

**9.50am** **S90**
The impact of a hypoxic environment on alveolar macrophage immunometabolism: implications for lung fibrosis
E Jenkins, L Crowley, K Spencer, D Thickett, D Parekh, A Scott

**10.05am** **S91**
Defining the antifibrotic mechanisms of Treprostinil in pulmonary fibrosis
J May, B Liu, L Rahman, H Yao, AE John, JA Mitchell, RG Jenkins

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**9.00am-10.30am**
**Windsor, 5th floor**
**SYMPOSIUM**

## RESPIRATORY WORKFORCE OF THE FUTURE

*Chaired by: Dr Kate Lippiett (Southampton) and Christine Mwasuku (London)*

**9.00am** The value of clinical nurse specialists in respiratory medicine
Professor Nicola Ranger (London)

**9.30am** Ensuring a robust nursing curriculum for the respiratory nurses of the future
Professor Barry Hill (Northumbria)

**10.00am** What's on the respiratory nursing research horizon?
Dr Lindsay Welch (Bournemouth)

*Learning objectives*

*1) To explore the scope of practice of respiratory clinical nurse specialists and the definitions of specialist practice.*

*2) To be familiar with the career framework for respiratory nurses and explore the state of respiratory nursing education.*

*3) To explore respiratory research priorities.*

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**10.00am-11.00am**
**Whittle & Fleming and Britten, 3rd floor**

## COFFEE/TEA BREAK and visit the exhibition stands

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**10.45am-11.45am**
**Abbey, 4th floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Asthma

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.45am-11.45am**
**Rutherford, 4th floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

### Cystic Fibrosis

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.45am-11.45am**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

### Pharmacist

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.45am-11.45am**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

### Lung Cancer and Mesothelioma

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.45am-12.20pm**
**Westminster, 4th floor**
**SPOKEN SESSION: S92-S97**

### Enhancing outcomes in respiratory failure: from trials to real world practice

*Chaired by: Ms Pearlene Antoine-Pittersen (Birmingham) and Professor Nicholas Hart (London)*

**10.50am** **S92**
Establishing real world quality standards for domiciliary non-invasive ventilation
MT Rashid, AW Molyneux, E Crookes, DP Smith

**11.05am** **S93**
Clinician perspectives on the need and feasibility for an RCT comparing nasal high flow therapy to controlled oxygen in COPD patients intolerant of NIV
D Ghosh, S Bourke, B Messer, M Davies

**11.20am** **S94**
The lived experiences of patients using Positive Airway Pressure (PAP) therapy: a nested phenomenological study within the 3DPiPPIn randomised controlled trial
SK Mansell, L Geddes, F Gowing, ST Hilton, E Main, S Schievano, S Mandal, C Kilbride

**11.35am** **S95**
Remote monitoring to improve low adherence to non-invasive ventilation in patients with Obstructive Sleep Apnoea / Obesity Hypoventilation Syndrome (READ-NIV TRIAL)
A Alsharifi, G Kaltsakas, M Ramsay, J Owusu-Afriyie, G Radcliffe, G Rafferty, J Steier

**11.50am** **S96**
Weight management in patients with decompensated Obesity Hypoventilation Syndrome: understanding the changing landscape in the era of weight loss injections
CH Lim, A Lau, L Taylor, D Kardasopoulos, N Lane, H Tedd

**12.05pm** **S97**
Factors associated with overall survival in patients with motoneuron disease and respiratory symptoms
A Shaikh, IS Mohammed, D Freeman, A Bentley, A Bikov

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**10.45am-12.30pm**
**Churchill, ground floor**
**SYMPOSIUM**

### PLENARY SCIENTIFIC

*Chaired by: Professor Mona Bafadhel (London) and Professor Christopher Brightling (Leicester)*

**10.45am** Health research in the respiratory workforce
Professor Nicola Roberts (Edinburgh)

**11.10am** Cellular effect and mechanisms of exposure
Professor Matthew Loxham (Southampton)

**11.35am** The breathless patient, spanning COPD, HF and long COVID-19
Professor Rachael Evans (Leicester)

**12.00pm** Identification of novel therapeutic targets in pulmonary hypertension through omics approaches
Dr Christopher Rhodes (London)

# Thursday 27 November 2025

*Learning objectives*

*1) To learn about how to impact the respiratory workforce.*

*2) To learn about cellular mechanisms following exposure to the lung.*

*3) To learn about the breathlessness patient.*

*4) To learn about mechanisms of pulmonary arterial hypertension.*

**12.00pm-2.15pm**
**Pickwick, 1st floor and Whittle & Fleming, 3rd floor**

## LUNCH and visit the exhibition stands

*(Lunch is not included in the delegate fee but is available to purchase onsite. Card payments only)*

**1.00pm-1.45pm**
**Churchill, ground floor**
**MORRISTON DAVIES GUEST LECTURE**

## VACCINE PREVENTABLE RESPIRATORY ILLNESS

*Introduced by: Professor Adam Hill (Edinburgh)*

*Guest Lecturer: Professor Wei Shen Lim KBE (Nottingham)*

**2.00pm-3.00pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Sleep

*All participants are welcome to attend and hear more about the work of the SAGs.*

**2.00pm-3.25pm**
**Abbey, 4th floor**
**POSTER DISCUSSION: P77-P87**

## Best practice in pleural procedures and pneumothorax

*Chaired by: Professor Eleanor Mishra (Norwich) and Dr Benjamin Pippard (Newcastle upon Tyne)*

**P77** A national survey of real-time ultrasound guidance use in pleural procedures in the United Kingdom
CA Mounsey, B Iqbal, DN Addala, NI Kanellakis, NM Rahman

**P78** The role of the general medical registrar in performing out-of-hours pleural procedures: a UK national survey of trainee and trainer perspectives
D Jajbhay, A Aujayeb, P Denniston, M Juniper, J Liang, H Shotton, J Zhang, O Kadwani

**P79** Pleural procedures: how prepared are general medical and respiratory trainees?
S Ananth, O Cornwall, N Oldham, P Ramachandran

**P80** Improved recycling of elective pleural procedural waste
B Rowlands, B Callow, Z Sein, J Corcoran

**P81** "Knowing the unknown": a systematic review of literature investigating the optimal management of pneumothorax after CT-guided lung biopsy
B Iqbal, A Sundaralingam, D Addala, A Elsheikh, M Bhatnagar, N Kaushal, S Kette, C Mounsey, NM Rahman, RJ Hallifax

**P82** Characteristics of patients developing iatrogenic pneumothorax due to CT guided lung biopsy in a busy tertiary care hospital (Glenfield)
S Rizvi, E Smith, S Kalam, N Iqbal, A Yasmeen, T Wijayaratne, A Ussaid, M Rahman, S Mohammad, M Muhammad, S Agrawal, J Bennett, RC Sudhir

**P83** Respiratory complications including pneumothorax in patients receiving tyrosine kinase inhibitors
J Treves, A Warrier, J Harrington, S Marciniak

**P84** Weather changes and the incidence of spontaneous pneumothorax: an observational study from a single UK centre
M Vele Witharamalage, S Gudur

**P85** Clamp or remove? A national survey of intercostal chest drain management in spontaneous pneumothorax
AW Martinelli, N Veale, NM Rahman, RJ Hallifax, SP Walker

**P86** The accuracy of ICD-10 diagnostic codes for the identification of pneumothorax and pulmonary embolism: a retrospective study in two UK centres
T Hamakarim, P Bhutra, D Henshall

**P87** Surgically managed pneumothorax: a 5 year institutional appraisal of histopathological findings and their clinical implications
L Burke, H Ashe, A O'Mahony, D Ganesan, KM Phoo Thwe

**2.00pm-3.50pm**
**Westminster, 4th floor**
**POSTER DISCUSSION: P88-P102**

## Insights and advances in ILD care

*Chaired by: Dr Shaney Barratt (Bristol) and Dr Rachel Clifford (Nottingham)*

**P88** Does routine liver test monitoring for patients established on Nintedanib add clinical value? Experience of an UK Tertiary ILD centre
J Bradley, T Capstick, J Slough, A Boland, TJT Sutherland, P Beirne

**P89** A 'Call to Action' in determining and estimating a Minimum Important Difference (MID) in 6-Minute Walking Distance (6MWD) in PH-ILD
D Lewis, A Joseph, A Smith, S Graziadio, M Green

**P90** SEEing what's important: eliciting expert judgements to estimate the minimally clinically important difference for six-minute walking distance in PH-ILD
D Lewis, A Joseph, A Smith, S Graziadio, M Green

**P91** Real-world healthcare resource utilization in patients with idiopathic pulmonary fibrosis initiating antifibrotic therapy in the Medicare fee-for-service population
B Wu, A Yu, M Lin, E Roberts-McCarthy, K Morland, S Robinson, M Ramaswamy

**P92** A novel CT-chest scoring system to predict nodal sarcoidosis in patients with isolated hilar and mediastinal lymphadenopathy
E Abugassa, B Al-Harbawi, T Kareem, H Jawad, SS Doctor, M Haris

**P93** Concomitant presence of Allergic Rhinitis (AR) in Chronic/Fibrotic Hypersensitivity Pneumonitis (C/F-HP) patients: a significant correlation!
A Salman, A Shakeel, R Mudassar, A Sumbal, A Muhammad, F Manal

**P94** Development and validation of the Hypersensitivity pneumonitis South Asian Questionnaire for Exposure (HP-SAQE)
S Singh, A Sharma, K Pilania, P Singh, AK Sharma, S Dhooria, V Hadda, N Gupta, M Gupta, A Rathnapala, A Agarwal, N Singh, M Joshi, A Mondal, J Bairwa, V Singh, K Johannson

**P95** Evaluation of ChatGPT and Gemini in generating differential diagnoses in diffuse interstitial lung diseases (DILDs) from transcribed radiological reports
J Benson, S Desai

**P96** The first three years of the R421 pulmonary fibrosis genetic testing panel: outcomes from a regional familial ILD clinic
GC Heslin, F Sutcliffe, AR Bowden, H Parfrey, JA Dickens

**P97** Comparing inflammatory and fibrotic hypersensitivity pneumonitis: insights from a UK tertiary centre
T Raj, J Shaw, C Hayton, B Vijayakumar

**P98** The burden of comorbidity in fibrotic Interstitial Lung Disease (fILD): a prospective study in a regional ILD centre
H Emms, C Crook, JK Prague, K Norris, R Sayers, M Steward, H Thould, A Savill, J Mandizha, S Lines, A Barnes, J Kirkwood, H Almond, S Stanel, P Rivera-Ortega, DM Baird, AM Russell, MA Gibbons, CJ Scotton, A Duckworth

**P99** **WITHDRAWN** Comorbidity and consequences: a retrospective analysis of demographic trends in mortality from coexisting interstitial lung disease and ischaemic heart disease
A Ali, MF Hemida, MR Sarfraz, AA Ibrahim, W Qaisi, M Al-saadi, A Eldeeb, Y Shariff, A Munim, I Mushtaq

**P100** Place of death in people with pulmonary fibrosis: a cohort study using electronic healthcare records in England
V Navaratnam, AD Morgan, JC Porter, R Chambers, RG Jenkins, JK Quint

**P101** Case series: the role of bronchoalveolar lavage in interstitial lung disease management and patient outcomes
A Muthusami, S Naureen, S Hart

**P102** Strength in partnership: advancing ILD care through collaboration
M Koulopoulou, A Fahim, L Fabri , D Barbonchielli, N Chien, S Mulholland, R Boorsma, J Burrough, M Naqvi

# Thursday 27 November 2025

**2.00pm-3.50pm**
**Moore, 4th floor**
**POSTER DISCUSSION: P103-P117**

## The road to change in chronic cough

*Chaired by: Professor Lorcan McGarvey (Belfast) and Dr Claire Slinger (Preston)*

**P103** Patients' perspective of control in chronic cough
EC Mackay, KL Rhatigan, RD Turner, KJ Myall, WJ Song, JH Hull, SS Birring, PSP Cho

**P104** The validity of a cough severity numerical rating scale
KL Rhatigan, EC Mackay, RD Turner, KJ Myall, JH Hull, SS Birring, PSP Cho

**P105** An audio trimming algorithm for cough analysis in ambulatory recordings: a proof-of-concept study
J Smith, T Taylor, E Travassos, D Drennan, F Keane, Y Zigel, H Venn

**P106** Comparison of two different nebulisation systems for citric acid cough challenge testing
J Mitchell, K Holt, R Dockry, J Smith

**P107** Evaluating the loss of cough frequency resolution when using cough seconds instead of explosive coughs
T Taylor, J Smith, Y Zigel, D Drennan, H Venn, F Keane, M Kitt, O Poirot

**P108** Provocation laryngoscopy in the management of chronic cough; what is the added value?
H Lever, C Slinger, B Tidmarsh, P Marsden

**P109** Treatable traits in cough: patients with refractory chronic cough undergoing laryngoscopy and cough control therapy
J Sutanto, J Butler, C Hunter, E Green, L Peace, S Fearby, S Parker

**P110** Upper airway speech and language therapy-led laryngoscopy clinics are safe and enable bespoke therapy programmes to be delivered
JM Butler, J Sutanto, C Hunter, S Parker

**P111** Reduced cough severity and improved confidence in self-management following completion of a Virtual Physiotherapy Group Intervention for Chronic Cough (ViP-Cough)
CC Reilly, E Shiner, SV Floyd, I Vermeulen, C Ross, P Cho, CJ Jolley, SS Birring

**P112** Differences in AX-8 and placebo effects on cough bouts in refractory or unexplained chronic cough: a new count- and duration-based analysis
JA Smith, O Poirot, DP Drennan, Y Zigel, TE Taylor, HE Venn, MM Kitt

**P113** Efficacy and safety of nalbuphine extended-release in refractory chronic cough: results from the phase 2a RIVER trial
JA Smith, KR Chapman, I Satia, É Millaire, SM Parker, L McGarvey, A Morice, P Marsden, SS Birring, A Turner, M Garin, J Cassella

**P114** Development of accessible digital resources to enhance upper airway control therapy
J Butler, C Hunter, B O'Connell, S Parker

**P115** Association between baseline cough burden and gefapixant discontinuations due to taste-related adverse events
J Smith, S Birring, P Dicpinigaitis, L McGarvey, A Morice, J Xu, A Martin Nguyen, G Philip, C La Rosa

**P116** Clinically relevant attenuation of cough with gefapixant: a post hoc analysis
S Birring, P Dicpinigaitis, L Dupont, L McGarvey, A Morice, J Smith, J Xu, A Martin Nguyen, G Philip, C La Rosa

**P117** Patient global impression of change: contextualizing patient-reported outcomes in gefapixant phase 3 trials
L Dupont, S Birring, P Dicpinigaitis, L McGarvey, A Morice, J Smith, J Xu, A Martin Nguyen, G Philip, C La Rosa

**2.00pm-3.50pm**
**Rutherford, 4th floor**
**POSTER DISCUSSION: P118-P132**

## Respiratory realities: the patient perspective

*Chaired by: Dr Natalie Harper (Dorset) and Mrs Cris Roxas (London)*

**P118** Understanding the experience and unmet needs of patients living with severe asthma on biologic therapy; including views on clinical remission
V Patel, H Rupani, LG Heaney, P Patel, L Jones, M Moore, D Montalto, G Turley, A Fynney, A Whittamore

**P119** Implementing inhaler recycling schemes – experience within a large UK NHS Trust
TGD Capstick, NF Azeez, M Chawla, R Bowers, M Powell, IJ Clifton

**P120** How can we embed sustainability in the inhaler prescription-use-disposal pathway?
AA Onasanya, AM Ignatowicz, AM Turner

**P121** User data from a validated online inhaler technique competency test: a novel approach to standardised assessment
C Chen, P Pfeffer, P Elston, A Hakim

**P122** The relationship between breathlessness and loneliness in individuals with chronic respiratory disease
R Alajmi, A Cumella, M Zawieja, A Laverty, NS Hopkinson, KEJ Philip

**P123** Does ethnicity influence the investigative pathway after presentation with breathlessness? An epidemiological study using CPRD records
H Sathanapally, U Karsanji, G Doe, K Khunti, M Steiner, A Singh, RA Evans

**P124** Influence of deprivation on COPD patients with respiratory acidaemia requiring non-invasive ventilation
MH Naseer, K Brewin, E Pakpahan, J Gray, S McCarthy, SC Bourke, MH Naseer, K Brewin, SC Bourke

**P125** Dedicated respiratory service for rough sleepers
S Balasubramanian, C Singh, H Young, M Tickle, S Nagra, S Joseph, S Stimson, A Fogarty, D Shaw

**P126** Understanding the acceptability and process mechanisms of a pathway navigation intervention to engage non-responders in lung cancer screening
LG Akwa, D McInnerney, L Mitchinson, G Hutton, I Simmonds, N Hancock, S Rogerson, J Lindop, D Vulkan, C Marshall, PAJ Crosbie, R Gabe, MEJ Callister, SL Quaife

**P127** Health inequities in preventive screening: a qualitative exploration of TLHC non-engagement in BAME communities
S Ramadore, A Hegazi, D Offen

**P128** Patient- and physician-reported symptom burden among patients with bronchiectasis in Europe
U Sommerwerck, P Donga, D Baldock, M Lauterio, M Small, J Fastenau

**P129** IKEN-ILD: Involvement, Knowledge, ENgagement in Interstitial Lung Disease (ILD) – a novel patient passport
AEJ Hogg, M Ward, A-M Russell, DJ Dhasmana

**P130** Improving respiratory outcomes in people with learning disabilities: evaluation of the 'Keeping My Chest Healthy' pathway
DL Smalley, TJ Barningham

**P131** Severe asthma patient perceptions and preferences on the frequency of biologic administration; including opinions on upcoming 6 monthly biologic
B Wright, I Berrar-Torre, A-M Bucataru, E McHugh, S Mamo, K Azarce, P Patel

**P132** Antifibrotic prescribing in a tier 2 centre: the ILD integrated clinical care pathway in practice
J Sutanto, W Angwin, W Ong, E Payne, R Henderson, G Barrett, L Mackay, S Haney

**2.00pm-3.50pm**
**Albert, 2nd floor**
**POSTER DISCUSSION: P133-P147**

## Paediatric infection, inflammation and physiology

*Chaired by: Dr Claire Edmondson (London) and Dr Dominic Hughes (London)*

**P133** Impact of elexacaftor/tezacaftor/ivacaftor on clinical outcomes and quality of life in children with cystic fibrosis aged 6-11 years in the real-world setting
JH Rayment, V Daccò, F Ratjen, J Duckers, A Solé, P Azevedo, M Cipolli, P Daigneault, MI Ahmed, QN Do, E Muller, M Jennings, S Sutharsan, TST the TRAJECTORY Study Team

**P134** Introduction of Lung Clearance Index (LCI2.5) measured by Nitrogen Multiple Breath Washout (MBW) into clinical practice
JL Forster, DS Urquhart, D Miller, K Moffat, KA Macleod

**P135** Paediatric bronchiectasis – the unmet need for psychological support in a tertiary centre
S Brown, C Lambert, M Dahabreh, C Pao, B Westphal, A Whitehouse

**P136** Clinical characteristics of a combined adult and paediatric cohort of patients with primary ciliary dyskinesia
J Poole-Cowley, E Johnson, M Beaton, A Devenny, L McElroy, E Ross, R Langley

**P137** Exploring parental experiences and coping with having children with primary ciliary dyskinesia: a qualitative secondary data analysis
S Suran, J Lucas, S Punton, K Dexter

**P138** Outcomes of children undergoing clinically indicated bronchoscopy for severe recurrent respiratory symptoms
J Ngeh, K Hillson, K Bonner, P Robinson, M Gore, K Mayoral, S Lacbay, R Peto, S Sonnappa, L Fleming, A Bush, S Saglani

**P139** Determinants of tidal volume in Nepali infants less than 6 weeks of age: a cross-sectional study
O Prakash Kurmi, S Neupane, B Kumar Gupta, N Chaudhary

**P140** Home spirometry to optimise respiratory secondary care new patient appointments
E Raywood, A Whiting, C Oliver, H Parrott, S Gray

**P141** The utility of home spirometry monitoring in children with respiratory symptoms
R Tuli, S Salehian, Z Awan, S Singham, E Raywood, C Oliver, C Mclean, L Fleming

**P142** Hospital attendance for recurrent preschool wheeze is not determined by disease severity
L Whittaker, R Chandra, A Selby, GJ Connett

**P143** Childhood asthma and air quality conversations: community pharmacy intervention conversations
R Parker, R Micallef, S Shah, R Waters, L King, S Patel, E Duncan, D Johal, S Kangulec, S Puaar, G Saunders, A McLeavy, N Lusardi, A Whitehouse

**P144** What information should a paediatric asthma discharge letter contain – a DELPHI study
A Gawlik-Lipinski, R O'Beirne, JW Dodd, T Wilkinson, I Sinha

**P145** Impact of breathing pattern interventions on breathing pattern disorders in teenagers with post-COVID syndrome
C Wells, C Richardson, T Segal, S Sonnappa

**P146** Development and implementation of a physiotherapy standardised assessment package for children with asthma or dysfunctional breathing across the NHS
C Wells, C Hepworth, N Barker

**P147** A novel approach to the use of an adjustable one-way valve in paediatric tracheostomy patients with complex airways: a case series
C Shaw, S Judge, J De Bolfo, L Ritchie

**2.00pm-4.00pm**
**Cambridge, 5th floor**
**MODERATED POSTER DISCUSSION: M17-M32**

## The multi-morbidity of asthma

*Chaired by: Professor Hannah Durrington (Manchester) and Dr Deepak Subramanian (Derby)*

**M17** Sex differences in the relationship between shift work status and prevalent asthma in the Our Future Health cohort
RJ Maidstone, MK Rutter, HJ Durrington

**M18** Shifting COPD and asthma care left: analysis of the 'one stop' community diagnostic clinic in the West of Ireland
CY Song, N Byrne, N Duignan, E Hall, O Healy, R Kelly, M McGoldrick, F Moore, P Needham, E O'Regan, C Sherlock, SM Walsh

**M19** Real-life evaluation of forced expiratory ratios with asthma exacerbations and symptoms
JY On, D Ahmed, F Paterson, R Poto, A Bhattacharjee, R Greig, J Brown, F Jongh, Z Diamant, T Fardon, B Lipworth, R Chan

**M20** Can measures of small airway dysfunction aid with the diagnosis or management of asthma exacerbations? A systematic review
A Alshehri, MI Alshahrani, E Sapey, RA Stockley, M Almeshari

**M21** Impact of the SENTINEL programme on asthma care across six primary care networks in England
MG Crooks, H Cummings, J Cohen, C Huang, L Pitel, J Turgoose, S Faruqi, M Watt, T Morris, V Raja Gopal, K Shah

**M22** Key indicators of care for patients hospitalised with asthma and COPD with comorbid mental health or cognitive impairment: preliminary findings from the National Respiratory Audit Programme (NRAP)
C Morgan, GW Nava, A Adamson, R O'Beirne, J Henderson, P VanGeffen, I Patel, JK Quint, TMA Wilkinson, JW Dodd

**M23** Obesity and anxiety impact on symptom burden in newly diagnosed and misdiagnosed asthma
R Wang, C Gill, C Uttewill, M Bennett, C Murray, A Simpson, SJ Fowler, RW Costello

**M24** The prevalence of psychological symptoms in severe and difficult asthma
K Stewart-Knight, A Siddika, K Walas, J Gates, L Osman, F Haris, L Green, M Fernandes, L Thomson, C Roxas, J Lam, A De Looper, G D'Ancona, AM Nanzer, J Dhariwal, DJ Jackson

**M25** Impact of severe asthma service on breathlessness: comparison of median ACQ at service entry with latest available median ACQ after treatment did not reach the minimal clinically important difference across 1628

patients within asthma and breath mastery learning health system
M Wildman, S Farrell, LYH Lai, S Dawson, S Cameron, A Tyas, A Bahra, C Stockton-Powdrell, D Sammut, H Astell, J Rosario, K O'Hara, M Sperrin, P Whelan, R Russell, S Saha, T Murray

**M26** Are symptom questionnaires a useful tool in the assessment of inducible laryngeal obstruction
C Slinger, H Lever, B Tidmarsh

**M27** A pain in the neck? Healthy volunteer acceptability of trans laryngeal ultrasound to assess vocal fold movement in mimicked inducible laryngeal obstruction
C Slinger, R Govender

**M28** Impact of biomarker led rapid access clinics on exacerbation management in a severe asthma population. Findings from the Bristol complex airways service
C Morgan, S Rudman, M Broad, J O'Connor, J Grenville, GW Nava, C Ryan, V Cobham, D Higbee, R Shrimanker, JW Dodd

**M29** The effectiveness of benralizumab in the management of chronic eosinophilic pneumonia
J Gates, F Haris, C Roxas, M Fernandes, L Green, A Dean-Jalloh, L Thomson, J Lam, G d'Ancona, AM Nanzer, DJ Jackson, J Dhariwal

**M30** Double or nothing: experience of dual monoclonal antibody prescriptions at a tertiary asthma centre
H Joplin, F Fyles, R Burton, S Zaidi, L Chishimba, H Burhan

**M31** Stringent American (ATS/ACAAI/AAAAI) endorsed 6-component definition of asthma remission is not achievable in a real-world benralizumab cohort
J Logan, K Wetherall, L Gillespie, A McConnachie, WTN Lee, H Burhan, T Brown, S Faruqi, DJ Jackson, R Kurukulaaratchy, AH Mansur, D Saralaya, SJ Fowler, P Patel, J Brown, J Lordan, S Siddiqui, PJ McDowell, R Djukanovic, ID Pavord, LG Heaney, CE Brightling, R Chaudhuri

**M32** A novel pathway for pharmacist-led stepwise de-escalation of background asthma therapy in patients stable on biologics. Experience from Bristol Complex Airways Service
H Sakota, GW Nava, C Morgan, J Grenville, S Mulholland, N Leung, E Hunter, D Higbee, JW Dodd, R Shrimanker

**2.15pm-3.15pm**
**Churchill, ground floor**
**SYMPOSIUM**

## JOINT BTS/A+LUK/BALR MID-CAREER LECTURES

*Chaired by: Professor Mona Bafadhel (London), Professor Philip Molyneaux (London) and Professor Karl Staples (Southampton)*

**2.15pm** Familial ILD: clinical challenges and insight into mechanisms of disease in pulmonary fibrosis
Dr Jennifer Dickens (Cambridge)

**2.45pm** Unmasking the long-term effects of respiratory virus infection
Dr Lindsay Broadbent (Surrey)

**2.15pm-3.45pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## LUNG CANCER STAGING: TNM9 AND RECENT DEVELOPMENTS IN ONCOLOGICAL THERAPY – IMPLICATIONS FOR PRACTICE

*Chaired by: Dr Liz Fuller (Newcastle upon Tyne) and Dr George Tsaknis (Kettering/Leicester)*

**2.15pm** Precision staging of the mediastinum – essential for current oncological management
Professor Corinne Faivre-Finn (Manchester)

**2.45pm** Lung cancer staging – TNM9: rationale for changes and clinical implications
Professor Paul van Schil (Antwerp)

**3.15pm** Implications for the UK – optimal practice and data acquisition
Professor Neal Navani (London)

*Learning objectives*

*1) To consider the importance of systematic mediastinal nodal staging with EBUS in planning oncological therapy (both neo-adjuvant treatment and radiotherapy planning).*

*2) To provide an update on the new lung cancer staging (TNM9), outlining the rationale for changes and potential clinical impacts on the diagnostic pathway for lung cancer patients.*

*3) To discuss implications for UK practice – the practicalities of mediastinal nodal staging, and data implications (BTS EBUS Audit and National Lung Cancer Audit).*

**2.15pm-3.45pm**
**Windsor, 5th floor**
**SYMPOSIUM**

## THE STORY OF THREE RARE LUNG DISEASES

*Chaired by: Dr Helen Barr (Nottingham) and Professor Daniel Peckham (Leeds)*

**2.15pm** What's next for cystic fibrosis?
Professor Jane Davies OBE (London)

**2.45pm** Where are we with primary ciliary dyskinesia?
Professor Amelia Shoemark (Dundee)

**3.15pm** What's new in alpha 1 antitrypsin deficiency?
Professor Alice Turner (Birmingham)

*Learning objectives*

*1) To outline key successes and developments in CF, PCD, A1AT deficiency.*

*2) To outline potential therapies and care developments in CF, PCD, A1AT deficiency.*

*3) To facilitate exchange of ideas and sharing of practice and networks.*

**2.15pm-3.50pm**
**St James, 4th floor**
**SPOKEN SESSION: S98-S103**

## Co-morbidity and novel treatments in COPD

*Chaired by: Professor Charlotte Bolton (Nottingham) and Dr Hamish McAuley (Leicester)*

**2.20pm** **S98**
Two decades, two destinies: when chronic obstructive pulmonary disease hearts beat differently – the divergent mortality trajectories of atrial fibrillation vs other arrhythmias
MR Sarfraz, MF Hemida, A Ali, S Ishtiaq, K Patel, M Hussein, P Tabasum, A Basit Kayani, H Mehmood, I Mushtaq, S Rehman

**2.35pm** **S99**
Cardiac arrhythmias during stable COPD
S Bartlett-Pestell, M Ogden, B Chong, R Lopez, A Braddy-Green, J Mah, H Shahbakhti, LJ Finney, A Marion, L Alves Moreira, H Mullerova, J Marshall, JP Allinson, T Wong, JA Wedzicha

**2.50pm** **S100**
Relationship of cardiac biomarkers in COPD at stable state and exacerbation
P Dobson, W Ee, J Baker, S Cass, S Ramakrishnan, M Bafadhel, REK Russell

**3.05pm** **S101**
Impact of dupilumab in patients with chronic obstructive pulmonary disease with cardiovascular or metabolic comorbidities: BOREAS and NOTUS trials
JR Hurst, P Rogliani, KF Rabe, NM Hawkins, NN Hansel, CF Vogelmeier, SP Bhatt, C Xia, J Heble, M Soliman

**3.20pm** **S102**
The potential effect of Glucagon-Like Peptide 1 Receptor Agonists (GLP1s) on chronic obstructive pulmonary disease (COPD) exacerbations, a case control analyses
B Alcázar, H Heatley, A Kaplan, TT Lee, MS Koh, N Sattar, J Townend, D Skinner, V Carter, D Price

**3.35pm** **S103**
The effects of sodium/glucose co-transporter-2 (SGLT-2) inhibitors in COPD patients' admission frequency, duration and use of acute NIV.
A single-centre 48-month retrospective observational study in a UK tertiary care centre
A Kan, J De Soyza, R Rao, N Murthy, J Bhat

**3.15pm-4.15pm**
**Whittle & Fleming and Britten, 3rd floor**

## COFFEE/TEA BREAK and visit the exhibition stands

**4.15pm-5.15pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

**Nurse**

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**4.15pm-5.45pm**
**Churchill, ground floor**
**SYMPOSIUM**

## TRANSFORMING RESPIRATORY ACROSS THE CURRENT LANDSCAPE

*Chaired by: Ms Maria Parsonage (Carlisle) and Professor Richard Russell (London)*

| | |
|---|---|
| **4.15pm** | Introduction |
| **4.20pm** | Highlights from the latest BTS workforce document – Perspectives from:<br>Dr Daniel Smith (Mansfield)<br>Professor Anne-Marie Russell (Birmingham)<br>Mrs Lizzie Grillo (London) |
| **4.40pm** | A perspective from Wales<br>Dr James Calvert (Newport) |
| **5.00pm** | The respiratory transformation partnership for England<br>Dr Jonathan Fuld (Cambridge) |
| **5.20pm** | Discussion and Q&A |

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**4.15pm-5.45pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## PERSPECTIVES IN PULMONARY VASCULAR DISEASE: MECHANISMS, MONITORING AND MANAGEMENT

*Chaired by: Dr Colin Church (Glasgow) and Dr Nina Karia (London)*

| | |
|---|---|
| **4.15pm** | Contemporary evidence for the interventional management of pulmonary embolism<br>Professor Stavros Konstantinides (Mainz) |
| **4.45pm** | Devices for remotely monitoring pulmonary hypertension<br>Professor Alexander Rothman (Sheffield) |
| **5.15pm** | Latest updates in genetics and management of hereditary haemorrhagic telangiectasia and pulmonary AVM<br>Professor Claire Shovlin (London) |

*Learning objectives*

*1) Evaluate the emerging data on catheter directed therapy and explore the increasing complexity of management of pulmonary embolism.*

*2) Review how implantable devices improve remote monitoring in PH, enabling early detection of clinical worsening.*

*3) Discuss recent advances in the genetic understanding of hereditary haemorrhagic telangiectasia and the management of pulmonary AVMs.*

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**4.15pm-5.45pm**
**Windsor, 5th floor**
**SYMPOSIUM**
**JOINT BTS/BPRS SYMPOSIUM**

## ABNORMALITIES OF LUNG DEVELOPMENT – LOOKING TOWARDS A BRIGHTER FUTURE

*Chaired by: Dr Don Urquhart (Edinburgh) and Professor Louise Wain (Leicester)*

| | |
|---|---|
| **4.15pm** | Defining the new era of prematurity-associated lung diseases<br>Professor Sailesh Kotecha (Cardiff) |
| **4.45pm** | Classification and radiological features of congenital lung anomalies<br>Dr Tom Semple (London) |
| **5.15pm** | Can we fix it? Repairing damaged lungs<br>Dr Charlotte Dean (London) |

*Learning objectives*

*1) To understand the range of developmental lung disorders and their radiological appearances.*

*2) To learn about therapeutic approaches to prevent or ameliorate the development of bronchopulmonary dysplasia.*

*3) To review new advances and novel approaches to treatment, including lung regeneration.*

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**4.15pm-5.45pm**
**Albert, 2nd floor**
**OPEN SESSION – LIFEARC**

## TRANSLATIONAL RARE LUNG DISEASE RESEARCH

*Chaired by: Professor Kevin Dhaliwal (Edinburgh) and Ms Philippa Thomas (London)*

| | |
|---|---|
| **4.15pm** | Introduction, overview and PPIE<br>Ms Philippa Thomas (London) and Professor Kevin Dhaliwal (Edinburgh) |

**4.30pm** In vitro models of cystic and fibrotic lung diseases for high-throughput drug screening
Dr Sara Cuevas Ocaña (Cambridge)

**4.50pm** Translational rare disease modelling
Professor Simon Johnson (Nottingham)

**5.10pm** Application of gene therapy to PCD
Professor Pleasantine Mill (Edinburgh)

**5.30pm** Studying drug action in the human lung
Dr Nikhil Hirani (Edinburgh)

*Learning objectives*

*1) To understand the aims and structure of the LifeArc Translational Rare Respiratory Disease Centre.*

*2) To gain insight into methods of studying genetic lung diseases and how this can be applied to develop new therapies.*

*3) To gain insight into complex human cell culture models and how these can be applied to high throughput drug screening platforms.*

*4) To understand how new genetic therapies can be used to target diseases such as PCD which are resistant to pharmacological therapies.*

*5) To gain insight into cutting edge technologies to study drug action in the human lung.*

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**4.15pm-5.50pm**
**St James, 4th floor**
**SPOKEN SESSION: S104-S109**

## Deep dive into the asthma airways

*Chaired by: Dr Sarah Diver (Leicester) and Professor Timothy Hinks (Oxford)*

**4.20pm** **S104**
Blood and airway eosinophils in severe eosinophilic asthma are phenotypically and functionally distinct
J Gates, P Khooshemehri, S Cass, J Baker, A del Rosario, V Kanabar, J Dhariwal, AM Nanzer, RT Martinez Nunez, DJ Jackson

**4.35pm** **S105**
The airway transcriptome in type2 cytokine biomarker-high and -low severe asthma
J Shen, R Chaudhuri, S Bicknell, A Mansur, R Shrimanker, I Pavord, S Fowler, V Brown, L McGarvey, P Howarth, J Arron, L Heaney, D Choy, E Marchi, T Hinks, P Bradding

**4.50pm** **S106**
Spatial transcriptomic profiling of airway inflammation and structural changes in asthma
J Melhorn, RY Yamani, B Brassanyi, E Bridges, J Norman, ID Pavord, S Taylor, E Marchi, TSC Hinks

**5.05pm** **S107**
Can plasma cell-free DNA methylation predict response to Mepolizumab in asthmatic individuals?
P Rajasekar, J Patel, JL MacIsaac, MS Kobor, YL Pang, I Sayers, D Shaw, RL Clifford

**5.20pm** **S108**
Mepolizumab treatment influences airway remodelling and improves epithelial cell biology in severe asthma as determined by single cell transcriptomics
O Zanin, D Freydina, V Kanabar-Raivadera, J Naftel, W Soe, P Dennison, A Eminton, J Ward, K Horton, L Lau, C Blume, E Swindle, R T Martinez-Nunez, H Rupani

**5.35pm** **S109**
B cell receptor repertoire sequencing to characterise antibody subclass expression and somatic hypermutation in severe eosinophilic asthma and EGPA
J Arora, E Bessell, S Beyatli, D Thenet, I Kontra, J Brown, A Nissim, M Lewis, LK James, PE Pfeffer

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**4.15pm-6.05pm**
**Rutherford, 4th floor**
**SPOKEN SESSION: S110-S116**

## Occupational hazards of exposure to particulates

*Chaired by: Dr Johanna Feary (London) and Dr Ryan Hoy (Melbourne)*

**4.20pm** **S110**
Location and deprivation in silicosis cases: data from the SWORD scheme 1996-2024

RE Wiggans, L Byrne, D Fishwick, MJ Van-Tongeren, M Carder, CM Barber, M Johnson

**4.35pm** **S111**

Sarcoidosis AND Silica: a case-contol sTudy of OccupatioNal Exposures (SANDSTONE study)

CC Huntley, K Nettleton, S Wallbanks, C Reilly, H Morgan, T Duffin, A Crawshaw, T Kharodia, A Phillips, S Coelho, H Bothwell, A Fahim, U Tomo, S Bikmalla, H Stone, S O'Hickey, A Durie, G Hagan, G Burge, AM Turner, PS Burge, GI Walters

**4.50pm** **S112**

Description of 32 cases of artificial stone silicosis from a single UK centre

J Feary, T Kabir, J Szram, A Devaraj, P Howlett

**5.05pm** **S113**

Silicosis prevalence and association with cumulative silica exposure among current and ex- underground small-scale miners in Northern Tanzania

P Howlett, B Said, E Mwanga, OM Kon, A Amaral, S Mpagama, J Feary

**5.20pm** **S114***

Longitudinal change in respiratory function in firefighters; results from the Grenfell Firefighter Study

S Barathan, S Schofield, T Kabir, A Bevan, J Feary

**5.35pm** **S115**

Asbestos-related respiratory disease in UK Armed Forces personnel: reports from the SWORD Surveillance Scheme (1996–2023)

GI Walters, RE Wiggans, L Byrne, M Carder, Z Iheozor-Ejiofor, M van Tongeren

**5.50pm** **S116**

Biological therapy in severe occupational asthma

S Ali, H Badri, S Johnson, K Ballance, E Karpetis, M Cheadle, T De Souza, J Chan, R Barraclough, T Pantin, R Wiggans, JL Hoyle, NJ Meghani, N Sehgal

*S114 – BTS Medical Student Prize Highly Commended

**4.15pm-6.05pm**
**Westminster, 4th floor**
**POSTER DISCUSSION: P148-P162**

## Risk predictors and diagnostics in COPD

*Chaired by: Professor Shawn Aaron (Ottawa) and Professor Jennifer Quint (London)*

**P148** AI-driven identification of high-risk COPD patients for biologic therapy: pathway development opportunities

C Carlin, S Burns, A Cushing, A Taylor, E Walker, D Anderson, DJ Lowe

**P149** The landscape of marketed and pipeline diagnostic tests for chronic lung conditions

SM Warburton, S Williams, M Tseng, K Green, EJ Kennington, R Holmes

**P150** Diagnostic classification of the emphysema subtype of COPD using N-Tidal® capnography

RH Lim, LG Reinders, J Axmann, WT Gaik, L Talker, G Lambert, A Mackay, D Mohan, V Erpenbeck, J Dorn, AX Patel, SO Simons

**P151** Home high-flow therapy: insights from remote-monitoring and linked routine clinical data

L Hickey, E Walker, A Taylor, Z Imtiaz, S Kalli, C Carlin

**P152** Admission blood eosinophil levels in patients with COPD requiring acute Non-Invasive Ventilation (NIV)

JJ Gill, A Manuel, R Russell, A Hughes, P Tinnion

**P153** Associates of, and time to recovery from, eosinopenia in severe exacerbation of COPD (A-TREC)

PJ Ireland, R Sobala, G Mussell, A Prasad, M Scott, J Scott, E Pakpahan, AJ Simpson, J Steer, SC Bourke

**P154** Blood eosinophil counts and escalation to high dose ICS, in COPD patients from a community service

A Beech, T James, Z Paterson, R Danby, T Nagarajan, D Singh

**P155** Determinants of dynamic Troponin rise within 30 days of moderate COPD exacerbation

P Dobson, W Ee, J Baker, S Cass, S Ramakrishnan, M Bafadhel, REK Russell

**P156** Prevalence of cardiopulmonary events and risk factors before COPD diagnosis: ABACOS RISKS Study

R Russell, V Pradeep, J Patrick, C Morgan, H Chen, M Pollack, S Dods, H Mullerova, CF Vogelmeier, D Singh, CP Gale, D Verghese

**P157** Effect of benralizumab on airway function measured by impulse oscillometry during eosinophilic exacerbations: results from the ABRA study
A Lino-Alvarado, M Zheng, S Ramakrishnan, C Mwasuku, REK Russell, M Bafadhel

**P158** The association between GLP-1 receptor agonist use and mortality among COPD patients
H Alaws, J Soto, C Akin, J Mallea, S Helgeson

**P159** Stability and relationship between digitally monitored Inhaled corticosteroids adherence and type 2 biomarkers in COPD
H Aung, K Ntotsis, P Novotny, P Ashley, H Evans, K Balasundaram, W Monteiro, SE Parker, SA Edwards, M Bourne, M Iqbal, M Steiner, RA Evans, CE Brightling, A Murphy, A Wright, NJ Greening

**P160** Can we predict length of hospital stay and mortality using NIVO score for patients admitted with acute exacerbation of COPD requiring non-invasive ventilation?
E Ibrahim, H Brown, R Yadavilli, A Gervasutti, A Amin

**P161** Predictors of in-flight desaturation and need for supplementary oxygen therapy during air travel. Hypoxic Challenge Test (HCT) in patients with Chronic Obstructive Pulmonary Disease (COPD) [Preliminary Results]
V Athanasopoulou, KT Vandorou, I Voulgareli, K Bartziokas, V Papavasileiou, S Loukides, D Toumpanakis

**P162** Changes in blood Cystatin C during COPD exacerbations
Y Owadally, MA MacLeod, S Bartlett-Pestell, S Tan, R Lopez, A Braddy-Green, D Wiseman, J Mah, A Marion, LA Moreira, JP Allinson, JA Wedzicha, H Shahbakhti, LJ Finney

**4.15pm-6.05pm**
**Moore, 4th floor**
**POSTER DISCUSSION: P163-P177**

## Cancer: from screening to therapy

*Chaired by: Dr Theresia Mikolasch (Cambridge) and Dr Helen Morgan (Nottingham)*

**P163** An update regarding the Meso-ORIGINS study: part of the PREDICT-Meso International Accelerator Network
MDJ Neilly, S Tsim, J Maclay, R Bilancia, A Bibby, T Gatheral, A Ionescu, A Griffiths, A Aujayeb, N Rahman, J Fallon, C Craig, M Macdougall, C Daneshvar, R Panchal, S Rajini, M Chetty, A Ashraf, P Short, K Ferguson, E Sage, L Allan, R Crooks, R Reddy, M Morrison, P Reid, A Stanton, K Conroy, B Purdon, L Succony, D DeFoneska, M Hashim, M Haris, J Roche, A MacPherson, L Alexander, N Walker, J Gourlay, J McGarry, K Blyth

**P164** Insights from public interviews in the United Kingdom about the acceptability of risk-based eligibility criteria in lung cancer screening
GK Hutton, E Katsampouris, SE Scott, SW Duffy, M Johansson, HA Robbins, SL Quaife

**P165** A service evaluation of the Targeted Lung Health Check (TLHC) screening programme in a major English city
S Ramadore, B Yee, D Offen, E Woo, A Alzetani

**P166** Lung cancer screening programme in socially deprived East London: outcomes and early cancer detection
GM Tan, A Frater, S Uys

**P167** The risk of lung cancer in the population that declined enrolment in Targeted Lung Health Check programme
A Hussain, J Barnett, A Fitzgerald, I Hussain

**P168** Reassessment of "moderate risk" lung cancer screening participants in the Somerset, Wiltshire, Avon and Gloucester (SWAG) Cancer Alliance
J Chalmers, E Buckley, J Frost, V Masani, A Edey, A Bibby

**P169** Comparative cost-effectiveness of screening for lung cancer using different risk scores and thresholds: the Yorkshire Lung Screening Trial
Y Zhang, S Hinde, C Marshall, N Hancock, I Simmonds, M Soares, R Gabe, D Vulkan, P Crosbie, M Callister, M Callister

**P170** Diagnoses of never-smokers referred to the lung cancer multi-disciplinary team: lung cancer and beyond
G Guvensen, SB Naidu, A Bhamani, T Ahmad, SM Janes, MD Forster, N Navani

**P171** Is there additional diagnostic value of staging computed tomography (CT) to positron emission tomography (PET-CT) in patients with suspected early-stage lung cancer?
M Hussain, V Oakden, E Abugassa, F Ali, MM Hassan, Y Elakhdar, M Ifzaal, A Salman, M Majeed, MB Ganaie, S Khan

**P172** Novel use of Somatostatin receptor imaging with 99mTc-EDDA/HYNIC-TOC in lung neuroendocrine neoplasms
Y Cheng, P Beckett, J Birchall

**P173** Understanding late-stage and emergency lung cancer presentations across Dorset
S Wood, E Saliu, G Ruffino, M Khan, F Rabbani, K Sann, E Strawbridge, H Davies, J Graves

**P174** Utilising pulmonary rehabilitation services to deliver rapid intense prehabilitation for selected lung cancer patients on diagnostic timed pathways
J Dunlop, S Parkinson, P Deus, K Hughes, A Wight

**P175** Using dynamic 19F MRI of lung ventilation properties to prioritise functional lung in radiotherapy treatment planning for non-small cell lung cancer
CW Holland, RA Pearson, P Atherton, D Harrison, A Hassani, H McCallum, FE McDonald, MA Neal, J Phillips, R Pickles, E Smith, A Stamp, H Turnbull, S Warren, N Willis, M Zarroug, P Thelwall

**P176** Central airway obstruction: patient, carer and public perspectives
K Leslie, L Telisinghe, C Daneshvar

**P177** Double jeopardy: escalating mortality trends and disparities in lung cancer patients with sepsis – a retrospective epidemiological study
MF Hemida, MR Sarfraz, T Khan, I Mushtaq, AA Ibrahim, V Chandak, M Al-saadi, Z Sharhiar, A Ali

**4.15pm-6.05pm**
**Abbey, 4th floor**
**POSTER DISCUSSION: P178-P192**

## Respiratory infections: pathogens to patients

*Chaired by: Dr Kay Roy (London) and Dr Anand Shah (London)*

**P178** Clinical and biochemical features of adults hospitalised with Human Metapneumovirus (hMPV): comparison with Influenza A and RSV, insights from the UNIVERSAL study
M Darwish, M Purcell, T Morelli, A Mohammed, P Rodrigues, ABP Jones, PH Lee, A Allen, K Thorne, NJ Greening, M Pavitt, J Myerson, SJ Marciniak, C Daneshvar, MG Crooks, P Mitchelmore, JD Chalmers, S Siddiqui, TW Clark, A Freeman, T Wilkinson

**P179** Asthma reshapes the in-hospital phenotype of adults with respiratory viral infection: heightened symptomatology but attenuated severity
T Morelli, A Jones, P Rodrigues, A Mohammed, M Purcell, A Freeman, T Wilkinson

**P180** The impact of steroids and immunosuppression on outcomes in patients with Pneumocystis Jirovecii Pneumonia (PJP): a retrospective analysis at a large tertiary centre
C Dominic, E Kakouri, V Nanda, S Drazich-Taylor, S Dervisevic, A Kamath

**P181** What is the value of repeat chest radiography in the recovery of patients hospitalised with community-acquired pneumonia?
W Mercer, E Moore, J Lopez, K Thomas, K Bidari, V Fraser, T Bewick

**P182** Chest radiographic consolidation in adults hospitalised with respiratory viral infections (RVI): prevalence and predictors
M Purcell, M Darwish, T Morelli, A Mohammed, P Rodrigues, ABP Jones, PH Lee, KI Thorne, AP Allen, NJ Greening, M Pavitt, J Myerson, SJ Marciniak, C Daneshvar, MG Crooks, P Mitchelmore, JD Chalmers, S Siddiqui, TW Clark, A Freeman, T Wilkinson

**P183** Enhancing antibiotic effectiveness using the repurposed multiple sclerosis drug, glatiramer acetate, to compromise membrane integrity of CF-relevant pathogenic bacteria
A Daly-Gourdialsing, S Gilles, RF Rollo, ACL Micallef, M Zhao, RA Murphy, DA Hughes, T Vorup-Jensen, LP Allsopp, JC Davies

**P184** Readmissions and mortality following index hospitalisation for community-acquired pneumonia: a population-based analysis using HES data
K Bansal, J Harris, J Chalmers, R Scott, R Evans, N Maskell, S Walker, D Arnold

**P185** Incidence of diabetes mellitus following hospitalisation with influenza
S Middleton, TM McKeever, Y Huang, CE Bolton

**P186** Prospective study of antimicrobial RESIstance in chronic lung DIsease (PRESIDE) – a new global registry. Protocol and pilot phase results
K Pates, C Premuda, F Frost, A Gramegna, M Ekkelenkamp, S Thee, A Shah

**P187** Diversity of Pseudomonas aeruginosa in non-cystic fibrosis bronchiectasis: insights from a Norfolk patient population
C Dominic, HV Pye, R Krishnamurthi, CKA Elek, G Beasy, J Ahn-Jarvis, A Hayhoe, EK Mishra, MC Pasteur, EM Adriaenssens

**P188** What does care for adults with non-tuberculous mycobacteria pulmonary disease (NTM-PD) look like? Results from a UK-wide survey
N Sulaiman, A Malhotra, S Ahmed, M Anwar, S Aston, J Ayling-Smith, P Bailey, L Baker, M Bracchi, R Bunce, M Burman, S Capocci, B Choo-Kang, G Clive, M Coleman, S Cowie, A Cowley, S Cowman, D Dhasmana, C Edwards, NP Gbajie, T Gorsuch, B Green, A Hardy, E Holden, B Hulance, K Hurt, N Johl, K Jones, H Kunst, H Lawrence, A Lithgow, V Lloyd, A Malachira, L Martin, R Merriman, D Morgan, A Mughal, S Murphy, N Murray, J Myers, NK Numbere, P Papineni, O Pirzada, S Rashid, A Rosser, N Sabtu, M Shahidi, S Smith, P Tate, D Wat, A White, R Breen, E Rattigan, J Hurst, J Brown, M Lipman

**P189** Mycobacterium chimaera predominance and host vulnerability in non-CF bronchiectasis – A UK cohort study
K Goh, H Saumtally, AP Sawant

**P190** Management of non-tuberculous mycobacterial pulmonary disease (NTM-PD): a European wide NTM-NET survey
R Merriman, AM Malhotra, N Lorent, H Kunst

**P191** Investigating the relationship between Interleukin-6 (IL6) and health outcomes one year after hospital discharge for COVID-19
MM Iqbal, K Ntotsis, LV Wain, CE Brightling, RA Evans

**P192** Pulmonary long COVID is associated with airway microbial dysbiosis and immune dysregulation
M Kusumoto, J McKenzie, S Bamashmous, H Chown, CG Green, MM Jackson, S Laverty, T Shahridan Faiez, F Kamal, A Reed, X Xu, P Kelleher, PM George, L Heaney, T Abujamel, A Shah, A Singanayagam

**5.45pm-7.00pm**
**Britten, 3rd floor**

## BTS PRESIDENT'S RECEPTION

*All participants registered to attend on Thursday 27 November are warmly invited to attend this social occasion*

# SCIENTIFIC PROGRAMME

# Friday 28 November 2025

**8.00am-9.00am**
**Whittle & Fleming, 3rd floor**
## COFFEE/TEA and visit the exhibition stands

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**8.45am-2.00pm**
**Whittle & Fleming, 3rd floor**
**POSTER VIEWING**
*Authors present: 10.00am-11.00am*

### P193-P207
### The logic of biologics
*Discussion of abstracts will take place from 1.30pm to 3.20pm in the St James, 4th floor*

### P208-P222
### The interventionalist's toolkit: bronchoscopy to thoracoscopy
*Discussion of abstracts will take place from 1.30pm to 3.20pm in the Westminster, 4th floor*

### P223-P237
### Ventilatory support in focus
*Discussion of abstracts will take place from 1.30pm to 3.20pm in the Moore, 4th floor*

### P238-P252
### New insights from the fundamental sciences
*Discussion of abstracts will take place from 1.30pm to 3.20pm in the Abbey, 4th floor*

### P253-P268
### Updates in lung disease diagnostics (and PFTs)
*Discussion of abstracts will take place from 1.30pm to 3.30pm in the Rutherford, 4th floor*

### P269-P278
### Real world care of pleural fluid and infection
*Discussion of abstracts will take place from 3.30pm to 4.45pm in the Westminster, 4th floor*

### P279-P288
### Advances in pulmonary vascular medicine
*Discussion of abstracts will take place from 3.30pm to 4.45pm in the Moore, 4th floor*

### P289-P303
### More than just inhalers in asthma
*Discussion of abstracts will take place from 3.30pm to 5.20pm in the St James, 4th floor*

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**8.45am-5.15pm**
**Cambridge, 5th floor**
**MODERATED POSTER VIEWING**
## M33-M48
## TB: from bench to bedside
*Discussion of abstracts will take place from 3.15pm to 5.15pm in the Cambridge, 5th floor*

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**8.00am-8.30am**
**Moore, 4th floor**
**JOURNAL CLUB**
## RESPIRATORY PHARMACOLOGY

*Mrs Grainne d'Ancona (London)*

*Learning objectives*

*To review the latest publications and evidence in respiratory pharmacology.*

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**8.30am-10.00am**
**Churchill, ground floor**
**SYMPOSIUM**
## PREDICTING THE FUTURE IN ILD

*Chaired by: Dr Giles Dixon (Bath) and Dr Brintha Selvarajah (London)*

| | |
|---|---|
| **8.30am** | Can we predict ILD by understanding changes in the lung before disease?<br>Dr Alison John (London) |
| **9.00am** | Can we use ILAs to predict ILD?<br>Dr Pilar Rivera-Ortega (Exeter) |
| **9.30am** | AI is the future in ILD<br>Dr Paul Minnis (Antrim) |

*Learning objectives*

*1) To explore recent developments in the understanding of critical pathways and cellular states that result from lung injury and result in development of fibrosis.*

*2) How to manage ILAs from a clinical perspective with better understanding of the prognostic implications.*

*3) To evaluate the current and future roles for the prognostication and management of ILD.*

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# Friday 28 November 2025

**8.30am-10.00am**
**Windsor, 5th floor**
**SYMPOSIUM**

## BREATHING ROOM: CONFRONTING LUNG DISEASES FROM MODERN WORKPLACES

*Chaired by: Dr Clare Burton (Sheffield) and Dr Patrick Howlett (London)*

**8.30am** Breaking bad – addressing silicosis in the age of artificial stone
Dr Ryan Hoy (Melbourne)

**9.00am** Made to measure? Personalised care for work-related asthma
Dr Vicky Moore (Coventry)

**9.30am** At the cutting edge – lessons from SWORD surveillance on evolving workplace risks and lung health
Professor Martie van Tongeren (Manchester)

*Learning objectives*

*1) Recognise emerging UK risks to respiratory health associated with working with artificial stone, containing respirable crystalline silica. Gain insights into recent advances in diagnosis and patient management.*

*2) Explore the latest advances in the understanding of asthma phenotypes linked to workplace exposures, highlighting the implications for personalised diagnosis and management.*

*3) Visualise the latest trends in work-related respiratory disease in the UK and understand how the changing nature of work leads to new risks and clinical presentations.*

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**8.30am-10.05am**
**Westminster, 4th floor**
**SPOKEN SESSION: S117-S122**

## New insights in COPD pathogenesis

*Chaired by: Dr Jonathan Baker (London) and Professor Karl Staples (Southampton)*

**8.35am** **S117**
Biomarkers of accelerated aging and low early-life lung function trajectory in the Avon Longitudinal Study of Parents and Children (ALSPAC)
J Grenville, P Yousefi, RC Richmond, JW Dodd

**8.50am** **S118**
Spatial transcriptomic profiling of airway disease in COPD: investigating the impact of antiviral therapy
M Glenn, JC Kidney, J James, C Greene, H Guo-Parke, KL Redmond, P Erwin, S El Jadid, C Ward, CC Taggart, DA Simpson, DA Linden

**9.05am** **S119**
Dysregulated inflammatory, immune and fibrinolytic pathways are associated with COPD in subjects with alpha-1 antitrypsin deficiency
DA Spittle, J De Soyza, A Pye, AM Turner

**9.20am** **S120**
The role of fibroblast-derived extracellular vesicles in epithelial cell senescence in COPD
NK Tzionis, JV Devulder, PS Fenwick, PJ Barnes, LE Donnelly

**9.35am** **S121**
Effect of a novel senotherapy, geraldol, on cellular senescence markers in epithelial cells
K Najafi, PS Fenwick, JV Devulder, PJ Barnes, LE Donnelly

**9.50am** **S122**
Oestrogen and ER-β: implications for COPD
C Mwasuku, J Baker, L Rose, M Bafadhel

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**8.30am-10.05am**
**Abbey, 4th floor**
**SPOKEN SESSION: S123-S128**

## Patient centred research in respiratory infection: from design to outcomes

*Chaired by: Professor Rachael Evans (Leicester) and Ms Erika Kennington (London)*

**8.35am** **S123**
Research priorities for community acquired pneumonia in the UK: A James Lind Alliance Priority Setting Partnership
DT Arnold, FW Hamilton

**8.50am** **S124**
Development and validation of Chronic Pulmonary Aspergillosis Health Assessment Questionnaire (CPA-HAQ)

D Marghlani, A Alasfour, E Bradshaw, C Kosmidis, M McGouldrick, C Deas, D Connell, A Johar, P Lagergren, T Soukup, A Singanayagam, M Zhifang Ni, D Armstrong-James, A Shah

**9.05am** **S125**

Navigating the BOAT trial development: a PPIE-led journey to improve patient participation in bronchiectasis research

H Upadhyay, A Robinson-Barella, A Husband, A Goodman, P Punja, L Selfe, H Templin, H Madabhushi, A De Soyza

**9.20am** **S126**

Introducing and evaluating the UK's first medicine-focussed patient information resources for non-tuberculous mycobacterial (NTM) disease: what do we find?

J Gadsby, M Lipman, T Capstick, J Hockley, L Rose, T Sinnett, J McCallum, E Rattigan, C Chen

**9.35am** **S127**

Exploring the effect of different respiratory diseases on patients' perspective of antimicrobial resistance: an AMR-Lung/European Lung Foundation global patient survey

DL Sykes, E Papadopoluou, P Goeminne, A Machado, D Marghlani, L McNeil, J Norwell, J Boyd, C Paixão, A Shoemark, M Ekkelenkamp, S Thee, A Shah

**9.50am** **S128**

The association between patient-reported outcomes and pulmonary exacerbation frequency in patients with alpha-1 antitrypsin deficiency

JA Almahmoudi, A Pye, AM Turner

**8.30am-10.20am**
**St James, 4th floor**
**SPOKEN SESSION: S129-S135**

## Asthma remission and beyond

*Chaired by: Dr Andrew Chadwick (Oxford) and Dr Hitasha Rupani (Southampton)*

**8.35am** **S129**

Long term clinical remission on biologics: an analysis of real-world data from the UK Severe Asthma Registry

C Redmond, J Busby, AH Mansur, M Patel, PH Patel, PE Pfeffer, LG Heaney, H Rupani

**8.50am** **S130**

Association between HRT use and asthma attacks: a UK population-based cohort

H Anpalagan, B Lee, T Tan, E Wong, C Bloom

**9.05am** **S131**

Airway physiology is a stronger contributor to asthma exacerbation risk than type 2 inflammation

L Zeitouni, C Popoola, J McKeever, B Lipworth, M Cottini, R Chan

**9.20am** **S132**

Achieving asthma remission with extrafine beclomethasone dipropionate, formoterol fumarate, and glycopyrronium bromide (BDP/FF/GB) pMDI in adults with moderate-to-severe asthma: a post-hoc analysis of the TRIMARAN and TRIGGER Studies

G Bruselle, D Singh, A Papi, JC Virchow, GW Canonica, L Franzini, A Jackson, U Scaffidi-Argentina, A Vele

**9.35am** **S133**

Improvements in FEV1 and St George's Respiratory Questionnaire (SGRQ) at 4 and 16 weeks on benralizumab are strongly associated with clinical remission at 1 year

J Logan, K Wetherall, L Gillespie, A McConnachie, WTN Lee, H Burhan, T Brown, S Faruqi, DJ Jackson, R Kurukulaaratchy, AH Mansur, D Saralaya, SJ Fowler, P Patel, J Brown, J Lordan, S Siddiqui, PJ McDowell, R Djukanovich, ID Pavord, LG Heaney, CE Brightling, R Chaudhuri

**9.50am** **S134**

Dominant clinical improvements and durability of biologic response in severe asthma

L Wiffen, TL Jones, D Neville, TP Brown, AJ Chauhan

**10.05am** **S135**

Twice-yearly depemokimab demonstrates efficacy in patients with asthma across baseline medium- and high-dose ICS subgroups: Phase III SWIFT-1/2 studies

ID Pavord, M Bonini, D Jarreta, N Bird, A Vichiendilokkul, P Chanez

**8.45am-10.15am**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## REMOVING BARRIERS TO BETTER TUBERCULOSIS CARE

*Chaired by: Dr Toby Capstick (Leeds) and Dr Jessica Potter (London)*

**8.45am** TB in inclusion health groups: how can we better serve the underserved?
Dr Emily Shaw (London)

**9.15am** Asymptomatic transmission of TB: science turning dogma on its head
Professor Keertan Dheda (Cape Town)

**9.45am** Approaches to identifying early TB
Dr Pranabashis Haldar (Leicester)

*Learning objectives*

*1) Highlight the prevalence of TB among inclusion health groups and the importance of system-wide approaches to address health inequalities in this setting including patient-centred strategies to promote equitable access to diagnosis and treatment.*

*2) Describe new data that changes our understanding of the asymptomatic transmission of TB, and which is particularly relevant to underserved communities' health and consider what implications this has for clinical and public health policy in the UK.*

*3) Understanding novel advances in immunological discrimination of latent and active TB.*

**8.45am-10.20am**
**Moore, 4th floor**
**SPOKEN SESSION: S136-S141**

## Pulmonary rehabilitation: what works and why

*Chaired by: Mrs Claire Hepworth (Liverpool) and Ms Lynsey Wright (Surrey)*

**8.50am** **S136**

Implementation of the BTS Clinical Statement on Pulmonary Rehabilitation 2023: evidence from a community COPD service

T James, J Kinsey, T Nagarajan, D Singh, A Beech

**9.05am** **S137**

Predictors of meeting the minimal clinically important difference in pulmonary rehabilitation outcomes using a UK national dataset

H Drover, L Wang, M Richardson, JK Quint, R Evans, SJ Singh, MW Orme, E Daynes

**9.20am** **S138**

Timely access to pulmonary rehabilitation in stable COPD and following an acute exacerbation of COPD: insights from the National Respiratory Audit Programme

H Drover, A Adamson, E Rochell, A Lewtas, A Fowles, H Kwok, JK Quint, T Wilkinson, SJ Singh

**9.35am** **S139**

Comparing the effectiveness of centre-based and home-based pulmonary rehabilitation using a UK national dataset

H Drover, L Wang, M Richardson, JK Quint, R Evans, SJ Singh, MW Orme, E Daynes

**9.50am** **S140**

The impact of multiple long-term condition profiles on exercise capacity in those enrolled in pulmonary rehabilitation: a latent class and regression analysis

O Fiassam, E Daynes, M Baldwin, N Gardiner, SJ Singh, J Manifield

**10.05am** **S141**

An interim analysis of the long-term outcomes of an exercise-based rehabilitation programme in previously hospitalised COVID-19 survivors

S Chakraverty, E Hume, D Megaritis, G Cucato, E Neto, E Daynes, R Evans, S Singh, C Echevarria, I Vogiatzis

**9.00am-10.00am**
**Rutherford, 4th floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Critical Care, Respiratory Failure and Mechanical Ventilation

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**9.00am-10.00am**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## BTS/ARTP – Physiology

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**9.00am-10.00am**
**Albert, 2nd floor**
**OPEN SESSION**

## From data to impact: using NRAP

*Chaired by: Aleksandra Gawlik-Lipinski (Leicester) and Professor Alice Turner (Birmingham)*

**9.00am** Research data considerations in NRAP
*(What can and can't be done with NRAP data, identifying potential applications of the data and the insights aimed to be generated)*
Dr Alexander Adamson (London)

**9.20am** Research conducted using NRAP data
*(An overview of published research, upcoming work, the application process and governance)*
Professor James Dodd (Bristol)

**9.40am** Where does improvement stop and research begin?
*(Pulmonary rehabilitation context – healthcare improvement compared to research)*
Professor Sally Singh (Leicester)

*Learning objectives*

*1) To demonstrate potential and limitations of NRAP data in addressing research questions.*

*2) Understand the breadth of research conducted to date using NRAP data.*

*3) Understand difference between Healthcare Improvement (HI) analysis and research analysis.*

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**10.00am-11.00am**
**Whittle & Fleming and Britten, 3rd floor**

## COFFEE/TEA BREAK and visit the exhibition stands

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**10.30am-11.30am**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Occupational and Environmental Lung Disease

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.30am-11.30am**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## COPD

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**10.30am-11.50am**
**Rutherford, 4th floor**
**SPOKEN SESSION: S142-S146**

## Translational pulmonary vascular science

*Chaired by: Dr Andrew Cowburn (London) and Dr Elaine Soon (Cambridge)*

**10.35am** **S142**
Proteomic signatures for GCN2 and BMPR2-mutation driven pulmonary vascular disease
E Swietlik, M Schwiening, L de Batisda-Casero, NW Morrell, A Cruz-Utrilla, E Oliver, SJ Marciniak, E Soon

**10.50am** **S143**
StratosPHere 1 Study – a novel BMP target engagement biomarker panel for use in clinical trials demonstrates sotatercept alters gene expression in PAH
RJ Jones, EMDD De Bie, N Deliu, AYKC Ng, BJ Dunmore, S Gräf, C Guignabert, M Humbert, L Savale, L Tu, A Boucly, J Newman, G Polwarth,

PD Upton, A Lawrie, CJ Rhodes, MR Wilkins, SK Binmahfooz, AMK Rothman, A Hemnes, SS Villar, J West, MR Toshner

**11.05am** **S144**
Investigation of whole body and tissue specific metabolic profiles during the development of pulmonary arterial hypertension in the monocrotaline rat model
CY Xie, AS Cowburn

**11.20am** **S145**
Interrogation of the glycolytic axis in pulmonary arterial hypertension models and how modifying these can lead to potential therapeutic options
R Sutcliffe, I Cuthbertson, S Moore, B Dunmore, N Morrell, P Caruso, E Soon

**11.35am** **S146**
Inflammation and obesity correlate in pulmonary hypertension but associate with diverging outcomes
EMDD De Bie, P Correa-Jaque, R Jones, HJ Bogaard, J Chan, C Church, JG Coghlan, A Gaur, S Ghio, HA Ghofrani, ZM Goh, LS Howard, M Humbert, G Kovacs, A Lawrie, J Lordan, WY Lin, D Neelam-Naganathan, J Newman, CJ Rhodes, K Sheares, O Sitbon, TW Willis, SJ Wort, S Gräf, DG Kiely, RL Benza, AMK Rothman, C Wallace, MR Toshner

**10.30am-12.00pm**
**Churchill, ground floor**
**SYMPOSIUM**

## APPROACHING RESPIRATORY PHYSIOLOGY WITH A DIFFERENT PERSPECTIVE

*Chaired by: Ms Laura Jess (Edinburgh) and Dr Helen Ward (Wolverhampton)*

**10.30am** Individualised lung function monitoring
Dr Sanja Stanojevic (Halifax, Canada)

**11.00am** Using BOLD to shed light on the small airways
Dr Andre Amaral (London)

**11.30am** Is one enough in lung function testing?
Mr Joshua Barnes (Cambridge)

*Learning objectives*

*1) To explore the alternative options to the current method of determining whether an individual's lung function can be considered 'normal or abnormal' and to reflect on other potentially more appropriate methods at monitoring an individual's change in their lung function.*

*2) To demonstrate the utility of assessing small airway function and its association with respiratory symptoms, cardiometabolic disease and quality of life.*

*3) This presentation will allow reflection on whether it is necessary to perform lung function to strict international guidance in every instance if they do not impact clinical management. Whether there is the option to perform fewer manoeuvres without changing the outcomes for the patient.*

**10.30am-12.00pm**
**Windsor, 5th floor**
**SYMPOSIUM**

## EX-VIVO LUNG PERFUSION: LESSONS IN PATHOPHYSIOLOGY AND THERAPEUTICS FROM LUNG TRANSPLANTATION

*Chaired by: Dr Clementine Fraser (Birmingham) and Dr Caroline Patterson (Cambridge)*

**10.30am** Current status and challenges in the UK
Dr Caroline Patterson (Cambridge)

**10.40am** Breathing outside the body: the evolution of ex-vivo lung perfusion
Professor Anna Reed (London)

**11.05am** Deciphering mechanisms in acute lung injury: lessons from EVLP
Professor Andrew Fisher (Newcastle upon Tyne)

**11.30am** EVLP as a therapeutic platform for modulating lung biology
Dr Matthew Hartwig (Durham, North Carolina)

*Learning objectives*

*1) To appreciate the physiology of ex-vivo lung perfusion and evolution of its clinical utility to assess and recondition marginal donor lungs for transplantation.*

*2) To understand how EVLP helps us decipher the pathophysiology of acute lung injury and identify therapeutic targets to improve early outcomes after lung transplantation.*

*3) To recognise the potential for EVLP as a therapeutic platform for advanced therapies to the lung both prior to transplantation and for other indications.*

**10.30am-12.05pm**
**Westminster, 4th floor**
**SPOKEN SESSION: S147-S152**

## Pleural disease – basic science, new trials

*Chaired by: Dr Anthony Martinelli (Cambridge) and Professor Najib Rahman (Oxford)*

**10.35am** **S147**
An integrated unbiased multi-omic approach to understand the immune response to pleural infection in a disease context
Y Feng, K Cano-Gamez, E Antoun, A Brown, J Chu, A Elsheikh, B Iqbal, N Manoharan, T Dong, N Rahman, J Knight, N Kanellakis

**10.50am** **S148**
Citrullination of fibrin by PAD4 is an important contributor to fibrinolytic deficiency in pleural infection
S Hollins, JY Chu, N Manoharan, Y Feng, A Elsheikh, A Tzouvelekis, E Koulousousa, F Sampsonas, T Dong, JC Knight, NM Rahman, NI Kanellakis

**11.05am** **S149**
Bacteria Responsible for Indwelling Pleural Catheter Colonisation (BRICC)
DK Sethi, G Wickham, H Felgate, T Seecharran, J Rhodes, S Tsim, K Blyth, N Rahman, M Webber, M Webber, E Mishra

**11.20am** **S150**
REDUCE 2: a randomised feasibility study comparing recurrent therapeutic thoracentesis to indwelling pleural catheter insertion, daily drainage and talc instillation for the management of medically-resistant cardiogenic pleural effusions
H Welch, S Walker, E Tucker, S Patoole, L Stadon, A Milne, A Aujayeb, T Nagarajan, J Corcoran, R Panchal, K Conroy, M Haris, N Rahman, O Kadwani, C Craig, A Ionescu, J Goldring, R Miller, P White, R Bhatnagar, N Maskell

**11.35am** **S151**
When to test for Birt-Hogg-Dubé Syndrome? UK national data
FF Yasin, R Legg, CM Schmid, CM Schmid, SJ Marciniak

**11.50am** **S152**
Pragmatic non-inferiority Randomised trial Investigating Needle aspiration versus ChEst drain for Secondary Spontaneous Pneumothorax. The PRINCE-SSP study
S Walker, E Taylor, A McAndrew, E Carlton, E Chaudhuri, P Ghorpade, C Goodwin, J Connors, A Palmer-Walsh, H Cook, R Luengo-Fernandez, S Creanor, NA Maskell

**10.30am-12.05pm**
**Abbey, 4th floor**
**SPOKEN SESSION: S153-S158**

## The importance of early diagnosis in ILD: novel approaches

*Chaired by: Dr Amanda Goodwin (Nottingham) and Professor Philip Molyneaux (London)*

**10.35am** **S153**
ILA phenotype and progression in ILD patients in a tertiary ILD centre
PA Hatton, ML Battenfield, MD Avoseh

**10.50am** **S154**
Patients with new-onset interstitial lung disease (ILD) do not have higher mortality than those with established ILD when admitted to intensive care with severe acute respiratory failure (SARF)
I Soo, TC Aw, M Gribbon, D Wiseman, M Kokosi, PL Molyneaux, E Renzoni, AU Wells, R Hewitt, V Kouranos, P George, J Alcada, M Passariello, J Dalton, B Garfield, B Patel, S Anwar, D Ransley, D Woods, J Doyle, K Bramley, A Brame, F Chua, S Singh

**11.05am** **S155**
Potential of AI to detect ILD in primary care settings: a real-world evaluation
C Vairelles, M Rakić, E Smets, P Desbordes, SSC Kon, S Sehdev, W D-C Man, M Topalović

**11.20am** **S156**
Targeted clinical management of probands and at-risk relatives in familial pulmonary fibrosis: a regional model with national relevance
C Crook, V Elworthy, SC Stanel, H Hanson, M Bradford, D Baker, T Coats, A Duckworth, J Fasham, B Hudson, A Kulasekararaj, H Longhurst, D Morris-Rosendahl, E Renzoni, CJ Scotton, MA Gibbons, P Rivera-Ortega

**11.35am** **S157**
A single centre retrospective study investigating the utility of bronchoalveolar lavage in the diagnosis of interstitial lung disease
SE Marguerie, P Periwal, W Soe, P Thet, L Veryard, L Ralph, S Kooner-Evans, RM Limbrey, BG Marshall, KM Spinks, MG Jones, SV Fletcher, T Wallis

**11.50am** **S158**
Applications of lung ultrasound in interstitial diseases
EJ Soto Hurtado, E Salcedo Lobera, J Martínez Molina

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**10.35am-12.10pm**
**St James, 4th floor**
**SPOKEN SESSION: S159-S164**

## Thinking outside the (asthma) biologic box

*Chaired by: Dr Jessica Gates (London) and Dr Pujan Patel (London)*

**10.40am** **S159**
Biologic therapy is green! Findings from the Asthma BioCarbon Study
DL Sykes, S Faruqi, H Cummings, A Bahra, D Sammut, IJ Clifton, G Baynes, S Davies, C Fewings, H Bladon, T Shemald, L Jones, MG Crooks

**10.55am** **S160**
Improved quality of life, early symptom control and reduced surgery with mepolizumab in chronic rhinosinusitis with nasal polyps (CRSwNP): results from the SYNAPSE study
M Wagenmann, E De Corso, L Walrave, L Zhang, A R Sousa, P Howarth, J Mullol

**11.10am** **S161**
Tezepelumab reduces inflammatory biomarkers in adults with severe chronic rhinosinusitis with nasal polyps with and without comorbid asthma: results from the phase 3 WAYPOINT study
BJ Lipworth, A Allahyar, M Cardner, D Sutton, J Cartwright, M Lehmann, G De Baets, D Muthas, A Platt, T Li, V Mankad, J McLaren, JK Han, JD Sherrill

**11.25am** **S162**
Emerging biologic use may reduce the need for corticosteroid treatment and surgery for chronic rhinosinusitis with nasal polyps (CRSwNP) in France, Germany, Italy, Spain and the UK
W Ahmed, J Hwee, A Edgecomb, L Walrave, A Haq, L Turner, F Barrable

**11.40am** **S163**
Managing ABPA with Tezepelumab: a single-centre experience
R Sehajpal, APS Trindade, IB Torre, AM Bucataru, A Shah, D Armstrong-James, P Patel

**11.55am** **S164**
Mepolizumab improves and sustains asthma control and lung function in patients with severe asthma, independent of baseline IgE and FeNO in the real world: 2-year results from REALITI-A
GW Canonica, MC Liu, C Pilette, JK Lee, R Chaudhuri, S Gould, L Zhang, R Alfonso-Cristancho, P Howarth

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**10.45am-12.15pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## BTS AUDIT AND QUALITY IMPROVEMENT

*Chaired by: Dr Andrew Molyneux (Mansfield)*

**10.45am** Overview of BTS QI and Audit Programme
Dr Andrew Molyneux (Mansfield)

**10.50am** Findings from the Endobronchial Ultrasound (EBUS) Pilot Audit
Dr Haval Balata (Manchester)

**11.10am** Using BTS ILD Registry data to drive better care

Dr Nazia Chaudhuri (Ulster) and Helen Morris (Manchester)

**11.30am** Maximising data sources to improve respiratory care
Dr Daniel Smith (Mansfield)

**11.50am** Quality improvement in asthma and COPD
Professor Alice Turner (Birmingham)

**12.10pm** Conclusion

*Learning objectives*

*1) To examine the findings from the first BTS EBUS Pilot Audit.*

*2) To learn more about utilising already collected respiratory data to improve care, including using BTS Registry data to improve ILD care.*

*3) To gain insight on recent work in quality improvement for asthma and COPD.*

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**10.45am-12.20pm**
**Moore, 4th floor**
**SPOKEN SESSION: S165-S170**

## The role of neutrophils in respiratory disease

*Chaired by: Dr Andrew Higham (Manchester) and Dr Emily Watts (Edinburgh)*

**10.50am** **S165**
Acute systemic hypoxia alters circulating neutrophil biomechanical and transcriptional phenotype
A John, S Nakanishi, J Guck, T Peel, DJ Arachchillage, AS Cowburn, KM Lodge

**11.05am** **S166***
Differential roles of the integrins CD11b and CD11c in human neutrophil extravasation across pulmonary and systemic endothelium
DTH Nguyen, IA Sava, Z Zhang, E Vozza, L Flower, N Farahi, C Summers

**11.20am** **S167**
A prospective observational study investigating neutrophil dynamics in idiopathic pulmonary fibrosis
LE Crowley, E Jenkins, T Smith, C Chen, C Mafham, K Spencer, R Mahida, R Stockley, D Thickett, A Scott, D Parekh

**11.35am** **S168**
Neutrophils display phenotypic and functional changes indicative of activation in a novel in vitro model of idiopathic pulmonary fibrosis
TS Smith, E Jenkins, C Mafham, KL Spencer, RY Mahida, DR Thickett, A Scott, D Parekh, LE Crowley

**11.50am** **S169**
Investigating the effect of baricitinib on neutrophilic inflammation in an ex vivo lung perfusion (EVLP) model of Acute Respiratory Distress Syndrome
D Dorrian, J Strickland, A Boyle, S Tandel, R Coll, DF McAuley, C O'Kane

**12.05pm** **S170**
Neutrophil elastase and NETs contribute to disease exacerbation in pleural infection
JY Chu, A Elsheikh, N Manoharan, S Hollins, Y Feng, A Tzouvelekis, E Koulousousa, F Sampsonas, T Dong, JC Knight, NM Rahman, NI Kanellakis

*S166 – BTS Medical Student Prize Winner

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**11.45am-12.45pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Specialty Trainee

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**11.45am-12.45pm**
**Victoria, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## TB and Non-Tuberculous Mycobacteria

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**12.00pm-2.00pm**
**Pickwick, 1st floor and Whittle & Fleming, 3rd floor**

## LUNCH and visit the exhibition stands.

*Exhibition closes at 2.00pm*

*(Lunch is not included in the delegate fee but is available to purchase on site. Card payments only)*

**12.30pm-1.15pm**
**Churchill, ground floor**
**BTS GRAND CHALLENGE GUEST LECTURE**

## FROM TARGET TO TREATMENT: SHAPING DRUGS FOR RESPIRATORY PATIENTS

*Introduced by: Professor Mona Bafadhel (London)*
*Guest Lecturer: Professor Clive Page OBE (London)*

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**1.30pm-2.30pm**
**Albert, 2nd floor**
**BTS SPECIALIST ADVISORY GROUP OPEN MEETING**

## Interstitial and Rare Lung Disease

*All participants are welcome to attend and hear more about the work of the SAGs.*

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**1.30pm-3.00pm**
**Churchill, ground floor**
**SYMPOSIUM**

## TRANSFORMING THE COPD LANDSCAPE: FROM DIAGNOSIS TO DIGITAL INNOVATION

*Chaired by: Dr Wadah Ibrahim (Leicester) and Dr Joanna Shakespeare (Coventry)*

**1.30pm** Can new technologies replace spirometry in the diagnosis of COPD?
Dr Helen Ashdown (Oxford)

**2.00pm** Harnessing precision medicine in the early detection and management of COPD
Professor Shawn Aaron (Ottawa)

**2.30pm** From self-management to telemonitoring: navigating the future of COPD care
Professor Hilary Pinnock (Edinburgh)

*Learning objectives*

*This session aims to provide a forward-looking perspective on integrating novel technologies and precision medicine into the clinical and research landscape of COPD.*
*By the end of this symposium, attendees will:*

*1) Understand the potential role of emerging technologies in redefining the diagnosis of COPD, including how these innovations could complement or even replace traditional spirometry in clinical practice.*

*2) Explore the applications of precision medicine in the early detection and personalised management of COPD, with a focus on tailoring interventions to individual patients to improve outcomes.*

*3) Gain insight into the latest advancements in remote digital health technologies and their ability to enhance the care and monitoring of patients with COPD, including their impact on accessibility, adherence, and long-term disease management.*

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**1.30pm-3.00pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## "THE USUAL SUSPECTS": BAD BUGS AND WHAT IS BEING DONE ABOUT THEM

*Chaired by: Dr Felicity Perrin (London) and Professor Amelia Shoemark (Dundee)*

**1.30pm** Novel data on multi-resistant NTM management
Professor Andres Floto (Cambridge)

**2.00pm** Novel diagnostics for infection (here and now versus the future)
Professor Michael Tunney (Belfast)

**2.30pm** Pseudomonas as a target in bronchiectasis: novel trial data
Dr Merete Long (Dundee)

*Learning objectives*

*1) NTM disease and how to manage it better.*

*2) Understand where novel techniques may fit into new drug development and clinical care.*

*3) Understand complexity of infection in bronchiectasis.*

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**1.30pm-3.00pm**
**Windsor, 5th floor**
**SYMPOSIUM**

## INSPIRING COLLABORATIVE RESEARCH IN RESPIRATORY MEDICINE

*Chaired by: Professor Anthony de Soyza (Newcastle upon Tyne) and Dr Abigail MacKintosh (West Midlands)*

**1.30pm** The power of trainee collaborative research
Dr Pip Nicolson (Birmingham)

**2.00pm** INSPIRE Research to date:
- DIVA-PCP
Dr Mia Cokljat (Oxford) and Dr Catharine Pearce (Torbay)

- INDEX
Dr Giles Dixon (Bath)

- LISP
Dr Eleanor Barton (Bristol) and
Dr Steven Walker (Bristol)

**2.30pm** The future of collaborative research in respiratory medicine
Dr Akhilesh Jha (Cambridge)

*Learning objectives*

*1) Facilitate research experience to contribute to Generic CiP 5 – Carrying out research and managing data appropriately.*

*2) Highlight the important studies undertaken by research collaboratives in other specialities to drive progress in respiratory medicine including research funding.*

*3) Stimulate discussion of future research projects to be undertaken by local or national collaboratives.*

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**1.30pm-3.20pm**
**St James, 4th floor**
**POSTER DISCUSSION: P193-P207**

## The logic of biologics

*Chaired by: Dr Harpreet Ranu (Brighton) and Dr Rashad Salman (Leeds)*

**P193** Development of a multi-modal model to predict asthma outcomes using digital monitoring tools
C Ottewill, C Gill, P Kerr, E MacHale, O Smith, H Doherty, V Brennan, G Greene, RW Costello

**P194** Is T2-guided treatment feasible in asthma exacerbations?
DL Sykes, H Cummings, C Riches, MG Crooks, K Overton, M Robinson, S Faruqi

**P195** Phenotypic characteristics associated with frequent asthma exacerbations on tezepelumab
F Haris, J Gates, F Cefaloni, P Khooshemehri, L Green, M Fernandes, L Thomson, C Roxas, J Lam, G d'Ancona, AM Nanzer, J Dhariwal, DJ Jackson

**P196** The impact of biologics in severe asthma: real-world registry data from the East of England
J Newman, K Zar Mon, E Pryer, M Navarro, L Kannampuzha, S Nasser, A Jha, C Loutsios, M Knolle, C Owen, R Gore, on behalf of East of England Severe Asthma Network

**P197** Dupilumab causes improvements in spirometry ratios in patients with moderate-to-severe asthma, which correlate with changes in fractional exhaled nitric oxide and asthma control
BJ Lipworth, C Taube, P Rogliani, T Shirai, F Wen, FJ González-Barcala, A Altincatal, R Gall, H Awad, M Castro

**P198** Tezepelumab reduces OCS use and improves sino-nasal symptoms in OCS-dependent patients with severe asthma and comorbid chronic rhinosinusitis (overall and with nasal polyps): results from the phase 3b WAYFINDER study
DJ Jackson, NL Lugogo, M Gurnell, LG Heaney, S Korn, G Brusselle, P Chanez, R del Olmo, J-P Llanos, N Keeling, J Moreno Sapena, B Cook, A Parulekar, N Martin, S Chandarana

**P199** Mepolizumab improves and sustains asthma control and lung function in patients with severe asthma independent of baseline blood eosinophil count in the real world: results from REALITI-A at 2 years
GW Canonica, MC Liu, C Pilette, JK Lee, R Chaudhuri, S Gould, L Zhang, R Alfonso-Cristancho, P Howarth

**P200** Real world experience; an interim analysis of effectiveness of tezepelumab in patients with severe uncontrolled asthma across the UK
P Patel, J Dhariwal, A Nanzer, H Burhan, P Pfeffer, G Hayes, H Rupani, K Pink, R Chan, T Fardon, A Mansur, A Smith, J Lipworth, C Lupton, M Watt, M Hale, C Clewes, J Pike, D Jackson

**P201** Twice-yearly Depemokimab efficacy is sustained across seasons in patients with asthma: analyses of pooled phase III SWIFT-1/2 studies
DJ Jackson, A Bourdin, L Jacques, C-Q Zhu, A Vichiendilokkul, P Howarth, F Schleich

**P202** On-treatment clinical remission with tezepelumab in OCS-dependent patients with severe asthma in WAYFINDER
DJ Jackson, NL Lugogo, M Gurnell, LG Heaney, S Korn, G Brusselle, P Chanez, R del Olmo, J-P Llanos, J Moreno Sepena, C Marchese, B Cook, N Martin, S Chandarana, A Parulekar

**P203** Uniform clinical benefit of biologic therapy across a regional severe asthma network
L Wiffen, D Neville, TL Jones, AJ Chauhan, TP Brown

**P204** Suppression of FeNO but not eosinophils with Tezepelumab is associated with improvements in small airways in uncontrolled severe asthma
R Greig, P Suter, R Chan, T Fardon, BJ Lipworth

**P205** Real world outcomes of Tezepelumab in obese and non-obese severe asthmatics
C Ajayi, S Khan, M Drew, L White, J Sullivan, J Marsh, E Lyng, M Sheego, S Gilbey, E Verrier, T Mehmood, A Pillai, B Sutton, R Berair, D Subramanian, S Message, E Idris, M Matonhodze, P Ryan, M Doherty, B Barker, AH Mansur

**P206** Effectiveness of Tezepelumab treatment in patients with severe asthma: real world outcomes from a severe asthma network
S Khan, M Drew, L White, J Sullivan, J Marsh, E Lyng, M Sheego, S Gilbey, E Verrier, T Mehmood, A Pillai, B Sutton, R Berair, D Subramanian, S Message, E Idris, M Matonhodze, P Ryan, M Doherty, B Barker, AH Mansur

**P207** Path to consensus: patient and physicians perspectives on reducing background medication in patients with severe asthma controlled on biologic therapy in the UK
D Jackson, H Burhan, H Cummings, T Fardon, L Heaney, AH Mansur, C Owen, P Patel, H Rupani

**1.30pm-3.20pm**
**Westminster, 4th floor**
**POSTER DISCUSSION: P208-P222**

## The interventionalist's toolkit: bronchoscopy to thoracoscopy

*Chaired by: Dr Amna Burzic (Nottingham) and Dr Jurgen Herre (Cambridge)*

**P208** Sub-glottic stenosis: a rare but clinically significant large airway disease. A single centre experience
HG Kamil, V Oakden, M Kalkat, M Haris

**P209** Safety profile in bronchoscopic sampling of bronchial carcinoid tumours: a retrospective review from a single tertiary centre
M Alzaid, J Yung, H Balata

**P210** The impact of heterogeneity and fissure integrity on the treatment response of airway scaffolds for emphysema-related hyperinflation
A Tana, A Valipour, A Ing, DP Steinfort, CM Orton, JL Garner, A Karir, K Klooster, T Kleem, JP Williamson, JJ Christie, D Koster, K Welz, M Van Dijk, SA Tuck, ML Mayse, DJ Slebos, PL Shah

**P211** Intraoperative real-time thoracic ultrasound to predict lobar collapse and pneumothorax during endobronchial valve insertion for lung volume reduction
R Tawalbeh, A Asqalan, H Hassan, H Spohr, A Dhrampal, P Browne, M Marquette, S Hand, V Kouritas

**P212** The role of EBUS-TBNA with/without EUS-B-FNA in undiagnosed isolated mediastinal lymphadenopathy
V Papavasileiou, A Dounavi, I Voulgareli, S Lampadakis, M Mplizou, V Athanasopoulou, M Sfika, EM Antonogiannaki, S Loukides, T Raptakis

**P213** Impact of nodule size on diagnostic yield in shape-sensing robotic-assisted bronchoscopy
A Tana, LT Chan, H Trainer, O Volhina, A Karir, ES Agaoglu, I Meireles, JL Garner, J Tonkin, CM Orton, PL Shah

**P214** Impact of procedural experience on radiation exposure in robotic-assisted bronchoscopy
O Volhina, H Trainer, LT Chan, A Tana, A Karir, E Agaoglu, I Meireles, J Tonkin, JL Garner, CM Orton, PL Shah

**P215** Implementing robotic-assisted bronchoscopy within the NHS: outcomes from the first 100 cases performed at a large UK diagnostic centre
H Balata, Y Loo, H Alexander, L Houghton, C Rowe, J Roberts, N Moran, E Sobczynska, T Wong, D Kelly, J Joseph, F Rodrigues, S Gold, C Craig

**P216** Robotic assisted bronchoscopy with cone-beam CT imaging for multiple nodules: diagnostic and safety outcomes
LT Chan, H Trainer, A Tana, O Volhina, A Karir, E S Agaoglu, I Meireles, J Tonkin, JL Garner, CM Orton, PL Shah

**P217** Does augmented fluoroscopy with lung vision navigation enhance the diagnostic yield of radial EBUS mini probe guidance for peripheral pulmonary lesions?
M Vele Witharamalage, M Hettiarachchi, SB Mehdi, M Munavvar

**P218** High frequency bronchoscopic microwave ablation for patients with lung cancer: interim results from the United Kingdom
ES Agaoglu, CM Orton, LT Chan, A Tana, A Karir, I Meireles, JL Garner, P Burn, PL Shah

**P219** Ten year audit of Endobronchial Ultrasound (EBUS) biopsies: diagnostic yield and clinical indications (2014-2024)
A Morozow, J Corcoran, T Howell, A Iyer, T Nicholson, L Taylor, C Daneshvar

**P220** A service evaluation of the Endobronchial Ultrasound Service (EBUS), including molecular diagnoses and patient satisfaction at University Hospital Service (UHS)
W Wilkinson, B Marshall

**P221** Feasibility and safety of single-port thoracoscopic pleurodesis with same-site indwelling pleural catheter insertion for management of malignant and unexplained exudative pleural effusion
Y Rahim, S Khan, M Naeem, G Tsaknis, RV Reddy

**P222** Evaluation of drainage and closure methods following Local Anaesthetic Thoracoscopy (LAT): a multi-centre analysis of complications and outcomes
V Oakden, E Finn, Y Dhary, HG Kamil, MH Naseer, A Banerjee, K Figa, A Aujayeb, R Reddy, M Haris

**1.30pm-3.20pm**
**Moore, 4th floor**
**POSTER DISCUSSION: P223-P237**

## Ventilatory support in focus

*Chaired by: Dr Indrajit Sau (Walsall) and Dr Christopher Turnbull (Oxford)*

**P223** Long-term adherence to non-invasive ventilation in patients with myotonic dystrophy
F Fyles, H Clay, J Wood, C Mason, A Bentley, B Chakrabati, R Parker, P Plant, A Bikov, A Manuel

**P224** "NIV? Not for me." A pilot retrospective 'service improvement' qualitative study examining the psychological impact of hospital-based Non-Invasive Ventilation (NIV) when treating acute type 2 respiratory failure
P Quinn, R Hall, K Ling, I Du Rand

**P225** Bronchiectasis is associated with a higher risk for infective exacerbations in patients with chronic hypercapnic COPD receiving long-term non-invasive ventilation
SE Weinberg, N Mostapha, M Injety, A Chai, E Croft, A Bentley, A Bikov

**P226** Utilising Rockwood Clinical Frailty Score (CFS) in evaluating the impact of pre- admission frailty on clinical outcomes in patients admitted to Acute Respiratory Care Unit (ARCU)
A Mehmood, H Hegab, O Idris, H Aslam, A Dwarakanath

**P227** Ethnic diversity in referral and acceptance patterns to a UK tertiary complex ventilation and specialist weaning unit
K Abernethy, P Dey, R Chavali, N Shah, K Al Arfi, S Bokhari, R D'Cruz, N Hart, G Kaltsakas, M Ramsay, S Srivastava, J Steier, PB Murphy, ES Suh

**P228** Evaluating capillary blood gas monitoring as a patient-centred alternative to arterial blood gas in acute NIV patients – A pilot project in the respiratory support unit at a district general hospital
S Mannan, K Irojah, S Dhadda, J Sayao, N Obisi, A Gulati, A Mowat

**P229** Single-centre experience of acute NIV outcome for acute hypercapnic respiratory failure
S Mannan, K Irojah, P Nasa, A Mowat, A Gulati

**P230** Is there any value? A 1 year prevalence study of patients requiring ventilatory support at a spinal cord injury centre
A Forrest, S Morgan

**P231** High flow nasal oxygen: is it suitable for everyone with respiratory failure?
T Woodhead, B Afzal, M Nayyar

**P232** Impact of Acetazolamide in refractory hypercapnic respiratory failure: experience from a respiratory support unit
C Eng, D Ghosh

**P233** Comparison of NIV outcomes in acute hypercapnic respiratory failure: does initiation pH affect the outcome?
A Sivaramakrishnan, V Gadwal, M Cheung, M Ghauri, P Ratnayake, A Rajhan, S Irshad, A Ali, A Thomas

**P234** Predicting in-hospital mortality in a real-world population of ward-based Non-Invasive Ventilation (NIV) in acute exacerbations of COPD

A Ozsancak Ugurlu, A Watson, P Ellis, RK Bange, SS Yangannagari, M Ahmed, A Oakes, MJ Akram, R Mukherjee

**P235** Multidisciplinary non-invasive ventilation outpatient service for patients with motor neurone disease at a tertiary complex ventilation centre
R Enriquez, K Abernethy, N Shah, K Whitby, B Whyte, S Bokhari, K Al Arfi, S Srivastava, R DCruz, P Murphy, G Kaltsakas, M Ramsay, E Suh

**P236** Acute hypercapnic respiratory failure: assessing the incidence in the emergency department
MJ Akram, J Jain, B Rashid, AO Ugurlu, R Mukherjee

**P237** Closing the gap in acute NIV: a scalable nurse-led approach to optimising time-critical care
D Housley, L McCormick, DP Smith, AW Molyneux, M Loy

**1.30pm-3.20pm**
**Abbey, 4th floor**
**POSTER DISCUSSION: P238-P252**

## New insights from the fundamental sciences

*Chaired by: Professor Jane Bourke (Melbourne) and Dr Robert Hynds (London)*

**P238** Spatial transcriptomic profiling at single-cell resolution reveals lung responses to sterile injury
X Zhang, S Li, M Greco, PV Gutierrez, E Bridges, L Cheng, A Simmons, P Klenerman, TSC Hinks

**P239** Endothelial HIF2a signalling modulates hypoxia driven myeloid cell migration into the lung
KM Lodge, S Nakanishi, A Palazon, N Pisacano, RS Johnson, A Cowburn

**P240** Pulmonary extracellular vesicle microRNAs induce alveolar macrophage dysfunction in ARDS
K Spencer, M Lindsay, B Naidu, DR Thickett, A Scott, R Mahida

**P241** Differential methylation analysis in fibrotic versus non-fibrotic sarcoidosis
L Huajian, P Kenny, M Watson, R Gilpen, P Logue, D Linden, P Minnis

**P242** Epithelial cell extrusion promotes the loss of KRT5 with implications for pulmonary fibrosis
K Bhatti, I Uwagboe, J Rosenblatt, M Parsons, G Jenkins, A John, I Stewart, C Dean

**P243** Characterisation of Src-family kinase inhibitors in lung fibroblasts shows distinct signalling complexes required for matrix synthesis and differentiation
M Rossanese, G Marchini, G Modafferi, B Pioselli, G Villetti, R Yuan, R Walters, A Kunzemann Martinez, M Platé, RC Chambers

**P244** The RNA binding protein Insulin Growth Factor 2 Binding Protein 3 (IGF2BP3) is a novel biomarker of T2 high asthma and inhibitor of IL-13/IL-4 signalling in human bronchial epithelium
P Khooshemehri, O Zanin, J Rynne, E Perucha, RT Martinez-Nunez

**P245** The Rev-erba clock agonist (SR9009) protects against house dust mite induced T2 asthma
A Chakraborty, J Cain, R Maidstone, V Deugi, N Begley, D Bechtold, J Gibbs, H Durrington

**P246** Glycine attenuates airway hyperresponsiveness associated with T2 asthma
A Chakraborty, A Alex, V Deugi, M Zhong, F Khanom, A Simpson, B Gaston, J Gibbs, M Plebanski, H Durrington

**P247** GSDMB mediates epithelial to mesenchymal transition (EMT) in human airway epithelial cells
Y Zhang, M Tanro, S Chi, E Chang, M Wang, Z Ma, H Zhao, K Ito, D Ma, WO Cookson, MF Moffatt

**P248** ORMDL3 influences ferroptosis through HMOX1 in human bronchial epithelial cells
S Chi, H Zhao, J Weinman, D Ma, K Ito, WO Cookson, MF Moffatt, Y Zhang

**P249** Knockout of ORMDL3 renders airway epithelial cell resistance to human rhinovirus infection
Y Zhang, H Du, H Guo, L Daly, J Wong, M Wang, E Chang, D Ma, K Ito, WO Cookson, MF Moffatt

**P250** GSDMB regulates human rhinovirus replication and inflammation in airway epithelial cells
H Du, J Ma, M Lei, H Guo, L Daly, E Zhang, M Wang, K Ito, WO Cookson, MF Moffatt, Y Zhang

**P251** Investigation of myeloid-derived suppressor cells as a biomarker for the differential diagnosis of lung diseases
AP Bizymis, AM Matthaiou, A Apostolidou, K Symvoulaki, G Pitsidianakis, N Tzanakis, A Liapikou, I Tomos, N Bizymi, KM Antoniou

**P252** Involvement of myeloid-derived suppressor cells in allergic airway diseases: preliminary results of a systematic review of the literature
N Bizymi, AM Matthaiou, AP Bizymis, K Symvoulaki, E Vasarmidi, N Tzanakis, S Schiza, A Liapikou, I Tomos, KM Antoniou

**1.30pm-3.30pm**
**Rutherford, 4th floor**
**POSTER DISCUSSION: P253-P268**

## Updates in lung disease diagnostics (and PFTs)

*Chaired by: Dr Vicky Moore (Coventry) and Dr Nicholas Weatherley (Sheffield)*

**P253** Impact of applying Global Lung Initiative 2022 race neutral equations to UK tertiary care lung function data
C Roberts, A Braddy-Green, P Raote, JH Hull, JP Allinson

**P254** Home spirometry monitoring as an adjunct to clinical care in patients with fibrotic interstitial lung diseases: findings from the REMOTE-ILD randomised controlled trial
SA Barth, R Borton, C Edwards, E Pizzo, W Adams, N Gay, R Aul, M Gibbons, PL Molyneaux, T Sutherland, F Khan, V Yioe, J Eaden, A Fahim, N Williams, Y Abed El-Khaleq, SL Barratt, G Jenkins, I Stewart, M Wickremasinghe

**P255** Obstructive spirometry in a severe pectus excavatum cohort: data from the National Pectus MDT
S Egan, W Ricketts, R Astin, J Dunning, S Kolvekar, G Wallace

**P256** Observational study of preserved ratio impaired spirometry in the Irish adult population
S Jafri, I Ahmad, A McGowan, A Subramaniam

**P257** Expiratory central airway collapse increases longitudinally in young smokers with preserved lung function
S Ananth, C Ridge, A Braddy-Green, ANJM Marion, LA Moreira, A Beech, CE Bolton, G Choudhury, L McGarvey, D Singh, TM Wilkinson, AI Ritchie, M Govoni, P Jones, H Mullerova, K Ostridge, JC Yates, EA Hoffman, J Vestbo, PM Calverley, JA Wedzicha, JP Allinson, British Early COPD Network (BEACON) Study

**P258** Evaluation of patients with subglotic-tracheal stenosis and utility of pulmonary function tests
EJ Soto Hurtado, E Salcedo Lobera, M García García, Y Rodríguez Gallego

**P259** Should primary care be offering Fractional Exhaled Nitric Oxide (FeNO) testing? A review of raised FeNO levels in patients referred to secondary care with a suspicion of asthma
S McArthur, S Baxter, K Cuthbert

**P260** Bronchial hyperresponsiveness in asthma: the use of direct and indirect challenge testing at diagnosis
WE Smith, LS Whipday, L Willmore, M Bennett, A Simpson, CS Murray, SJ Fowler, R Wang

**P261** Quantifying oxygen requirement in early warning scores improves recognition of deterioration compared to the National Early Warning Score 2 (NEWS2) in patients on oxygen
CH Harrison, S Gerry, OC Redfern, C Subbe, JP Bedford, PJ Watkinson

**P262** Even number bias in pulse oximetry measurements in hospitals in Denmark and the UK
BR O'Driscoll, ND Bakerly, MH Olsen, J Molgaard, EK Aasvang, APH Karlsen, CS Meyhoff

**P263** Assessment of diaphragm motor unit activity using high-density surface electromyography
X Ma, GF Rafferty, EN Kamavuako, CJ Jolley

**P264** The relationship between postural FVC change and respiratory muscle strength in patients with motor neurone disease (MND)
C Anagnostopoulou, E Koukaki, A Vontetsianos, N Chynkiamis, A Papasarantou, G Stratakos, P Bakakos, G Kaltsakas, N Koulouris

**P265** Forced oscillation technique indices are related to upper airway dysfunction in patients living with motor neurone disease (plwMND)
A Vontetsianos, N Shah, N Chynkiamis, C Anagnostopoulou, A Papasarantou, G Stratakos, P Bakakos, N Koulouris, G Kaltsakas

**P266** Transcutaneous electrical stimulation of the upper airway dilator muscles in patients with motor neurone disease and healthy volunteers
A Madkhali, A Almaghrabi, M Alkhathami, G Radcliffe, J Owusu-Afriyie, G Kaltsakas, J Steier

**P267** Assessment of the impact of nebuliser type on fugitive emissions in a treatment room when delivering inhaled medications
JA Suggett, L Clutterbuck, M Nagel

**P268** Extra-fine particle deposition of triple inhaler therapies using the next generator impactor
B Lipworth, E Zambelli, A Cavecchi, L Franzini, A Piraino

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**3.00pm-3.30pm**
**Britten, 3rd floor**
**COFFEE/TEA BREAK** *(Exhibition closed)*

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**3.15pm-4.45pm**
**Churchill, ground floor**
**SYMPOSIUM**

## A LOT OF YAP ABOUT CAP, HAP AND VAP

*Chaired by: Professor Neil French (Liverpool) and Dr Catherine Hyams (Bristol)*

**3.15pm** From the lung microenvironment to novel targets in pneumonia
Professor Charlotte Summers (Cambridge)

**3.45pm** Diagnostics: the new HAP-FAST approach
Dr Daniel Wooton (Liverpool)

**4.15pm** INHALE: rapid molecular methods in VAP
Dr Virve Enne (London)

*Learning objectives*

*1) Understanding more about the cellular mechanisms of acute pulmonary inflammation to help identify novel targets for therapeutic intervention.*
*2) From accurate, feasible and cost-effective diagnosis of pneumonia to also ensuring engagement with wider professional networks such as policy makers when scoping out research plans.*
*3) Exploring the value of rapid molecular testing and implications on care, antimicrobial stewardship and exploring healthcare worker perceptions when engaging in these newer methods and approaches.*

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**3.15pm-4.45pm**
**Mountbatten, 6th floor**
**SYMPOSIUM**

## BRONCHIECTASIS – RECENT ADVANCES AND CLINICAL TRIALS

*Chaired by: Dr Freddy Frost (Liverpool) and Ms Pamela Vaughn (Glasgow)*

**3.15pm** Disease progression, lung function and novel imaging in bronchiectasis
Professor Harm Tiddens (Rotterdam)

**3.45pm** Physio, mucus: CLEARing the way?
Professor Judy Bradley (Belfast)

**4.15pm** Novel data and novel targets: cutting edge trials for bronchiectasis
Professor James Chalmers (Dundee)

*Learning objectives*

*1) Understand where novel imaging techniques may fit into new drug development and clinical care.*

*2) Better understanding of physiotherapy and managing airway clearance emerging in bronchiectasis.*

*3) Better understanding of new therapies emerging in bronchiectasis.*

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**3.15pm-4.50pm**
**Windsor, 5th floor**
**SPOKEN SESSION: S171-S176**

## How the eosinophil and T2 biomarkers guide decisions in COPD

*Chaired by: Dr Lydia Finney (London) and Professor Dave Singh (Manchester)*

**3.20pm** **S171**
Clinical utility of FeNO in a tertiary COPD clinic
F Hickey, M Cuddihy, B Cushen

**3.35pm** **S172**
Digitally monitored inhaled corticosteroids adherence predicts clinical outcomes in COPD stratified by type 2 inflammation: a comparative analysis of blood eosinophils and FeNO
H Aung, P Novotny, K Ntotsis, K Balasundaram, P Ashley, H Evans, W Monteiro, SE Parker, SA Edwards, M Bourne, M Iqbal, M Steiner, RA Evans, CE Brightling, A Murphy, A Wright, NJ Greening

**3.50pm** **S173**
Eosinophilic phenotype and bacterial load in hospitalised patients with exacerbations of COPD
A Thumbe, N Ahmad

**4.05pm** **S174**
Reduction of exacerbations according to type 2 inflammatory biomarkers with dupilumab treatment in patients with chronic obstructive pulmonary disease
S Couillard, S Ramakrishnan, I Pavord, Y Çolak, R Buhl, G Deslée, D Bauer, M Soliman

**4.20pm** **S175**
Tracking eosinophils following prednisolone or Benralizumab at the time of an exacerbation in the ABRA trial
N Smallcombe, M Zheng, S Ramakrishnan, S Cass, C Mwasuku, K Krassowska, L Bermejo-Sanchez, REK Russell, M Bafadhel

**4.35pm** **S176**
A subgroup analysis of COPD-HELP – Mepolizumab for COPD with eosinophilic phenotype following hospitalisation – stratified by eosinophil levels
CA Flynn, HJC McAuley, O Elneima, HWWW Aung, W Ibrahim, TJC Ward, M Bourne, T Thornton, V Mistry, HR Gilbert, AKA Wright, RA Evans, MC Steiner, M Richardson, CE Brightling, NJ Greening

**3.15pm-5.15pm**
**Cambridge, 5th floor**
**MODERATED POSTER DISCUSSION: M33-M48**

## TB: from bench to bedside

*Chaired by: Professor Keertan Dheda (Cape Town) and Dr Felicity Perrin (London)*

**M33** Localisation and ultrastructure of Mycobacterium tuberculosis in human mediastinal lymph nodes by high resolution electron microscopy
A Fearns, J Murray, C Tweed, M Omar, P Naran, E Thompson, A Patel, MG Gutierrez, M Lipman, TD Pillay

**M34** Semi-automated quantification of PET-CT in TB contacts and its association with prospective outcomes
JW Kim, A Kamil, J Lee, I Novsarka, P Haldar

**M35** Evaluating the RISK-6 signature as a TB diagnostic using a novel laboratory-based tuberculosis host immune response assay (TB-HIRA): a retrospective pilot study
N Grolmusova, J Lee, I Novsarka, R Verma, W Monteiro, C Morais, K Barr, M Arefi, M Selby, R Page, T Nilsen, P Haldar

**M36** Peripheral blood inflammatory and immune biomarkers as correlates of TB infection phenotypes characterised by PET-CT: a prospective cohort study in household contacts
JW Kim, A Kamil, J Lee, I Novsarka, W Branchett, A O'Garra, P Haldar

**M37** The burden and determinants of tuberculosis among small-scale miners and community members in Northern Tanzania
P Howlett, B Said, E Mwanga, A Amaral, OM Kon, J Feary, S Mpagama

**M38** Effect of chest radiograph reporting codes on time to diagnosis of suspected pulmonary tuberculosis (PTB) – a retrospective cohort study
N Grolmusova, A Gilmour-Caunt, H Patel, H Hopewell, G Woltmann, R Verma, I Das, P Haldar

**M39** A piece of the diagnostic puzzle: could histological results be used to trigger a specialist TB review?
R Naran, C Tweed, T Odubanjo, M Yasser, S Lozewicz, M Lipman, J Brown, J Potter

**M40** When does TB PCR change management? A retrospective case series examining role of PCR in initial and final TB diagnosis in a general hospital
AD McGown, K Nyikadzino, S Moore

**M41** High frequency of asymptomatic mediastinal lymph node TB diagnosed via EBUS: a retrospective cohort study from a UK TB-Network
F Fama, AT Read, J Murray, D Creer, M Belton, M Brown, A Garcia Mingo, J Goldring, P Gothard, H Ip, H Jarvis, S Lee, Z Mangera, R Moores, N Navani, A Patel, R Thakrar, C Tweed, C Valerio, JP Brown, RK Gupta, JL Potter, S Capocci, MCI Lipman

**M42** A multi-centre audit of the diagnosis and management of lymph node tuberculosis
E Rooney, TS Anand, J Sturman, SH Jung, D Newport, K Banner, MK O'Shea, M Dedicoat, P Haldar, NF Walker

**M43** Hospitalisation for tuberculosis: a retrospective evaluation of burden and length of stay for a UK TB hotspot
H Mansoor, N Grolmusova, R Verma, D Bell, M Pareek, S Bandi, G Woltmann, P Haldar

**M44** WITHDRAWN

**M45** Persistently positive sputum smears with sub-therapeutic Rifampicin levels in pulmonary tuberculosis – a call for change?
R Haider, A Idzik, E Roddy, K Thickett, R Carter

**M46** A meta-analysis of TB treatment outcomes in individuals with silicosis and silica exposure
A Dunlop, J Feary, P Howlett

**M47** Post-TB care in the UK: cross-sectional analysis from a national survey
A O'Reilly, C Martin, D Zenner, P Haldar, J Meghji, M Pareek

**M48** Revealing the hidden workload of TB services: using electronic patient records to support workforce planning
MO Azouaghe, A Hoxha, H Durkan, TB Whyte, P Sparkes, M Coleman, J Meghji, M Park, O Kon, L Martin

**3.30pm-4.45pm**
**Westminster, 4th floor**
**POSTER DISCUSSION: P269-P278**

## Real world care of pleural fluid and infection

*Chaired by: Dr John Corcoran (Exeter) and Dr Jenny Ferguson (Glasgow)*

**P269** Utility of the RAPID score as a risk prediction tool in the management of patients with pleural infection: a systematic review and meta-analysis
A Elsheikh, C Mounsey, B Iqbal, D Addala, W Mei Chew, K Kogh, E Bedawi, O Walsh, M Bhatnagar, E Harriss, S Llaneza Lago, E Mishra, R Hallifax, N Rahman

**P270** The impact of the pleural effusion microenvironment on macrophage phenotype and function
E McNally, A Rehill, K Slattery, A Newing, C Finlay, R Preston, L Gleeson

**P271** Impact of early versus delayed intrapleural fibrinolytics on pleural infection outcomes: an observational study
Y Rahim, M Zubair, H Virk, M Naeem, G Tsaknis, RV Reddy

**P272** Imaging change after intrapleural fibrinolytics in patients with indwelling pleural catheters and septated effusion: a retrospective review of the Oxford Pleural Unit data
OR Walsh, NM Rahman

**P273** A novel ambulatory pleural effusion pathway in a tertiary referral hospital in Ireland reduced pleural effusion admissions by 31% over 9 months
SJ O'Riordan, J Brady, F O'Connor-Power, R Farrell, C Quinn, L Gleeson

**P274** Diagnostic utility and cost effectiveness of pleural fluid analysis during local anaesthetic thoracoscopy
HG Kamil, V Oakden, E Finn, Y Dhary, A Chaudhry, S Khan, M Haris

**P275** Do self-drainers have higher indwelling pleural catheter infection rates than non-self-drainers?
A Baguneid, N Newnham, S Johnstone, F Hinchcliffe, A Hand, D Li, S Mohammad, R Sudhir, R Panchal

**P276** Talc pleurodesis-anything but a dry subject: understanding the challenges of delivering talc pleurodesis as a first-line treatment to patients with malignant pleural effusion in a large UK pleural centre
K Warner, B Iqbal, N Rahman

**P277** LENT score utilisation in Indwelling Pleural Catheter (IPC) pathways: an audit-based recommendation
M Ilyas, T Sathyamoorthy, P Ramachandran

**P278** Unexpected activity of the pleural nurse specialist for IPC patients in a tertiary centre
N Newnham, S Johnstone, F Hinchcliffe, A Hand, A Baguneid, D Li, S Mohammad, R Sudhir, R Panchal

**3.30pm-4.45pm**
**Moore, 4th floor**
**POSTER DISCUSSION: P279-P288**

## Advances in pulmonary vascular medicine

*Chaired by: Dr Nicola Gray (Norwich) and Dr Joseph Newman (Cambridge)*

**P279** Evaluating age-adjusted D-Dimer cut-offs and the role of higher multipliers in pulmonary embolism diagnosis
M Fisher, I Aganin, J Foden, D Edward, M Alsahab

**P280** Incidental Pulmonary Embolism (iPE) and the value of CT pulmonary angiography follow-up versus expert review
CRT Kidson, H Sohn, J Page, JCL Rodrigues

**P281** Diagnostic accuracy of a deep learning model for coronary calcification detection on unenhanced CT in pulmonary hypertension patients
TN Alnasser, A Hokmabadi, M Sharkey, K Dwivedi, M Salehi, DG Kiely, S Alabed, AJ Swift

**P282** Automated three-dimensional measurements of maximum pulmonary artery diameter and ratio with aorta on CT pulmonary angiogram are predictive of pulmonary hypertension in patients with low probability echocardiography
J Rossdale, A Mir, P Charters, H Wong, J Page, R Mackenzie Ross, J Suntharalingam, A Cookson, J Clark, JCL Rodrigues

**P283** Pulmonary hypertension associated with COVID-19-interstitial lung disease
S Mehta, S Herbert, B Garfield, M Heightman, K Myall, B Mukherjee, B Rawal, C Harries, H Nashat, SJ Wort, C McCabe, K Dimopoulos, A Devaraj, S Bax, G Jenkins, A Wells, P George, E Renzoni, LC Price

**P284** Interim results from PHINDER: Pulmonary Hypertension Screening in Patients with Interstitial Lung Disease
S Sahay, MB Scholand, E Belloli, A Swaminathan, M Broderick, D Lee, K Maher, K Paxton, CM Thrasher, D Kiely

**P285** Change in right ventricular ejection fraction in group 3 pulmonary hypertension has prognostic value: insights from the ASPIRE Registry
R Reader, H Abbas, E Cerrone, ZM Goh, S Alabed, K Dwivedi, H Zafar, A Hameed, A Charalampopoulos, JA Hurdman, CA Elliot, ARK Rothman, AJ Swift, R Condliffe, AAR Thompson, DG Kiely

**P286** Is there diurnal or seasonal variation in the 6-minute walk test distance in patients with pulmonary vascular disease?
L Robertson, M Toshner, J Pepke-Zaba, K Sheares, J Cannon, K Bunclark, D Taboada, KP Sylvester, J Newman, B Knox-Brown

**P287** Intrapulmonary shunt detection in HHT: standardized technique for agitated saline (Bubble Echo) studies
L Selvakumar, J Choy, R Rashkovetsky, T Jeerakathil, M Hill, D Vethanayagam

**P288** Risk factors for recanalisation following pulmonary arteriovenous malformation embolisation: results from a tertiary referral centre
CJ Lim, Y Shahin

**3.30pm-5.20pm**
**St James, 4th floor**
**POSTER DISCUSSION: P289-P303**

## More than just inhalers in asthma

*Chaired by: Dr Tom Fardon (Dundee) and Dr Kathryn Prior (Preston)*

**P289** Patient and clinician experiences of a FeNO-guided asthma management intervention in primary care
G Lewis, M Santillo, L Yardley, K Wang, B Ainsworth, S Tonkin-Crine

**P290** Feasibility of a digital-first approach to inhaler optimisation in asthma care: supporting net zero NHS goals
SA Brown, AC Murphy, CE Morris, D Gokal

**P291** Clinical effectiveness of anti-inflammatory reliever therapies (AIR) for exacerbation prevention: a non-inferiority study
M Crooks, F Tyrer, R Malone, A Jackson, R Beasley, V Carter, N Eleangovan, W Henley, I Pavord, P Wark, B Lipworth, D Price

**P292** Effectiveness of extrafine single-inhaler triple therapy on asthma control and quality of life one year post treatment according to patients' smoking status: the TRIANGLE study
P Katsaounou, A Korkontzelou, E Papapostolou, P Katerelos, P Efstathopoulos, K Kostikas

**P293** Exacerbation and rescue medication reduction with extrafine formulation single-inhaler triple therapy in adult asthma patients: outcomes from the TriMaximize UK study
J Richards, A Rostron, R Russell

**P294** The BETRI study: preliminary information on characteristics of patients with asthma or asthma-COPD overlap treated with Medium-Strength Inhaled Corticosteroid / Long-Acting Beta-Agonists (ICS/LABA) and switching to High-Strength ICS/LABA or Medium-Strength Beclomethasone /Formoterol / Glycopyrronium (BDP/FF/G)
G Bruselle, C Grainge, N Roche, A Gayle, V Carter, D Price, L Di Palma, A Piraino, A Jackson, U Scaffidi-Argentina, D Singh

**P295** Treatment with extrafine formulation single-inhaler triple therapy improves health-related quality of life and asthma control – results from the TriMaximize UK study
J Richards, A Rostron, R Russell

**P296** Pressure Metered Dose Inhalers (pMDI) Vs. Dry Powder Inhalers (DPI) for bronchodilation and inhibiting exercise-induced bronchoconstriction in athletes: a systematic review
T Burrell, B Kolosz, M Crooks, A Simpson

**P297** Budesonide/glycopyrronium/formoterol fumarate dihydrate triple therapy for inadequately-controlled asthma: KALOS and LOGOS study results
A Papi, RA Wise, DJ Jackson, N Lugogo, R Chen, T Trasieva, C Obasi, C Movitz, J Helman, P Salter, K Springer, M Bondoc, M Shah, K Knappenberger, K Bowen, H Pandya, A Megally, M Patel

**P298** Time course of exacerbation reduction with extrafine beclomethasone dipropionate, formoterol fumarate, and glycopyrronium bromide (BDP/FF/GB) pMDI in adults with moderate to severe asthma: a post-hoc analysis of the TRIMARAN and TRIGGER studies
D Singh, A Papi, JC Virchow, L Franzini, A Vele, U Scaffidi-Argentina, A Piraino, A Jackson

**P299** Adrenal suppression in asthma
H Butt, HJ Durrington, R Stacey

**P300** Effect of baseline alveolar gas transfer measurement (KCO) on response to biologics in severe asthma in the UK Severe Asthma Registry
PE Pfeffer, T Brown, P Patel, M Patel, AH Mansur, JW Dodd, S Message, K Prior, J Busby, M Masoli, M Doherty, L Heaney, SE Brill, S Doe, S Faruqi, H Rupani

**P301** Eosinophilic inflammation in asthma and COPD: a UK biobank study
H Emms, N Aveyard, M Masoli, J Masoli

**P302** The virtual patient stratifies patient-specific response to bronchial thermoplasty with high accuracy
H Kaul, RJ Russell, M Ola, M Richardson, M Laviolette, J Chakir, JT Annema, P Howarth, P Chanez, R Chaudhuri, ASDD Singapuri, D Stolz, DM Schumann, DK Hogarth, M Castro, P Bonta, CE Brightling

**P303** Real world assessment of impact of digital home monitoring in difficult-to-treat asthma
CM Gill, LM Piggott, E Babikir, C Ottewill, S Shelley, O Smith, H Doherty, E MacHale, G Greene, BD Kent, RW Costello

**Declarations of Interest**

Please see page A371 onwards for declarations of interest relating to Spoken, Poster and Moderated Poster abstracts.

# SPEAKERS' BIOGRAPHICAL DETAILS

## Biographical details for Speakers and Chairs in Symposia, Guest Lectures and Journal Clubs

**Dr Shawn Aaron** is a Professor of Medicine at the University of Ottawa in Ottawa, Ontario, Canada. He is a Respirologist at the Ottawa Hospital and a Senior Scientist at the Ottawa Hospital Research Institute. His research focuses on asthma, COPD and cystic fibrosis. He conducts clinical trials and cohort studies related to correct diagnosis and treatment of these conditions. Dr Aaron's most recent research has concentrated on case-finding studies to discover people with undiagnosed asthma and COPD, in an effort to diagnose and treat these patients earlier and improve their health outcomes.

**Professor Sanjay Agrawal** is National Specialty Advisor for Tobacco Dependency at NHS England, RCP Special Advisor on Tobacco and a Consultant in Respiratory and Intensive Care Medicine in Leicester. Sanjay has led past iterations of the BTS National Hospital Tobacco Audit. In 2023 he co-delivered the BTS Quality Improvement Programme in Tobacco Dependency and co-chaired the BTS Clinical Statement on the Medical Management of Tobacco Dependency, published in 2024. Sanjay chairs the RCP Tobacco Advisory Group publishing regular reports and recommendations on all aspects of tobacco control.

**Dr André Amaral** is a Senior Lecturer in Respiratory Epidemiology and Global Health at the National Heart and Lung Institute of Imperial College London. He leads an epidemiology research group whose work focuses on understanding the causes of poor lung function and chronic lung disease in populations from around the globe. Dr Amaral is the Project Lead of the Burden of Obstructive Lung Disease (BOLD) study, as well as the principal investigator of a network focusing on multimorbidity in sub-Saharan Africa. In addition, Dr Amaral is Associate Editor of the *International Journal of Epidemiology* and *BMJ Open Respiratory Research*. He is also Chair of the Epidemiology Group of the European Respiratory Society.

**Dr Harry Apperley** is a Paediatric Registrar currently working in Paediatric Respiratory Medicine at King's College Hospital, London. Harry recently completed a post at the Royal College of Paediatrics and Child Health as a Clinical Fellow, working on education, policy and advocacy on the impact of air pollution on child health, in partnership with the Clean Air Fund. He has interests in environmental health and the impact of health inequalities; particularly their clinical interface with respiratory medicine.

**Dr Kerri I Aronson** is an Assistant Professor of Medicine in the Division of Pulmonary and Critical Care Medicine at Weill Cornell Medicine in New York City. She cares for patients in the Interstitial Lung Disease Programme. Dr Aronson has a background in clinical epidemiology and health services research. Her research programme focused on measuring and improving symptoms and quality of life for people living with ILD. She has specific expertise in patient reported outcomes and psychometrics and is currently running a clinical trial to evaluate the feasibility and effectiveness of a behavioural and educational intervention to improve quality of life in ILD.

**Helen Ashdown** is an academic GP at the University of Oxford, and Co-Lead of the Respiratory Theme of the NIHR HealthTech Research Centre in Community Healthcare. Her research focuses on improving care of respiratory disease in the community, particularly related to earlier diagnosis, biomarkers, phenotyping and use of novel healthcare technologies, including those involving artificial intelligence. Helen leads or co-leads several research programmes generating evidence for new technology in respiratory primary care across diagnosis and monitoring in airways disease, including a diagnostic platform study to investigate multiple technologies in parallel. She is Research Lead of the Primary Care Respiratory Society and an Honorary GP at Beaumont Elms Practice in Oxford city centre.

**Professor Mona Bafadhel** is a National Institute for Health Research (NIHR) Research Professor and Professor of Respiratory Medicine at King's College London (KCL). Mona is the Director of the King's Centre for Lung Health, Professor of Asthma+Lung UK and Consultant Respiratory Physician at Guy's and St Thomas' NHS Foundation Trust. Mona completed her PhD in 'Biomarkers of exacerbations of chronic obstructive pulmonary disease (COPD)' and was the first to identify the utility of the peripheral blood eosinophil count in COPD and COPD exacerbations. This work has directly influenced international guidance and is now routinely used in the management of patients with COPD.

In 2019, Mona was awarded the prestigious Goulstonian Lectureship from the Royal College of Physicians, London. She is only the fourth woman and first woman from an ethnic minority in the Royal College of

Physicians 400-year-old history to have received this for Excellence in the Clinical Sciences. In 2024, Mona was the recipient of the European Respiratory Society COPD Gold Medal. Mona is the current Chair of the British Thoracic Society Science and Research Committee.

**Dr Haval Balata** is a Consultant Respiratory Physician based at Wythenshawe Hospital, Manchester University NHS Foundation Trust. He is also an Honorary Senior Lecturer at the School of Biological Sciences, University of Manchester.

Dr Balata's clinical and research interests include the risk reduction, screening and early detection of lung cancer and interventional bronchoscopy. Dr Balata enjoys extensive involvement in the pioneering 'Manchester Lung Health Check' service for targeted lung cancer screening, having overseen the initial pilot study as part of his PhD thesis in 2016. He is now the Responsible Clinician for the UK's largest lung screening programme. His other roles include being the Clinical Lead for the Interventional Bronchoscopy Unit at Wythenshawe Hospital which includes regional navigation and robotic (ION) bronchoscopy services.

**Joshua Barnes** completed the Scientist Training Programme in 2021, achieving an MSc in Clinical Science (Respiratory and Sleep Science). He is currently working as a Respiratory Clinical Scientist and Lead CPET Physiologist at Royal Papworth Hospital NHS Foundation Trust, in Cambridge. He also works as a Healthcare Science Practice Educator for the NHS East of England and is currently undertaking a PGCert in Medical and Clinical Education.

**Dr Helen Barr** is an Associate Professor at the University of Nottingham, UK and Honorary Consultant at Nottingham University Hospitals NHS Trust, UK. She is the Clinical Director of the Nottingham Wolfson Adult Cystic Fibrosis Centre. Dr Barr leads the chronic infection stream of the University of Nottingham NIHR Respiratory Biomedical Research Centre. Her main work focuses on chronic respiratory infection, particularly *Pseudomonas aeruginosa*, point of care diagnostics and epidemiology.

Dr Barr has extensive clinical trials expertise and is the UK principal investigator for the international trial FORMAT. For the past two years, she has been Clinical Lead for the UK CF Clinical Trials Accelerator Platform Network. She provides strategic oversight for the UK CF Clinical Trials Network and actively contributes to the Research Scientific Oversight Board at the CF Trust. She is a member of the NIHR Respiratory Translational Research Collaboration National Research Strategy Group and the European CF Clinical Trials Steering Committee.

**Dr Emily Bartlett** is a Consultant Thoracic Radiologist at the Royal Brompton Hospital (RBH), London. During a fellowship at RBH, she obtained a PhD at Imperial College London focusing on lung cancer screening implementation and lung nodule management. She has a particular interest in oncological imaging, thoracic oncological intervention and lung cancer screening (LCS). She is a co-author of the European Guidelines for the Management of Incidental Findings in LCS and has published widely in the field of thoracic oncological imaging and LCS. She is a member of the BTS Nodule Guidelines Group and is a Section Editor for *Lung Cancer Journal*.

**Dr Eleanor Barton** is a Respiratory Registrar and Pleural Clinical Research Fellow currently undertaking a PhD at the Academic Respiratory Unit at Southmead Hospital, Bristol. Her research interests include spontaneous pneumothorax, including identifying factors that can predict and improve patient outcomes. She was study co-ordinator for the Longitudinal Investigation of Secondary Pneumothorax (LISP) study, Associate PI for the PRINCE study and developed the ERS IMPACT drug induced effusion database.

**Paul Bastard**, MD PhD, is currently working in the Paediatric Haematology and Immunology Department of Necker Hospital for Sick Children (AP-HP, Paris, France), while also doing research in the Necker branch of the laboratory of Jean-Laurent Casanova, at the Imagine Institute (University of Paris Cité, INSERM) and the Rockefeller University (New York, USA). From 2020 onwards, he identified the role of auto-Abs to type I IFNs in the pathogenesis of more than 15% of cases of life-threatening COVID-19 pneumonia, and in several other severe viral diseases. His research currently focuses on the causes and consequences of autoantibodies against type I interferons.

**Dr Rahul Bhatnagar** is a Consultant Senior Lecturer at the University of Bristol, an Honorary Respiratory Physician at North Bristol NHS Trust, and an Adjunct Professor of Respiratory Medicine at the University of Southern Denmark.

# SPEAKERS' BIOGRAPHICAL DETAILS

His primary research interests lie in pleural disease, with an emphasis on malignant effusions and their management, novel diagnostics and interventions, and thoracic ultrasound.

**Anna Bibby** is an Associate Professor of Respiratory Medicine at the University of Bristol, and Honorary Consultant at North Bristol NHS Trust. She is Clinical Director for the Somerset, Wiltshire, Avon and Gloucestershire Lung Cancer Screening Programme and sits on the BTS Lung Cancer and Mesothelioma Specialist Advisory Group. Her research interests include lung cancer screening and mesothelioma. She holds an NIHR Advanced Fellowship for the IMPALA trial and is Chief Investigator of the multi-centre ASSESS-meso study. She is Deputy Chair of Mesothelioma UK's Research and Audit Committee and is an invited member of the Roy Castle Lung Cancer Foundation Clinical Expert Group.

**Professor Jane Bourke**, PhD, FThorSoc, ATSF, FAPSR, leads the Respiratory Pharmacology Group at the Biomedicine Discovery Institute, Monash University, Melbourne, Australia. Her innovative research spans three major themes: (1) novel bronchodilators and antifibrotics for asthma, pulmonary hypertension and IPF; (2) repurposing pirfenidone for COPD exacerbations and (3) early detection and drug screening in silicosis. She introduced the novel precision cut lung slice (PCLS) technique to Australia, pioneering its integration into translational studies across these research areas. Jane's research has resulted in high impact respiratory, pharmacology and multidisciplinary publications (***Thorax, ERJ, AJRCMB, BJP, Nature Comms***) and external recognition as an invited speaker (ERS, ATS, BTS) and Editorial Board member (***Respirology, AJRCMB, Fibrosis***).

**Professor Chris Brightling** is a Fellow of the Academy of Medical Sciences, National Institute for Health Research Senior Investigator, Respiratory and Infection Theme Lead for Leicester NIHR Biomedical Research Centre, Director Institute for Lung Health, Director for the Institute for Precision Health and Honorary Consultant Respiratory Physician, Leicester, UK. He has published >640 papers and his h-index=145. He leads the MRC PHOSP-COVID and EU-IMI 3TR consortia.

Professor Brightling's research focuses on improving the clinical management and understanding the immunopathogenesis of asthma, chronic cough, COPD and long-COVID. He is a member of the Global INitiative for Asthma (GINA) Scientific Committee.

**Dr Lindsay Broadbent** joined the Section of Virology at the University of Surrey as a Lecturer in July 2022. Previously, Lindsay was a Wellcome Trust ISSF Fellow at Queen's University Belfast. Her research focuses on respiratory virus-host interactions and subsequent innate immune responses. Lindsay's expertise in developing 3D human primary airway cell cultures facilitates investigation of virus infection in a physiologically and morphologically relevant model. Her current research is directed towards the role of respiratory viruses in longer term lung damage and the development and progression of chronic lung disease. In addition to her research, Lindsay delivers teaching and is the Programme Director for Microbiology at the University of Surrey. She is also involved with science communication and has taken part in hundreds of media appearances and outreach events.

**Hassan Burhan** leads the Liverpool Severe Asthma Service and serves as the Cheshire and Merseyside Respiratory Clinical Network Asthma Lead. He is a Ronald Finn Clinical Senior Research Fellow at the University of Liverpool and an Honorary Research Associate at the Liverpool School of Tropical Medicine. He has served as PI and CI on multiple early and late phase asthma and COPD studies and is the Deputy Director of the NIHR Cheshire and Merseyside CRDC.

Dr Burhan's research interests include real world evidence generation in asthma, novel airway disease diagnostics and improving access to care in non-communicable respiratory disease.

**Dr Clare Burton** is a Respiratory Consultant of 10 years, based at Sheffield Teaching Hospitals. She has a special interest in occupational lung disease (OLD), running a weekly OLD specialist clinic and MDT with her colleagues. During Dr Burton's training, she completed an MD thesis on 'Respiratory disease in workers exposed to metal working fluids' and she has several associated publications. Dr Burton is an active member of GORDS (Group of Occupational and Respiratory Disease Specialists). As a newly appointed member of the BTS Occupational and Environmental Disease Specialist Advisory Group, Dr Burton looks forward to working more closely with BTS, in a position where she can advise on the direction of future meetings and guidance.

**Professor Mat Callister** is a Consultant Respiratory Physician at Leeds Teaching Hospitals, and an Honorary Professor of Respiratory Medicine at the University of Leeds. His research interests are in the early detection

of lung cancer through low-dose CT screening and symptomatic detection, and smoking cessation. He has led/co-led various studies (Yorkshire Lung Screening Trial, YESS, MEDLEY, YorQuit), is a member of the NHS England Lung Cancer Screening Programme Expert Advisory Group, and is a sub-group chair for the IARC Lung Cancer Screening Handbook currently in development.

**Dr Toby Capstick** is Consultant Pharmacist – Respiratory Pharmacist – at the Leeds Teaching Hospitals NHS Trust. He is a member of the British Thoracic Society Multi-Drug Resistant Tuberculosis Clinical Advice Service, NTM Network UK, the UK Inhaler Group, and the UK Clinical Pharmacy Association Respiratory Group. He has clinics in the Leeds Interstitial Lung Disease and Tuberculosis services, and works with the Integrated Care Board in Leeds to improve respiratory medicines optimisation across the interface of care.

**Emily Cash** is a third year postgraduate research student in the School of Medicine at the University of Nottingham, funded by the Wellcome Trust Drug Discovery and Team Science Doctoral Training Programme (DTP). Emily currently serves as the PhD Representative for the British Association for Lung Research. Emily's PhD investigates the molecular mechanism of caffeine in bronchopulmonary dysplasia (BPD), with a broader interest in cell signalling and translational research.

**Professor James D Chalmers** is Asthma + Lung UK Chair of Respiratory Research at the University of Dundee, and a Consultant Respiratory Physician. He leads a translational research group focusing on developing new treatments for bronchiectasis, and also has interests in COPD, pneumonia and associated conditions. He has published >500 peer reviewed papers, including landmark trials that have changed practice in bronchiectasis. He has chaired five international guidelines panels including the recent ERS bronchiectasis guidelines. He is Chief Editor of the *European Respiratory Journal*. He was Chair of the British Thoracic Society Science and Research Committee from 2022-2024 and will serve as Chair of the BTS Standards of Care Committee from 2025 onwards.

**Dr Anita Chandra** is a Consultant Immunologist at Cambridge University Hospitals NHS Foundation Trust and an Affiliate Assistant Professor at the University of Cambridge. In her clinical work, she manages patients with both primary and secondary immunodeficiency and immune dysregulation and she is involved in medical education, clinical trials, and translational academic work.

She undertook her PhD on antibody diversification at the MRC Laboratory of Molecular Biology and then went on to undertake her postdoctoral studies, holding a Wellcome Trust Career Development Fellowship at the University of Cambridge, studying immune defects affecting the PI3Kinase delta pathway. She is regarded as an international expert in the field of Activated PI3K Delta Syndrome (APDS), a rare immunodeficiency syndrome, and is a principal investigator on the European Society of Immunodeficiencies: APDS Registry. She has published extensively in the field of APDS, PI3Kinase delta and B cells, including describing immunodeficiency as an aspect of PTEN deficiency. She has a special interest in respiratory immunology and manages patients with respiratory complications of immunodeficiency with the respiratory team, in the joint immunology and respiratory clinic at Addenbrooke's Hospital, Cambridge.

**Dr Nazia Chaudhuri** is a Respiratory Physician with a specialist interest in interstitial lung disease (ILD). She is Senior Clinical Lecturer at Ulster University and is Lead of the Northern Ireland ILD Network. She is Chair of the British Thoracic Society (BTS) ILD Registry Working Group and was Chair of the BTS Interstitial and Rare Lung Disease Specialist Advisory Group from November 2020 to 2023. She is also a member of the European Respiratory Society (ERS) ILD Long Range Planning Committee, an ERS ILD mentor and has just been appointed as the Secretary of the ERS Idiopathic Interstitial Pneumonia ILD Assembly. She is on the Irish Thoracic Society Council and ILD Working Group. She is a Trustee of the patient charity Pulmonary Fibrosis Northern Ireland (PFNI) and the NI Rare Disease Partnership.

**Dr Colin Church** is a Consultant in Pulmonary Vascular and Respiratory Medicine. He trained in Glasgow, Cambridge, Papworth and Sydney. He is one of three consultants in the Scottish Pulmonary Vascular Unit which is the national referral centre for the Scottish population. This unit investigates and manages all patients in Scotland with pulmonary hypertension. He is also one of the principal clinicians involved in management of venous thromboembolic disease in the Queen Elizabeth University Hospital. Dr Church is the Secretary of the Greater Glasgow and Clyde Thrombosis Committee and is current Chair of the

# SPEAKERS' BIOGRAPHICAL DETAILS

British Thoracic Society Specialist Advisory Group on Pulmonary Embolism and Other Pulmonary Vascular Diseases.

**Professor Robina Coker** is Professor of Practice in Respiratory Medicine at Hammersmith Hospital and Imperial College London. She developed the interstitial lung disease (ILD) service at Hammersmith from a monthly clinic with one ILD consultant in 1999 to the current service which runs 10 clinics a week involving four ILD consultants, an ILD clinical nurse specialist, and an ILD pharmacist. Robina was Clinical Director of the NIHR North West London (NWL) Comprehensive Local Research Network from 2010 to 2014, and Clinical Director of the NIHR NWL Clinical Research Network from 2014 to 2024. Robina was President of the Harveian Society of London in 2022.

Robina has worked closely with the British Thoracic Society (BTS) since 1998, when she was the junior doctor representative on the Education Committee. She has been a member of the Standards of Care Committee; Chair of the BTS Air Travel Working Party and later the Clinical Statement Group; and Honorary BTS Treasurer. She was also a founding member of the BTS Lung Disease Registry Programme.

Robina was awarded an individual Bronze Medal from the Royal Aeronautical Society in 2019 in recognition of her work on air travel safety, and in 2021 she received the BTS Meritorious Service Award. She currently represents the Society on the NHSE Specialised Respiratory Commissioning Clinical Reference Group (CRG). From November 2025, Robina will be President of the British Thoracic Society.

**Mia Cokljat** is a Respiratory and Intensive Care Trainee within the Thames Valley Deanery. Her research interests lie within epidemiology; she hopes to use large data to better understand the temporal and national trends of respiratory illnesses. She has previously completed works at the Infectious Diseases Data Observatory at the University of Oxford and the World Health Organisation, and is completing an MSc in Epidemiology at the London School of Hygiene and Tropical Medicine under the supervision of Professor Jennifer Quint. In collaboration with Dr Catharine Pearce and INSPIRE, Dr Cokljat is planning a multi-centre study to describe the association between pharmacological agents and fungal lung disease.

**David Connell** is Co-Chair of the Scottish Health Protection Tuberculosis Network, and is a Consultant Physician in Respiratory and General Internal Medicine at Ninewells Hospital, NHS Tayside. His main clinical interests incorporate complex lung infections, particularly tuberculosis, chronic pulmonary aspergillosis, and non-tuberculous mycobacteria. He has interests in the delivery of care in both chronic and acute respiratory infections, and was part of the groups which produced the BTS Clinical Statements on Ocular Tuberculosis, and *Aspergillus*-related Chronic Lung Disease. He is a Clinical Service Advisor to the BTS MDR-TB Clinical Advice Service, and was a co-opted member of the UK New and Emerging Respiratory Virus Threats Advisory Group (NERVTAG) during the first 12 months of the COVID-19 pandemic. He is active in clinical research in NTM-PD, fungal lung disease, and bronchiectasis, and is local PI in a number of studies in these areas.

**Gráinne d'Ancona** is the Consultant Pharmacist for Respiratory Medicine at Guy's and St Thomas' NHS Foundation Trust and a Senior Lecturer at King's College London. She has contributed to national guidelines and training programmes, was a Clinical Champion for the Asthma Biologics Rapid Uptake Programme of the NHSE Accelerated Access Collaborative and holds several national committee seats, most notably on the NHSE and NHSI Inhaler Sustainability Delivery Board, the RCP National Respiratory Audit Programme, and is Chair of the BTS Pharmacist Specialist Advisory Group.

An advocate for integrated respiratory care and value-based interventions, her clinical roles include optimising care for patients with severe asthma, COPD, ILD and sleep disorders, in the hospital setting and also through virtual clinics in general practice. Gráinne's particular area of academic interest is medicines optimisation.

**Jane Davies** OBE, FMedSci, FRCPCH, is Professor of Paediatric Respirology and Experimental Medicine at the National Heart and Lung Institute, Imperial College London and an Honorary Consultant at the Royal Brompton Hospital. Her areas of research expertise are cystic fibrosis chronic lung infection and clinical trial design, including novel physiology and imaging outcome measures.

Jane has been global and national lead investigator on over 30 trials of CFTR modulator drugs, including in children and infants. She co-leads the multi-centre observational studies, RECOVER and ENHANCE, and the MATRIARCH Strategic Research Centre, investigating maternal and infant health in the CFTR

modulator era. She is Clinical Lead / Strategy Group member of the UK CF Gene Therapy Consortium, currently conducting a phase 1/2 study of lentiviral mediated gene therapy in adults. She is President of the European CF Society, directs the European Lung Clearance Core Facility and chairs the UK NIHR National Research Strategy Group in CF.

**Professor Tony De Soyza** is an Academic Clinician / Physician-Researcher and an Honorary Consultant Physician at Newcastle upon Tyne Hospitals NHS Foundation Trust. His interests are in translational gaps particularly in bronchiectasis and COPD. Tony works with a fabulous MDT team of physios, nurses, microbiologists, radiologists and immunologists in Newcastle looking after nearly 1000 adults with bronchiectasis. He has set up the multi-centre collaboration and UK National Registry in Bronchiectasis "BRONCH-UK" 2014-2017 with further funding from the US COPD Foundation. Tony also holds several NIHR HTA grants. He is a keen supporter of early career researchers and has greatly enjoyed working with the INSPIRE trainee network.

**Dr Charlotte Dean** is a Reader in the National Heart and Lung Institute, Imperial College London. Charlotte established her group working on the parallels between lung development and disease. A particular focus of Charlotte's work has been on understanding the mechanisms that drive lung development and how these are perturbed to cause congenital and adult lung diseases. She has identified key regulators of lung generation and investigated whether some of these factors could be harnessed to regenerate the lungs following injury/disease. Current projects include an investigation of Wnt5a as a potential pharmacological pro-repair treatment and a study of lung anatomy in mouse models of Down Syndrome. Charlotte is an associate editor for ***Thorax*** and a member of the NC3Rs Partnership and Impact Panel.

**Professor Keertan Dheda** is currently Head of the Division of Pulmonology at the University of Cape Town, and Professor of Mycobacteriology and Global Health at the London School of Hygiene and Tropical Medicine.

He serves, or has served, on several international academic and advisory bodies including those of the WHO, NIH, Wellcome Trust, and the editorial boards of the ***American Journal of Respiratory and Critical Care Medicine, Lancet Respiratory Medicine***, and the ***British Medical Journal***. He has published more than 400 peer-reviewed manuscripts (H index of 104) and is a co-inventor on eight patents related to diagnostics and vaccine development. He has been the recipient of several global research awards.

**Dr Jenny Dickens** is an Action for Pulmonary Fibrosis Mike Bray Research Fellow and Group Leader in the Cambridge Institute for Medical Research, and an Honorary Consultant at Addenbrooke's and Royal Papworth Hospitals. She has a research and clinical interest in interstitial lung disease with particular focus on familial pulmonary fibrosis. She works with novel alveolar organoid models to understand the triggers of epithelial dysfunction in ILD, including how surfactant protein variants are handled in health and disease. She has developed the familial pulmonary fibrosis service at Royal Papworth Hospital, is academic training rep for the East of England Deanery and is an Addenbrooke's Charitable Trust Research Advisory Committee Member.

**Giles Dixon** is a Clinical Research Fellow and Specialist Trainee in Respiratory Medicine with the Wellcome Trust GW4-CAT PhD Programme. His PhD is registered with the University of Exeter and is focussed on the prognostication and disease monitoring of fibrotic-ILD using quantitative CT.

**Dr Gillian Doe** PhD, is a Respiratory Specialist Physiotherapist and Research Programme Manager at the University of Leicester. Her research focus is in improving the diagnostic process, management and outcomes for individuals living with breathlessness. Dr Doe is a Lead Clinical Assessor for the Pulmonary Rehabilitation Service Accreditation Scheme, with the Royal College of Physicians, responsible for assessing PR services against evidence-based quality standards. She also holds a role as Associate Editor for the Nature Partner Journal, ***Primary Care Respiratory Medicine*** and is part of the International Primary Care Respiratory Group Working Group for Breathlessness.

**Dr Matthew Drake** MD, is the Division Head and Edwards Professor of Pulmonary Medicine within the Division of Pulmonary, Allergy, and Critical Care at Oregon Health and Science University, Oregon, USA. His research interests include asthma and chronic cough, with a specific focus on airway neurodevelopment and the role of neuroimmune interactions in disease pathogenesis.

**Dr Virve (Vicky) Enne** is a Senior Research Fellow at the Centre for Infectious Disease and Diagnostics

# SPEAKERS' BIOGRAPHICAL DETAILS

Research (CIDR), based at Guy's and St Thomas' Trust and King's College London. Her research focuses on the development and evaluation of novel molecular diagnostic strategies for bacterial infections and AMR, and their implementation to improve antimicrobial stewardship and patient outcomes, with a strong emphasis on respiratory tract infections and pneumonia in the critical care environment. She is the Clinical Evaluation and Research Lead for the national NHS Respiratory Metagenomics Network and the co-ordinator and joint Chief Investigator of the INHALE research programme She is also an editorial board member for the *Journal of Antimicrobial Chemotherapy*.

**Corinne Faivre-Finn** is a Professor of Thoracic Radiation Oncology at the University of Manchester and an NIHR Senior Investigator. She leads radiotherapy research for the Manchester Cancer Research Centre and CRUK Lung Cancer Centre. She is the Chair of the ESTRO Lung Focus Group and the Radiotherapy Chair of the EORTC Lung Group. An active member of IASLC and ESTRO committees, she is Principal Investigator for multiple lung cancer radiotherapy trials and has contributed to major clinical guidelines (ESMO, BTS, EORTC, ERS, ASTRO). She has authored over 300 papers, including seven practice-changing studies.

**Dr Katie Ferguson** is a Respiratory Registrar in her penultimate year of training in the East of Scotland, based at Ninewells Hospital in Dundee. Prior to registrar training, she worked in the Pleural Disease Unit at the Queen Elizabeth University Hospital in Glasgow. She has a special interest in pleural disease and completed her PhD on the evolution of mesothelioma from benign asbestos pleural inflammation. She continues to pursue research interests in her current role as a registrar, having completed the associate PI scheme and has been sub-investigator on several interventional pleural trials.

**Andrew Fisher** is Professor of Respiratory Transplant Medicine at Newcastle University and a Consultant Physician at the Institute of Transplantation, Freeman Hospital, Newcastle upon Tyne. He is Co-Director of the NIHR Blood and Transplant Research Unit in Organ Donation and Transplantation. His clinical and research interests are in advanced severe lung disease and lung transplantation and he leads a research group focused on understanding mechanisms of lung injury and repair, including after lung transplantation. He has published extensively in the field, including in 2024 an invited review of lung transplantation in the *New England Journal of Medicine*. He is a Past President of the International Society for Heart and Lung Transplantation.

**Andres Floto** is Professor of Respiratory Biology at the University of Cambridge, and Co-Director of the Cambridge Centre for AI in Medicine (CCAIM).

His basic research is focused on understanding: how bacteria evolve from environmental organisms into specialist lung pathogens; how they interact with, and are shaped by, the innate immune system; and how functional genomics and machine learning can be combined with fragment-based drug discovery to develop novel antibiotics.

His clinical research is centred around treating non-tuberculous mycobacteria (NTM), tackling chronic inflammation in CF and non-CF bronchiectasis, using AI to understand and predict pulmonary exacerbations.

Clinically he specialises in the management of non-tuberculous mycobacteria, cystic fibrosis, non-CF bronchiectasis, and recurrent chest infections.

**Clementine Fraser** trained on the NIHR Academic Clinical Fellow Pathway at the National Heart and Lung Institute, Imperial College London. She now works as a Consultant Respiratory Physician at the Queen Elizabeth Hospital in Birmingham where she has sub-speciality interests in lung transplantation and complex respiratory infection, specifically fungal and mycobacterial disease. She Chairs the regional complex infection MDT and combines her clinical commitments with research interests in the fields of transplant and chronic infection.

**Neil French** is Professor of Infectious Diseases and Global Health at the University of Liverpool. He is an infectious disease physician and clinical epidemiologist. He currently manages a multidisciplinary team of investigators spread across Malawi and Liverpool. He is known internationally for work on pneumococcal disease and other vaccine preventable disease problems. Other interests include clinical trials and their design for infectious disease topics and analytical approaches to large datasets. Professor French is CI of PRONTO, a national study investigating the role of Procalcitonin in emergency departments for sepsis management. He directs the Liverpool Clinical PhD Programme for Health Priorities in the Global South.

**Freddy Frost** is a clinical academic with a research and clinical interest in chronic infectious lung diseases. Freddy is currently Senior Lecturer and Consultant Respiratory Physician carrying out clinical work at Liverpool Heart and Chest Hospital, and research investigating the long-term health outcomes of people living with chronic lung infection with a focus on bronchiectasis and cystic fibrosis.

**Dr Jonathan Fuld** is Consultant Respiratory Physician based at Cambridge University Hospitals NHS Foundation Trust and the Victor Phillip Dahdaleh Heart and Lung Research Institute, University of Cambridge. Since February 2024 he has been National Clinical Director for Respiratory Disease and Chair of the Adult Specialised Respiratory Clinical Reference Group, NHS England.

Jonathan has a clinical and research interest in exercise physiology, chronic obstructive pulmonary disease, and access to services for those with chronic lung disease.

**Dr Liz Fuller** is a Consultant Respiratory Physician at Newcastle Hospitals NHS Foundation Trust and an Honorary Senior Lecturer at the University of Newcastle. In addition she is the Clinical Lead for Lung Cancer Screening for the Northern Cancer Alliance. Her research interests are lung cancer screening, early diagnosis and health inequalities of lung cancer. She is a member of the British Thoracic Society Lung Cancer and Mesothelioma Specialist Advisory Group and Lung Cancer Clinical Expert Group.

**Jonathan Grigg** is Professor of Paediatric Respiratory and Environment Medicine, Queen Mary University of London. He is also a part-time researcher at Helmholtz Zentrum, Munich.

Jonathan's research focuses on the adverse effects of air pollution. He has published papers that describe how air pollution increases susceptibility to viral and bacterial infections. He advised policy makers as a member of the UK Committee on the Medical Effects of Air Pollution. As Chair of the European Respiratory Society's Tobacco Control Committee he advocated limiting young people's access to vaping products. Clinically, he wrote NHS guidance on air pollution and asthma, and has directed a National Institute for Health and Care Research Global Health Research Group aimed at improving children's asthma care in Sub-Saharan Africa. In 2023, he launched the first NHS environmental assessment service for asthmatic children at the Royal London Hospital.

**Lizzie Grillo** is an Advanced Physiotherapist at Guy's and St Thomas' Hospital (Royal Brompton Hospital) and is completing a PhD as an NIHR Research Fellow with Imperial College London looking at the physiotherapy assessment of breathlessness. She has over 15 years of experience working in chronic disease and has a special interest in airway clearance, breathlessness, and breathing pattern assessment.

Lizzie has published several articles in this area and supported guideline development with the British Thoracic Society. She is a member of the British Thoracic Society Workforce Committee and a member of the Association of Chartered Physiotherapists in Respiratory Care (ACPRC). Additionally, she supports the Physiotherapy for Breathing Pattern Special Interest Group in delivering their study days.

**Chris Grime** read medicine at the University of Liverpool before starting his foundation training at St Helen's and Knowsley NHS Foundation Trust. He was then appointed to a paediatric training post with the Mersey Deanery. After completing five years of training and his Royal College of Paediatrics and Child Health membership, he successfully applied for specialist paediatric respiratory training in London and spent the next five years working at Royal Brompton Hospital, Evelina London Children's Hospital, King's College Hospital and finished his training at Great Ormond Street Hospital. During this time, he spent two years completing an MDRes with Imperial College London studying sleep disordered breathing and its association with asthma in children. After a further 10 months at GOSH working as a post-CCT fellow in long-term ventilation, he was appointed to a consultant post at Alder Hey Children's NHS Foundation Trust, Liverpool.

Chris currently works as part of the respiratory team at Alder Hey and has a special interest in severe asthma. He is also part of the Sleep and Long-Term Ventilation Service and has completed his training as a sleep practitioner. He was named WellChild Inspirational Doctor of the Year in 2021. In addition to his clinical role, Chris is also the Trust Chief Clinical Information Officer.

**Dr Tillie-Louise Hackett,** PhD, ATSF, is a Professor at the University of British Columbia and Tier I Canada Research Chair in Asthma and COPD Lung Pathobiology and Therapeutics. A global leader in ultra-resolution imaging and human ***in vitro*** lung models, her work has revealed early small airway loss in COPD, influencing global health strategies. She directs the James Hogg Lung Biobank and has received over 37 research and teaching awards. Her talk will explore how spatial

proteomics and imaging is revealing the heterogeneity of inflammation and fibrosis patterns in COPD to enhance therapeutic discovery.

**Ghady Haidar**, MD, is an Associate Professor of Medicine in the Division of Infectious Diseases at the University of Pittsburgh and a Transplant ID Physician at UPMC. He is the Medical Director of the Translational Research Unit at UPMC. Dr Haidar is a translational clinician investigator funded by the NIH, industry, and foundations, and has focused on optimising the management of difficult-to-treat infections in immunocompromised individuals. As Chair of Phage Clinical Trials and Therapeutic Applications at the Pittsburgh Phage Program (P3), he collaborates with basic scientists to oversee the "bench to bedside" applications of phage therapy at the University of Pittsburgh and other centres. Dr Haidar also collaborates with his P3 colleagues on translational bacteriophage research and has led both investigator-initiated and sponsor-initiated bacteriophage trials.

**Dr Jemma Haines** MBE, is Trust Chief Allied Health Professional at Manchester University NHS Foundation Trust. In her early career she worked as a voice clinician and quickly understood the significance of the larynx during respiration. Jemma is now an established consultant with over 20 years' experience working with upper airway respiratory disorders. Pioneering the role of speech therapy in UK respiratory healthcare, she developed and led two tertiary referral multidisciplinary complex breathlessness services and has co-authored numerous national professional respiratory guidelines. Jemma is extensively published with many peer reviewed publications relating to her clinical research work. Supported by an NIHR Manchester Biomedical Research Centre Research Fellowship she has recently completed her doctoral studies on the evaluation and treatment of inducible laryngeal obstruction.

**Dr Pranabashis Haldar** is a Consultant Physician and Associate Professor in Respiratory Medicine at Leicester, UK. He is also Training Programme Director for Respiratory Medicine in the East Midlands and Lead for National Recruitment. Over the past decade, Dr Haldar has developed a clinical and academic interest in the control and management of tuberculosis and through his role as TPD, been a strong advocate for the provision of mycobacterial training to doctors in training. He is a member of the BTS MDR-TB Steering Group and Clinical Advisory Service and a member of the BTS TB and NTM Specialist Advisory Group. In Leicester, he leads the Rapid Access Tuberculosis (TB) Clinical Service for the city, which provides a centralised pathway to accelerate investigation and diagnosis in patients with suspected TB. He is Academic Lead for Clinical TB Research within the Respiratory and Infection Theme of the Leicester NIHR Biomedical Research Centre (BRC) and provides clinical leadership to the Leicester TB Research Group (LTBRG).

Dr Haldar's primary academic interest is in the development and evaluation of novel candidate biomarkers with potential to be translated into new clinical tools for improving the care and control of TB. This work has included the development of novel blood transcriptional signatures for TB diagnosis and monitoring treatment response, ongoing development of a phage-based TB diagnostic and phenotypic stratification of latent TB infection using PET-CT. He has active collaborations with industrial and academic partners, including the Francis-Crick Institute, University of Birmingham and University of Nottingham.

**Dr Matthew Hartwig** is a Professor of Surgery and Biomedical Engineering with a clinical focus in lung transplantation and robotic assisted minimally invasive thoracic surgery for the treatment of diseases of the chest. He has served as the Surgical Director of the Duke Lung Transplant Programme and the Oesophageal Centre at Duke, North Carolina, USA. Additionally, he directs the Surgical Office of Clinical Research, which manages the clinical research portfolio for the Department of Surgery. He also leads a successful programme of clinical, basic and translational research in thoracic surgery and lung transplantation with over 200 peer reviewed publications. He currently directs the Duke Ex Vivo Organ Laboratory (DEVOL), is the Chief of Lung Transplant Research, and is a faculty member at the Duke Clinical Research Institute (DCRI).

**Professor Adam Hill** is Post Graduate Dean, NHS Education for Scotland and Professor of Respiratory Medicine, University of Edinburgh. He chairs the British Thoracic Society Standards of Care Committee and is the NHS Research Scotland Respiratory Lead, Respiratory Advisor to CMO, and working with Scottish Government long term conditions team. He is a trustee of the Morriston Davies Trust.

**Professor Barry Hill** is Professor in Nursing and Critical Care Practice and Associate Head of School at Northumbria University. As an active educator, researcher, academic, and clinical leader, he holds a BSc

in Nursing, a BSc in Critical Care, an MSc in Advanced Practice, and a PhD. Barry has a clinical background in intensive care nursing at Imperial College Healthcare NHS Trust, with experience across general, cardiac, and neurotrauma centres. He is author and editor of eleven books covering acute and critical care, prescribing, and pharmacology, including the *Advancing Practice* series. He has an extensive publication record, including peer-reviewed articles, book chapters, and conference presentations, and contributes to NHS England initiatives, steering groups, and editorial boards.

**Dr Lizzie Hill** is a Clinical Scientist with over 20 years' experience of working with adults and children with sleep disorders in clinical and research settings. She is currently a Senior Lecturer in Sleep Physiology at the University of the West of England (UWE Bristol), where she leads the PGCert Sleep Medicine programme, commissioned and funded by NHS England to address the skills gaps in clinical sleep services (https://courses.uwe.ac.uk/C9P100/sleep-medicine). Lizzie gained her PhD by research at the University of Edinburgh in 2016, exploring prevalence and treatment of obstructive sleep apnoea in adults with Down Syndrome. She is a Society Rep for the Physiological Society, past President of the European Society of Sleep Technologists and past Education Lead of the British Sleep Society.

**Dr Nikhil Hirani** is a Consultant Respiratory Physician leading the Interstitial Lung Disease (ILD) Service in Edinburgh and a Reader/PI in the Institute for Regeneration and Repair at the University of Edinburgh. He has a long-established interest in early phase lung fibrosis clinical trials, exploratory biomarkers, and macrophage biology in lung inflammation and repair. Nik has led multiple experimental medicine trials, has chaired impactful clinical international guideline committees and generated the ELFMEN study, one of the largest longitudinal cohorts of ILD globally. He is an Associate Medical Director in NHS Lothian. He co-leads the LifeArc Rare Respiratory Disease Biobank and Registry.

Nik grew up in Leicester and as a life-long supporter of Leicester City Football Club is well rehearsed in difficult long-term projects that are interspersed with very occasional sporadic successes.

**Dr Patrick Howlett** is a Respiratory Registrar in Northwest London. He also works in the occupational lung disease clinic at the Royal Brompton Hospital, London. Between 2021 and 2025, he was an MRC Doctoral Research Fellow at Imperial College London. His primary research and clinical interests are occupational lung diseases and respiratory infection, in particular the intersection of silicosis and tuberculosis.

**Dr Ryan Hoy** MBBS MOccEnvHlth FRACP PhD, is a Respiratory Physician from Melbourne, Australia with a Master's degree in occupational health. He is the Lead Physician for the Occupational Respiratory Clinic at the Alfred Hospital and is a Senior Research Fellow at the Monash University Centre for Occupational and Environmental Health. His primary research is in the field of artificial stone associated silicosis, and he has 28 publications on the topic. In 2019, Dr Hoy was appointed to the National Dust Disease Taskforce, and in 2022 chaired the National Silicosis Prevention Strategy, facilitated by the Lung Foundation of Australia.

**Professor Tracy Hussell's** research examines the mechanisms driving immunopathogenesis in respiratory infections and how the lung senses injury and challenge. She has unravelled many reasons for the long-term consequences on lung function of acute infection and injury. She developed and is leader of the Lydia Becker Institute of Immunology and Inflammation (www.manchester.ac.uk/lydia-becker-institute).

As current President of the British Society for Immunology, Tracy has dedicated her time to try and change the post-doctoral career providing better employment security and appropriate remuneration. She has trained 29 PhD students to date; several now hold senior positions at other establishments. She currently sponsors seven independent Wellcome Trust, MRC, Asthma + Lung UK, ESPID and BBSRC early career fellows. She has been a member of the Wellcome Trust's Scientific Interview Panel and Expert Review Group, she was a member of UoA5 for the Research Excellence Framework for 2022, and is a fellow of the Academy of Medical Sciences and the Royal Society of Biology, amongst many others.

**Dr Catherine Hyams** is a Clinical Lecturer at the University of Bristol and the Bristol Vaccine Centre. She leads the AvonCAP study within the Pfizer Centre of Excellence for Epidemiology of Vaccine-Preventable Diseases. Her research focuses on respiratory infections in adults, including disease severity, outcomes, and the impact of vaccination.

**Dr Robert E Hynds** is a Senior Research Fellow at University College London's Great Ormond Street Institute of Child Health, where he leads the Epithelial Cell Biology in ENT Research Group (EpiCENTR). His research focuses on developing advanced airway epithelial cell culture methods for regenerative medicine

and disease modelling. Dr Hynds has pioneered techniques for long-term expansion of primary human airway epithelial cells, enabling applications in cell therapy and respiratory disease research.

**Dr Wadah Ibrahim** is a Consultant in Respiratory Medicine at Glenfield Hospital, Leicester, with a clinical and academic interest in COPD. He is part of the complex COPD service, providing comprehensive assessments for patients with advanced disease and contributing to the community MDT and Glenfield lung volume reduction service. He holds a PhD in breathomics, focusing on its role in identifying different patient profiles in acute breathlessness.

**Dr Sriram Iyer** is a Respiratory and Sleep Consultant at Sheffield Teaching Hospitals where he leads the sleep service. He has a dedicated weekly respiratory and neurology sleep clinic. His research interests include cardiovascular effects of OSA, COPD-OSA overlap and the impact of poor sleep on surgical performance. He is the PI for several sleep-related studies including EPiC-HFT, ReACT and EPiC-OSA.

Dr Iyer is overseeing a British Sleep Society studentship on the effects of poor sleep on surgical outcomes. He is heavily involved in education – he runs the immensely popular Difficult Lung Disease meeting and is also the President of the Royal Society of Medicine Sleep Council. He sits on the BTS Sleep Specialist Advisory Group and BTS Council.

**Sam Janes** is the Director of Medicine at University College London and he works as a Respiratory Consultant at UCLH. Major contributions include defining that normal airway homeostasis is governed by stochastic division of basal cell; showing that airways genetically damaged by smoking can regenerate on quitting; mapping the molecular architecture of pre-cancerous squamous cell lesions, and identifying the immunological abnormalities that allow pre-cancerous lesions to progress to cancer. He was elected to the Academy of Medical Sciences in 2021.

Professor Janes is chief investigator of several trials ranging first-in-man, to SUMMIT, the largest lung cancer screening trial in Europe recruiting over 13000 people.

**Laura Jess** is a Clinical Scientist and is currently the Head of Paediatric Respiratory and Sleep Physiology for NHS Lothian. She has a keen interest in exercise physiology, personalised management of breathlessness and paediatric scoliosis surgery research. She currently sits on the Association for Respiratory Technology and Physiology (ARTP) Executive Board as Events Committee Chair and ARTP Scotland Committee Chair. In addition, Laura holds voluntary roles which include representing respiratory and sleep physiology on the Scottish Strategic Network for Diagnostics (SSND) Clinical Physiology Core Group and working with representatives across the Scottish Government to develop the strategic approach for Healthcare Science in Scotland.

**Akhilesh Jha** is a Medical Research Council (MRC) Clinician Scientist in Respiratory Medicine at the University of Cambridge and Addenbrooke's Hospital. His research approach is to use translational human challenge models combined with cutting-edge single-cell molecular biology techniques to investigate host innate immune responses and mechanisms of airway inflammation. His goal is to develop new approaches of treating acute flare-ups of chronic airway diseases.

Dr Jha is passionate about engaging the next generation of clinical researchers and is founding Chair of the Integrated Respiratory Research Collaborative (INSPIRE), supported by National Institute of Health Research (NIHR), Respiratory Futures and Asthma + Lung UK.

**Alison John** is an Advanced Research Fellow at the National Heart and Lung Institute, Imperial College London where she is a member of the Margaret Turner Warwick Centre for Fibrosing Lung Disease. Her research focuses on understanding the cellular and molecular mechanisms regulating development and progression of idiopathic pulmonary fibrosis including investigating the functional consequences of common and rare genetic variants on lung cell function to identify novel therapeutic targets and disease biomarkers. In support of these studies her lab is developing and utilising advanced 2D and 3D models systems using patient derived cells and multi-omic spatial analysis of healthy and diseased tissues to better understand IPF pathogenesis and patient heterogeneity.

**Simon Johnson** is Professor of Respiratory Medicine at the University of Nottingham, Director of the Nottingham NIHR Respiratory Biomedical Research Centre and runs the UK National clinical services for lymphangioleiomyomatosis (LAM) and rare cystic lung diseases. He is a co-director for the Rare Respiratory Diseases Centre. Simon's research group work on mechanisms and interventions for tissue damage in chronic lung diseases including LAM, and methods to

improve diagnostics and management of rare lung diseases. Simon is a member of the LAM Foundation Scientific Advisory Board and a professional advisor to the UK Tuberous Sclerosis Association.

**Stavros Konstantinides** is Professor for Academic Clinical Trials and the Medical Director of the multidisciplinary Centre for Thrombosis and Haemostasis (CTH) at the University of Mainz, Germany. The CTH is dedicated to integrated patient care and patient-oriented research. Dr Konstantinides is also appointed Professor of Cardiology at the Democritus University of Thrace, Greece.
His research focuses on the risk-adapted management of pulmonary embolism, with emphasis on pharmaceutical and catheter-directed reperfusion treatment. He has designed and co-ordinates several high-impact multi-national trials. He is a leading member of national, European and transatlantic committees and boards, guidelines task forces, and pulmonary embolism networks.

**Professor Sailesh Kotecha** is Head and Professor of Child Health at Cardiff University, UK. His interests lie in delineating and treating the processes that lead to the development of chronic lung disease of prematurity (CLD). His group has conducted the AZTEC randomised control trial to assess if azithromycin decreases rates of CLD in 796 preterm-born infants and are now recruiting 542 preterm-born infants to the BALLOON trial assessing if enhancing trained immunity can decrease rates of lower respiratory tract infections in discharged preterm-born infants. His group are also linking these studies to longer term outcomes using national cohorts including ALSPAC, MCS and Biobank as well as their own cohorts of RANOPs and RHiNO developing the concept of prematurity-associated lung disease or PLD in child-/adulthood following preterm birth identifying different phenotypes.

**Professor Wei Shen Lim** is Consultant Respiratory Physician at Nottingham University Hospitals NHS Trust and Honorary Professor of Medicine, University of Nottingham. He is the incoming Chair of the Joint Committee of Vaccination and Immunisation (JCVI), from 1 October 2025.

His research activities are in the field of acute respiratory infections, including pneumonia, pneumococcal infections and influenza. He developed the CURB65 severity scoring tool for community acquired pneumonia and co-led the evaluation of Dexamethasone for COVID-19 in the RECOVERY Trial. He is Chair of the Acute Respiratory Infections National Research Strategy Group, NIHR Respiratory Translational Research Collaboration and is co-lead of the UKHSA Hospital-based Acute Respiratory Infection Surveillance System (HARISS) in England.

**Dr Kate Lippiett,** BA (Hons), MSc, RGN, PhD, is a nurse academic with a joint role. First, as Senior Research and Knowledge Mobilisation Fellow at the University of Southampton/NIHR Applied Research Collaborative Wessex. Second, as a Clinical Research Fellow in Primary Care for NHS England South West. Kate's research interests are in multiple long term conditions and treatment burden. She is Chair of the Association of Respiratory Nurses Research and Education Committee.

**Dr Merete B Long** is a Research Fellow in Respiratory Science at the University of Dundee, focusing on bronchiectasis and other lung diseases, including COVID-19. Her work characterises immune responses and inflammation, with an emphasis on neutrophil biology, to identify novel therapeutic targets and evaluate immunomodulatory treatments. She is involved in translational research, encompassing multiomic analyses and clinical trials investigating DPP1 inhibition, protease inhibition, and anti-*Pseudomonas* monoclonal antibody therapy.
Merete serves as Publications Secretary for the British Association for Lung Research and Associate Editor for *ERJ Open Research*. She is a recipient of the BTS/BALR Young Investigator of the Year Award (2022) and ATS Rising Star Award (2025).

**Matt Loxham** is Professor of Respiratory Biology and Toxicology and BBSRC David Phillips Fellow in the Faculty of Medicine, University of Southampton. He leads a multidisciplinary research group whose focus is the differential toxicological effects of particulate matter air pollution from different sources. He is especially interested in particulate-associated metals, cellular homeostatic responses to these metals, and implications for lung disease, with a focus on fibrotic lung disease. Aside from a focus on *in vitro* toxicology, his projects and collaborations also incorporate particulate matter source apportionment, novel methods for monitoring the urban spread of particulates, and epidemiology-based studies.

**Dr Abigail MacKintosh** is a Respiratory Registrar training in the West Midlands Deanery. She graduated from the University of Birmingham in 2013 and has been a registrar since 2018. In July 2023, she took up the role of Chair of the British Thoracic Society

# SPEAKERS' BIOGRAPHICAL DETAILS

Speciality Trainee Specialist Advisory Group. She has a particular interest in education and training and during her time in this role would like to get trainees across the country connected and sharing areas of good practice to improve everyone's training.

**Dr Paul Marsden** is a Consultant Respiratory Physician at Manchester University NHS Foundation Trust, based at the North West Lung Centre, Wythenshawe Hospital, and Honorary Senior Lecturer at the University of Manchester. After undertaking a PhD in cough, asthma and airway inflammation, he completed training in the North West of England. He is Clinical Lead of the Manchester Cough Service and Lancashire/South Cumbria Cough Service based in Preston which sees patients from across the UK and beyond. He is an active researcher in chronic cough and contributed to the recently published British Thoracic Society Clinical Statement.

**Professor Refiloe Masekela** is Dean and Head of the School of Clinical Medicine at the University of KwaZulu-Natal, Durban, South Africa and an NIHR Global Health Research Professor. A leading paediatric lung health researcher with over 17 years of experience, her work focuses on asthma and lung function testing. She holds key leadership roles globally, including Co-Chair of the Global Asthma Network and Scientific Committee member of GINA. She is the President of the Pan African Thoracic Society. With over 100 publications and multiple textbook contributions, Professor Masekela is internationally recognised for advancing paediatric respiratory medicine through research, leadership, and mentorship.

**Nick Maskell** is Professor of Respiratory Medicine, University of Bristol and Honorary Consultant, North Bristol NHS Trust, Bristol. He undertook his DM thesis on pleural diseases in Oxford prior to taking up a consultant post at North Bristol NHS Trust in 2003. He was awarded a national Walport Senior Lecturer Award in 2005 and joined the University of Bristol.

His research interests include clinical trials in pleural infection, pneumothorax, pleural malignancy, and patient safety during pleural procedures. He leads the Academic Respiratory Unit (ARU) at the University of Bristol and is the Deputy Director of the University of Bristol NIHR-BRC. He is an NIHR Senior Investigator and currently the Chief Investigator for a number of NIHR multi-centre randomised controlled trials. His current H-index is 72 with over 300 peer reviewed publications and over 17000 citations. He is part of the faculty of the newly developed ERS Thoracic Ultrasound Certified Training Programme. He co-chaired the 2018 BTS mesothelioma guidelines and the 2019 ERS malignant pleural effusion taskforce statement. He also co-chaired the 2023 BTS pleural disease guidelines and 2023 ERS pneumothorax guidelines. Nick is the Chairman of the Board of Trustees for the charity Mesothelioma UK and President of the British Thoracic Society.

**Dr Jane McDowell** is a Clinical Lecturer at Queen's University Belfast and Honorary Respiratory Consultant at Belfast Health and Social Care Trust. After training at Bart's and the London Medical School, she continued her post-graduate training in London and Northern Ireland. Her PhD focused on mechanisms of inflammation underlying the residual exacerbations in biologics-treated severe asthma patients, and quantification of oral steroid-associated toxicity with reducing steroid exposure facilitated by biologics. Since then, interests include understanding non-T2 mechanisms that contribute to the burden of disease in severe asthma, both comorbid disease and non-T2 airways inflammation.

**Professor Pleasantine Mill** PhD FRSE, is an MRC Investigator at the University Edinburgh where she leads a programme to understand genetic disease and disease mechanisms arising from dysfunction of mammalian cilia – the ciliopathies. Her lab focuses on phenotype-driven projects which disrupt cilia structure and/or function to uncover underlying genetic changes, understand disease mechanisms and move towards much-needed therapeutics for rare diseases. She is co-lead of the MRC NMGN Congenital Anomalies Cluster and co-Director of the new LifeArc Centre for Rare Respiratory Diseases. For her contributions, she was awarded the 2019 BSCB Women in Cell Biology Medal and 2025 British Society for Developmental Biology Wolpert Medal.

**Dr Paul Minnis** is a Specialist in Interstitial Lung Disease and the Director of Research and Development at the Northern HSC Trust. He undertook a fellowship in ILD at St Vincent's University Hospital, Dublin. Here his research strands included the role of microRNA in IPF, observational studies in sarcoidosis and live data studies at the Insight Centre for Data Analytics UCD investigating the potential for connected health solutions in the diagnosis and management of chronic disease.

**Phil Molyneaux** is a Professor of Interstitial Lung Disease at Imperial College London and holds the Asthma + Lung UK Chair of Respiratory Research. He is a Consultant in Interstitial Lung Disease and Director of the NIHR Cardiorespiratory Clinical Research Facility at the Royal Brompton Hospital. He leads an active clinical and translational research programme, overseeing a multidisciplinary team of basic scientists and clinical trial staff focused on advancing the understanding and treatment of interstitial lung diseases.

**Dr Andrew Molyneux** is a Consultant Respiratory Physician, Sherwood Forest Hospitals. He trained and did his PhD in health services research in Nottingham and the East Midlands, with an MSc in epidemiology from the London School of Hygiene and Tropical Medicine. He has been a consultant for 22 years with interests in sleep/NIV and COPD/asthma. Andy's interest in QI and patient safety, particularly in the delivery of acute NIV, stems from his involvement in NCEPOD Inspiring Change. He has worked with NICE, chairing the COPD and Melanoma Guideline Committees, and Co-Chairing the BTS/NICE/SIGN Asthma Guideline Committee. He is the current Chair of the British Thoracic Society Quality Improvement Committee, and will be leading the Audit and Quality Improvement Symposium this year.

**Dr Vicky Moore** is a Consultant Clinical Scientist and Deputy Lead for Respiratory in the Respiratory and Sleep Sciences Department at University Hospital Coventry and Warwickshire (UHCW). Her specialist interests include asthma and occupational asthma, causal agent identification, specific and non-specific challenge testing, lung function testing and CPET. She is a keen researcher in these areas. She runs clinics in occupational asthma at UHCW and with the team at the Birmingham Chest Clinic. She completed her PhD in this area in 2010. She is the current ARTP/BTS rep and before this was the ARTP Education Chair. She is also a member of the ERS spirometry group. She is passionate about supporting training and education in respiratory physiology and occupational asthma.

**Helen Morris** is one of the Interstitial Lung Disease Specialist Nurses at Wythenshawe Hospital and has been with the ILD Specialist Nursing Service since 2013. The ILD nursing team provide supportive care throughout the patients' disease, managing palliative care needs and advising on end-of-life care. She has a respiratory and long-term conditions background in both primary and secondary care. Helen has an MSc in respiratory practise/non-medical prescriber. She is former Chair of the ILD-IN Committee, former ILD Lead for ARNS, Emeritus Board member ILD-IN, and nurse representative on the British Thoracic Society Lung Registry Steering Group.

**Christine Mwasuku** is a full-time PhD student in immunology at King's College London, supported by Asthma + Lung UK and supervised by Professor Mona Bafadhel. With a background in biological sciences and specialist training in respiratory nursing, she has an honorary clinical contract with Guy's and St Thomas' Trust. Chris has a strong interest in asthma and COPD. Her research focuses on the role of oestrogen in women with asthma and COPD, and she currently leads an observational study in this area as part of her PhD. She is passionate about advancing equity in respiratory health and bridging the gap between clinical care and translational science.

**Professor Neal Navani** MA (Cantab) MSc PhD FRCP, qualified in medicine from Cambridge University and UCL in 2000 with distinction and several university prizes. He trained in respiratory medicine at the Brompton and Hammersmith Hospitals before winning a Medical Research Council Fellowship in 2008 and completing his PhD at UCL in 2011. He has also completed an MSc in clinical trials and biostatistics at the London School of Hygiene and Tropical Medicine.
Professor Navani is Lead Clinician for the Lung Cancer Service at University College London Hospital, and Senior Clinical Lead of the National Lung Cancer Audit. He is also Clinical Director for the Centre for Cancer Outcomes for the North Central London Cancer Alliance. He is on the steering committee of the British Thoracic Oncology Group, UK Lung Cancer Coalition and on the Thoracic Oncology Board of the American Thoracic Society.
Professor Navani currently holds >£2.5m of grant funding as chief investigator including a CRUK grant for the early diagnosis of lung cancer, a separate CRUK grant for developing novel methods for cancer data collection and NIHR funding for studies on predicting lung cancer prognosis with artificial intelligence and early detection of lung cancer in never smokers. He is an author of current NICE guidelines on lung cancer and bronchoscopy and co-chair of the BTS guideline on nodule management as well as a US guideline on peripheral bronchoscopy. He has over 150 peer-reviewed publications. In 2020, Professor Navani won a prestigious MRC/NIHR Fellowship to research predictors of cancer in lung nodules and was appointed

# SPEAKERS' BIOGRAPHICAL DETAILS

to Professor of Respiratory Medicine at University College London in 2024. @LungConsultant

**Dr Helen Newman** PhD, is a Speech and Language Therapist with over 20 years' clinical experience in the UK and New Zealand. She has worked extensively with patients with a tracheostomy in critical care, head and neck cancer and brain injury rehabilitation. She is a member of the International Group of SLTs Working in Ultrasound, whose pioneering work explores clinical applications of ultrasound in voice, speech, swallowing and tracheostomy. She is Treasurer of the Royal College of Speech and Language Therapists Tracheostomy Clinical Expert Network and co-author of the SLT Pillar of the Intensive Care Society AHP Professional Development Framework. Her NIHR Clinical Doctoral Research Fellowship focused on how to choose the best size tracheostomy tube for adults in ICU.

**Dr Phillip Nicolson** is a Consultant Haematologist at the Queen Elizabeth Hospital in Birmingham and an Associate Clinical Professor of Cardiovascular Medicine at the University of Birmingham. Clinically he specialises in thrombotic and bleeding disorders as well as immune-haematology. He founded the trainee-led UK Haematology Research Network HaemSTAR in 2017 and ran it until 2023. He also runs a small lab investigating various aspects of platelet biology (https://www.birmingham.ac.uk/staff/profiles/cardiovascular-sciences/nicolson-pip).

**Professor Clive Page,** OBE, BSc, PhD, FRSB, FBPhs (Hon), FFPM (Hon), is an Emeritus Professor of Pharmacology, King's College London. Clive's main research interests are in the pharmacology of inflammation and respiratory disease, and he has published over 300 scientific papers and book chapters.

Clive was the co-founder and previous Chairman of the Board of Verona Pharma plc, a NASDAQ listed company and was instrumental in the discovery and development of Ensifentrine for the treatment of COPD and other respiratory diseases which was recently approved by the FDA.

He is a Trustee and Chairman of the Board of Trustees of the Fraunhofer Institute of Toxicology and Experimental Medicine in Hannover. Clive is also a Non-Executive Director of Ananda Developments. Clive started his early career in the pharmaceutical industry at Sandoz Ltd, Basel, Switzerland and regularly consults to both pharmaceutical and biotech companies. Clive has previously held non- executive board positions at Cardiome in Canada, Stirling Products Ltd in Perth, Australia, Immune Regulation Ltd, EpiEndo, PreP Biopharma, Babraham Biotechnology Ltd and as a Trustee of the Babraham Institute, Cambridge.

Clive is a former Chairman of the Animal Science Group of the Royal Society of Biology and has contributed widely to the public debate about the use of animals in research. Clive is a recent past President of the British Pharmacological Society.

Clive was awarded the Sandoz Prize from the British Pharmacological Society in 1988, the Tanabe Young Investigators Prize from the American College of Clinical Pharmacology in 1996 and the Royal Society of Biology President's Medal in 2012 for "Outstanding Contributions to the Life Sciences". He is a Fellow of the Royal Society of Biology and an Honorary Fellow of the British Pharmacological Society and the Faculty of Pharmaceutical Medicine, Royal College of Physicians. Clive was awarded an OBE for "Services to Pharmacology" in 2017.

**Dr Sean Parker** is a Consultant Respiratory Physician at Northumbria Healthcare NHSFT. In addition to being a general respiratory physician, he has run a specialist cough clinic since 2010 and has a particular interest in non-pharmacological cough control therapy, psychological aspects of cough and novel antitussives. Dr Parker is currently co-chairing the BTS quality standards for cough group and NEurocough CRC and previously co-chaired the BTS Clinical Statement on Chronic Cough in Adults.

**Caroline Patterson** studied medicine at the University of Nottingham. During a 16-year career as a Royal Air Force Medical Officer she completed higher training in respiratory and general internal medicine on the NorthWest Thames Rotation. She was awarded an MD from Imperial College, London, for research examining novel chest imaging techniques. On completion of clinical training, she spent six months at Duke University, North Carolina, USA, as the International Society for Heart and Lung Transplantation (ISHLT) Travelling Fellow in Lung Transplantation. She maintains an active role in ISHLT through which she has contributed to multiple international consensus statements and is the incoming Programme Chair for the 2026 ISHLT Annual Meeting in Toronto, Canada. Caroline was appointed as a Consultant Respiratory and Lung Transplant Physician at Royal Papworth Hospital in Cambridge, UK in 2016.

**Ian D Pavord**, MA DM FRCP FERS FMedSci, is Professor of Respiratory Medicine at the University of

Oxford and Honorary Consultant Physician at the Oxford University Hospitals. He is a Member of Congregation at the University of Oxford and a Professorial Fellow of St Edmund Hall. He was a Consultant Physician from 1995 and Honorary Professor of Medicine from 2005 to 2013 at the Institute for Lung Health, Glenfield Hospital, University Hospitals of Leicester NHS Trust. He was elected an NIHR Senior Investigator in 2011 and 2014, an inaugural Fellow of the European Respiratory Society in 2014 and a Fellow of the Academy of Medical Science in 2015.

He has a research interest in the clinical aspects of inflammatory airway diseases and has pioneered the use of non-invasive measures of airway inflammation in the assessment of these conditions. He has identified a number of clinically important phenotypes of inflammatory airway disease, has discovered clinically important biomarkers, and has played a lead role in the clinical development of three of the most promising new treatments for severe airway disease.

Professor Pavord was co-editor of *Thorax* from 2010-2015, Chief Medical Officer of Asthma UK from 2008-2014, Associate Editor of the *American Journal of Respiratory and Critical Care Medicine* from 2005-2010 and Associate Editor of the *European Respiratory Journal* from 2016-2023. He is the author of over 650 publications and has an H-index of 142 making him the highest cited researcher in asthma worldwide. He received the 2016 European Respiratory Society (ERS) Gold Medal for his research and gave the Cournand Lecture at the 2004 ERS meeting. He chaired the 2017 Lancet Commission on Asthma.

**Catharine Pearce** is a final-year Respiratory Registrar in the Southwest of England with interests in interventional respiratory medicine and palliative care. She recently completed a fellowship in lung cancer and interventional procedures in Plymouth, alongside a diploma in palliative care from Newcastle University. Catharine is actively involved in medical education, contributing chapters to publications by Cambridge University Press and Oxford Clinical Guidelines. Her work aims to integrate collaborative approaches to medical practice with a strong focus on patient-centred care.

**Daniel Peckham** is Professor of Respiratory Medicine and Deputy Director of the Leeds Institute of Medical Research at the University of Leeds. He is the Clinical Lead for the Leeds Adult Cystic Fibrosis, Primary Ciliary Dyskinesia and Bronchiectasis Services. He pioneered the design and implementation of chronic disease electronic patient records and established a strong base for clinical and basic research in cystic fibrosis. Active research programmes focus on CF-related inflammation, CFTR modulators, gut dysbiosis, cancer, drug allergy, big data, electronic patient records and clinical trials. He is passionate about education and was the founding Director of Education for the European Cystic Fibrosis Society.

**Dr Felicity Perrin** is a Consultant Respiratory Physician at King's College Hospital, London, and is joint lead for the TB/NTM services at King's and at Guy's & St Thomas' Hospitals. She is also a clinician in the Adult Cystic Fibrosis Unit at King's College Hospital and therefore has experience of managing NTM within cystic fibrosis, as well as in other pulmonary contexts. She trained largely in London, worked for nine months in Malawi and did a research degree in TB. She is the current Chair of the British Thoracic Society TB and NTM Specialist Advisory Group.

**Hilary Pinnock** is Professor of Primary Care Respiratory Medicine at the University of Edinburgh. She leads programmes of work in the Centre for Applied Respiratory Research, Innovation and Impact (CARRii), the RESPIRE Global Health Research Unit, and the European Respiratory Society Clinical Research Collaboration 'CONNECT'.

Her research focusses on delivery of care, specifically implementation of supported asthma self-management in routine primary care, and digital health interventions for non-communicable diseases. With colleagues from Bangladesh, India and Malaysia, she is evaluating pulmonary rehabilitation tailored to low resource settings.

Hilary chairs the Education Council of the European Respiratory Society and is involved with the International Primary Care Respiratory Group.

**Dr Jessica Potter** is an NHS Consultant in Respiratory Medicine and Tuberculosis at North Middlesex University Hospital. She is an Honorary Senior Clinical Lecturer at UCL and a member of the UCL-TB Delivery Group. Jess chairs the grass roots network UK Academics and Professionals to End TB and is a member of the innovations constituency of the Stop TB Partnership.

**Professor Nicola Ranger** joined the Royal College of Nursing in December 2022 as the Chief Nursing Officer. In 2024 she was appointed General Secretary and Chief Executive of the RCN. She was previously

# SPEAKERS' BIOGRAPHICAL DETAILS

Chief Nurse and Executive Director of Midwifery at King's College Hospital NHS Foundation Trust in London. Before that, she held Chief Nurse posts at both Brighton and Sussex University Hospitals NHS Trust and Frimley Health NHS Foundation Trust. Professor Ranger has also held a number of senior nursing roles at University College London Hospitals NHS Foundation Trust and Surrey and Sussex Healthcare NHS Trust. Earlier in her career, she worked at America's George Washington University Hospital in Washington and at Mount Sinai Medical Centre in New York.

**Anna Reed** is a practising lung transplant physician and has worked at Harefield Hospital since 2010. She is Professor of Practice (Respiratory and Transplant Medicine) at Imperial College in London and is the first woman to hold a professorship title in this field in the UK. She has over two decades of clinical experience in lung transplant medicine, both in the UK and abroad. She is committed to improving access to lung transplant and outcomes from transplant for all patients living with a diagnosis of a chronic respiratory disease. Her research interests are focussed on the host immunological responses to injury and infection in lung transplant recipients, with a particular spotlight on unravelling mechanisms leading to accelerated graft loss and rejection.

Within the clinical arena, Anna has led on multiple clinical improvement projects. Most recently, she built on her national expertise in the development of extra-corporeal life support strategies as a bridge to lung transplantation and spearheaded an international collaboration aimed at identification of optimal factors for success in this critically unwell cohort. She strives to improve outcomes after transplant with a focus on early identification of infectious and other inflammatory insults to the lung. She incorporates state of the art scientific techniques to understand mechanisms of graft injury at key time points after transplant with a view to early translation with modification of risk factors in clinical practice.

Anna holds international leadership positions within the International Society for Heart and Lung Transplantation with a particular focus on pulmonary vascular diseases and the development of novel diagnostics and immunosuppression agents for the prevention and treatment of chronic lung allograft dysfunction.

**Ms Katie Rhatigan** is Senior Research Practitioner, King's College Hospital, London, and PhD Student, King's College London. She co-ordinates international clinical trials in chronic cough, sarcoidosis and ILD, and is the site coordinator for the first pan European cough registry by NEuroCOUGH. Katie is completing her PhD with a focus on assessments and pathophysiology of cough. She is currently working on the validation of chronic cough outcome measures, including both patient-reported outcomes and objective cough monitoring. Katie is a member of the British Thoracic Society Cough Specialist Advisory Group.

**Dr Chris Rhodes** is an Associate Professor at the National Heart and Lung Institute at Imperial College London and was awarded his BHF Senior Basic Science Fellowship in 2022. His group focuses on defining clinical phenotypes and novel drug targets through the use and functional evaluation of omics data in pulmonary hypertension patients. This has included the analysis of genomic, epigenetic, transcriptomic, proteomic and metabolomic data in patients recruited from the Hammersmith Hospital National PH Service as well as through other UK and European Centres in the BHF/MRC-funded UK National PAH Cohort study, and international collaborators including cohorts from the US, France and Germany as well as industry partners.

**Dr Pilar Rivera-Ortega** is a Respiratory Consultant with a sub-specialty in interstitial lung disease (ILD). She joined the Exeter ILD service in November 2024, following six and a half years as the ILD Clinical and Research Lead at Wythenshawe Hospital in Manchester.

Currently, Dr Rivera-Ortega is the ILD Research Lead and Associate Director of Research and Development at Royal Devon University Healthcare NHS Foundation Trust; serving as Principal Investigator on multiple national and international clinical trials. She is a member of the British Thoracic Society Interstitial and Rare Lung Diseases Specialist Advisory Group, Secretary of the ERS Rare ILD Group, and Chair of the REG ILD Working Group.

**Dr Katy M Roach** is a Respiratory Lecturer at the Institute for Lung Health, University of Leicester, specialising in idiopathic pulmonary fibrosis (IPF). Her research highlights the pivotal role of ion channels and myofibroblasts in fibrotic lung disease. Dr Roach has developed ex vivo human lung models to investigate fibrotic signalling and evaluate novel drug compounds. Her findings show that selective $K_{(Ca)}3.1$ blockers can reverse pro-fibrotic pathways, supporting their potential as therapeutic agents for IPF.

**Professor Nicola Roberts** is a Professor in Respiratory Healthcare Delivery within the School of

Nursing and Social Care at Edinburgh Napier University. Her research focus is on improving the way respiratory healthcare is delivered. Recent work has focussed on how respiratory care is taught to pre-registration nursing students, and how education is delivered in pulmonary rehabilitation. She is education, communication and network lead for the ARRA network (Academic Respiratory Researcher Alliance) which promotes respiratory research for nursing and allied health professionals. She is Chair of the Scottish Pulmonary Rehabilitation Action Group and she is an Associate Editor for *npjPCRJ*.

**Alex Rothman** is Professor of Cardiology at the University of Sheffield and an Honorary Consultant Cardiologist at Sheffield Teaching Hospitals NHS Foundation Trust. He leads a programme of basic, translational, and clinical research, funded by the MRC, EPSRC, NIHR, BHF, and Wellcome Trust, focused on developing novel therapies and treatment strategies for patients with pulmonary hypertension, heart failure and cardiovascular disease. His work has identified therapeutically tractable aspects of disease pathophysiology and contributed to the development of regulatory-approved products now included in clinical guidelines and eligible for reimbursement.

**Professor Richard EK Russell** BSc (Honours), PhD, MBBS, FRCP (UK), trained in London and is currently a Professor of Respiratory Medicine at KCL London and a Respiratory Consultant at Southern Health NHS Foundation Trust. He is the Clinical Director of the West Hampshire Integrated Respiratory Service. In November 2024 he became the Chair of the British Thoracic Society.

His research focusses on the basic mechanisms of COPD, disease progression, COPD exacerbations, mechanisms of lung damage as well as phenotypes of all airways disease.

Professor Russell is the founding Editor of the *International Journal of COPD,* and is keen to increase the global awareness of COPD and provide a platform for scientific developments from across the world.

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H index 34

**Beth Sage** is a Consultant Respiratory Physician at NHS Highlands and Honorary Senior Lecturer at the University of Aberdeen. She is also Director of Research Development and Innovation for NHS Highland. She is Clinical Lead for Lung Cancer in NHS Highland and is PI on a number of lung cancer and mesothelioma clinical trials. Her current research projects are looking at improving diagnosis in mesothelioma using cell free DNA and she has a CSO/NRS Career Researcher Fellowship looking at late lung cancer presentations in rural populations. Previously she undertook an MRC Clinical Research Training Fellowship in Genetically Modified Stem Cell Therapies in Malignant Pleural Mesothelioma at UCL.

**Manuel Sánchez-de-la-Torre**, PhD, ATSF, FERS, is Scientific Director at IDISCAM and Associate Professor at the University of Castilla-La Mancha, Spain. A Ramón y Cajal researcher, he specialises in precision medicine in sleep apnoea, with significant contributions to cardiovascular research. He completed post-doctoral training at the University of Chicago, where he developed a pioneering technology for personalised sleep apnoea management. He has authored over 170 publications, led 17 competitive projects, and mentored 10 PhD students. He chairs ERS Group 4.01 and serves on SEPAR's Scientific Committee. In 2024, he was named Fellow of both the ATS and ERS.

**Dr Brintha Selvarajah** is a Principal Research Fellow at University College London and the Francis Crick Institute and an Honorary Respiratory Consultant at University College London Hospital. She has a research and clinical interest in interstitial lung disease with a particular focus on idiopathic pulmonary fibrosis (IPF). Her research is focused on dissecting the mechanisms by which alterations in cellular metabolism can promote fibrosis. She also leads the ASPIRE study at UCL, which uses an integrated multi-omics approach to characterise interstitial lung abnormalities (ILAs), aiming to identify predictive biomarkers of disease progression to pulmonary fibrosis and enable early diagnosis and intervention.

**Dr Joanna Shakespeare** is a Consultant Clinical Scientist in Sleep and Ventilation at University Hospitals Coventry and Warwickshire NHS Trust, having worked as a Respiratory Physiologist/Scientist for 30 years. Joanna is the Honorary Chair of the Association for Respiratory Technology and Physiology (ARTP). She regularly teaches on lung function and cardiopulmonary exercise testing (CPET) and is a faculty member for the ARTP CPET course, the UCLA/Cambridge CPET course and the ERS advanced clinical exercise testing course. Her specialist interests include cardiopulmonary exercise testing and non-invasive ventilation in both the acute and domiciliary settings.

# SPEAKERS' BIOGRAPHICAL DETAILS

**Dr Emily Shaw** is a Consultant in Infectious Diseases and General Medicine and holds joint NHS/UKHSA appointments with the Hospital for Tropical Diseases, University College London Hospitals and UKHSA TB Unit. Her studies were at Cambridge University, UCL and LSHTM and her specialist training was in London. She has delivered MDR-TB care in a programmatic setting for Médecins Sans Frontières in Uzbekistan. Her Wellcome UCL PhD was in BCG vaccinology. She is a tutor on TB at LSHTM DTM&H. Dr Shaw has worked with Find and Treat, piloting the delivery of TB care in HM Prisons and Immigration Removal Centres, and she leads on TB in secure sites for the UKHSA TB Unit.

**Professor Amelia Shoemark** is Asthma + Lung UK Chair of Respiratory Research at the University of Dundee, where she conducts a translational research programme in chronic respiratory disease with a focus on bronchiectasis and mucociliary clearance.

Amelia began her career as a clinical scientist at the Royal Brompton Hospital in London. She set up and led a nationally funded laboratory for the diagnosis of primary ciliary dyskinesia (PCD), a position she still holds part time.

Amelia completed her PhD at Imperial College in 2010 investigating inflammation in the chronic respiratory condition bronchiectasis. She is an active member of the management committee of the European Bronchiectasis Clinical Research Consortium, EMBARC, and is Chair of the BEAT-PCD European Respiratory Society Clinical Research Consortium. She chaired the recently published ERS/ATS diagnostic guideline for PCD.

These translational areas of respiratory research have led to >100 peer review articles and book chapters on bronchiectasis, primary ciliary dyskinesia and mucociliary clearance.

**Samira Shojaee**, MD, MPH, is an Associate Professor of Medicine in the Department of Medicine and Thoracic Surgery and the Director of the Pleural Disease Programme at Vanderbilt University Medical Centre. Her clinical areas of expertise include interventional pulmonology with a broad range of diagnostic, therapeutic and palliative airway and pleural procedures, lung cancer and pleural diseases.

Dr Shojaee's research focuses on pleural carcinogenesis, biomarkers of malignant and non-malignant pleural effusion, and lung cancer. She has led numerous clinical and translational investigator-initiated research projects and is the founder of the V-PEAK pleural fluid biobank at Vanderbilt. Her lab has discovered various molecular biomarkers of disease within the pleural space. Dr Shojaee completed a Master in Public Health from the Harvard School of Public Health with focus on biostatistics and epidemiology and contributes to the research mission of various respiratory societies.

**Dr Gerard A Silvestri**, MD, MS, Master FCCP, is Senior Vice-Chair of Faculty Development and the Hillenbrand Distinguished University Professor of Thoracic Oncology at the Medical University of South Carolina, Charleston. He completed his training in pulmonary and critical care medicine and received an additional degree in health services research, both at Dartmouth. His research includes screening for lung cancer, lung nodule evaluation, diagnosis and staging of lung cancer, development of biomarkers for disease risk and treatment, and disparities in delivering lung cancer care to underserved communities.

He has authored more than 300 scientific articles, book chapters and editorials. He currently serves on the editorial board of the journal ***Chest***. Dr Silvestri is past President of the American College of Chest Physicians (CHEST). Dr Silvestri received the 2020 Lifetime Achievement Award in Lung Cancer from the American Thoracic Society and was named a Master Fellow of the American College of Chest Physicians, a distinction awarded to only 40 members in the nearly 90 years of that organisation's existence.

**Dr Claire Smith** is Associate Professor of Respiratory Infection and Epithelial Biology at UCL's Great Ormond Street Institute of Child Health. Her research focuses on how respiratory viruses – including RSV, influenza, and SARS-CoV-2 – interact with airway epithelial cells, and how ageing airways and innate immune responses, particularly neutrophils, influence viral clearance, inflammation, and tissue damage. She leads development of disease relevant ***in vitro*** antiviral testing platforms and is a core investigator in the FLUTRAILMAP One Health consortium, which explores the risk of zoonotic spillover of highly pathogenic avian influenza to humans. Her work is supported by UKRI, GOSHCC, AFRUK, and industry.

**Dr Dan Smith** is a Respiratory Consultant at Sherwood Forest Hospitals in the East Midlands, with special interests in COPD, ventilation, and chronic disease management. Nationally, he works with NHS England to support organisations in delivering impactful quality improvement programmes. As a Clinical Associate for ECIST and Chair of the British Thoracic

Society's Workforce and Service Development Committee, he helps shape national standards and service innovation. Dan also leads the Royal College of Physicians' Improvement Hub as Editor-in-Chief of *Medical Care – Driving Change*, promoting evidence-based practice, collaboration, and a culture of continuous improvement across the NHS.

**Dr Laura-Jane Smith** is a Respiratory and Internal Medicine Consultant at Kings College Hospital with specialist interests in airways disease and lung cancer. She has a Diploma in Clinical Education and has written two books on respiratory medicine for doctors in training. She also has a Diploma in Lifestyle Medicine and is a campaign volunteer with Plant Based Health Professionals UK, advocating for plant-rich foods for personal and planetary health. LJ is Clinical Lead for Sustainability at King's and the BTS Trustee for Sustainability. She is the co-chair of the South East London Responsible Respiratory Prescribing Group and a member of King's Health Partners Clinical and Academic Group Executive with a focus on population health.

**Dr Sanja Stanojevic** is an Associate Professor in the Department of Community Health and Epidemiology at Dalhousie University, Nova Scotia, Canada. Dr Stanojevic is a respiratory epidemiologist with expertise in methodologies to characterise the normal growth and development of the lungs. Her research applies novel statistical approaches to improve the interpretation of respiratory function tests. She Chairs the ERS Global Lung Function Initiative Network and is Chair of the ATS Pulmonary Function Committee.

**Karl Staples** is Professor of Respiratory Immunology and Pharmacology and Associate Dean for Knowledge Exchange and Enterprise at the University of Southampton Faculty of Medicine. His research focuses on host-pathogen interactions in chronic inflammatory airways diseases, such as asthma and chronic obstructive pulmonary disease (COPD), and the contribution of these interactions to exacerbations of these respiratory conditions. To study these interactions he has developed novel *ex vivo* models of bacterial and viral infection using human lung cells and tissue. He is the Chair of the British Association for Lung Research.

**Dr Karl Sylvester** is Consultant Healthcare Scientist at Cambridge University Hospitals and Royal Papworth Hospital NHS Foundation Trusts. Part of this role involves clinical oversight of the breathlessness pathway at the Cambridgeshire and Peterborough Community Diagnostic Centres. He is a Fellow of the Academy of Healthcare Science. He has served as Honorary Chair of the Association for Respiratory Technology and Physiology (ARTP) and Chair of Group 9.1 at the European Respiratory Society, a group which represents respiratory and sleep scientists from across Europe. His specialist interest is the performance and interpretation of cardio-pulmonary exercise testing. He is faculty lead for the ARTP's annual CPET course, the BTS/ARTP physiology course and faculty director for the UCLA/Cambridge CPET course. Dr Sylvester initially joined Cambridge University Hospitals after completing a PhD investigating respiratory complications in patients with sickle cell disease.

**Philippa Thomas** has the rare lung disease LAM. She sits on the Patient Advisory Board of the LifeArc Rare Respiratory Diseases Centre. Her role is to work with other patients to reflect lived realities, help co-ordinate with patient groups, and bring her voice to decision-making about our Centre's priorities. Philippa is a professional communicator who spent three decades at BBC News as a political correspondent, foreign correspondent, and TV and radio presenter.

**Harm Tiddens** is a Paediatric Respiratory Physician and Emeritus Professor at Erasmus MC-Sophia Children's Hospital, with appointments in the departments of Paediatric Pulmonology and Radiology. Although trained in paediatrics, his clinical and research expertise spans lung diseases across the life course, including CF, COPD, bronchiectasis, asthma, and PCD. He founded the Erasmus MC LungAnalysis core lab, supporting imaging-based analysis for investigator-initiated and industry-sponsored trials. Since 2022, he has been Chief Medical Officer at Thirona, an AI company specialising in quantitative lung image analysis. His research focuses on structure-function relationships in respiratory disease, with a particular emphasis on quantitative image analysis of chest CTs. He actively collaborates worldwide on the development and validation of sensitive imaging biomarkers for use in clinical trials and regulatory evaluation.

**Dr George Tsaknis** is a Consultant in Respiratory Medicine and Clinical Lead for Lung Cancer at Kettering General Hospital and Clinical Director of the Lung Cancer Screening Programme in Northamptonshire. He is Co-Chair of the Lung ECAG and Lung Cancer ODN in East Midlands, a member of the British Thoracic Society Lung Cancer and Mesothelioma Specialist Advisory

# SPEAKERS' BIOGRAPHICAL DETAILS

Group, and Honorary Senior Lecturer in Respiratory Sciences, University of Leicester.

**Professor Michael Tunney** is a Chair in Clinical Pharmacy at the School of Pharmacy, Queen's University Belfast. His translational research programme focuses primarily on the improved detection and treatment of lung infection in patients with respiratory diseases such as cystic fibrosis, bronchiectasis and COPD. More recently, he has worked on the development of novel commercial point-of-care diagnostic tests for respiratory and urinary tract infections and on developing and testing new inhaled antibiotics to treat chronic lung infection.

Professor Tunney has published over 200 peer reviewed papers and serves on several scientific advisory and editorial boards.

**Professor Alice Turner** graduated from the University of Leicester and did postgraduate training via the universities of Dundee and Birmingham, the latter being where she completed a PhD focussed on COPD and alpha 1 antitrypsin deficiency (AATD). She has been a Consultant in Respiratory Medicine at Heartlands Hospital since 2011, and Professor of Respiratory Medicine at the University of Birmingham since 2020. She plays an active role with specialist societies, having been a member of the British Thoracic Society Specialist Advisory Group on COPD previously, and currently a member of their Quality Improvement Committee. She has published widely in COPD and AATD, and has ongoing research projects funded by the NIHR, Alpha 1 Foundation and others.

**Don Urquhart,** MD, FRCPCH, is Consultant in Paediatric Respiratory and Sleep Medicine, Royal Hospital for Children and Young People, Edinburgh, Honorary Reader, Department of Child Life and Health, University of Edinburgh and Centre Director, South East Scotland Paediatric Cystic Fibrosis Service. He is a clinician and researcher whose main interests are exercise and cystic fibrosis, and sleep medicine. Don undertook paediatric respiratory training in London, and has an MD from UCL. He undertook a post-CCT fellowship in paediatric sleep medicine in Brisbane. Don has been an author on more than 80 peer reviewed publications, and has contributed to the writing of international exercise testing and sleep medicine guidelines. Don has been PI on a variety of CF clinical trials, and has led several investigator-initiated studies in the fields of exercise in CF, most recently the NIHR-funded ExACT CF trial.

**Paul EY Van Schil** graduated from medical school with great honour in 1982 at the Antwerp University in Belgium. After completing his training in general surgery, he became a fellow in the Department of Cardiothoracic Surgery at the Antonius Hospital, Nieuwegein, The Netherlands. He was Chair of the Department of Thoracic and Vascular Surgery from 2000 until 2019. Currently, he is Professor Emeritus in Thoracic and Vascular Surgery at the Faculty of Medicine and Consultant Thoracic Surgeon at the Department of Thoracic and Vascular Surgery of the Antwerp University Hospital.

From October 2013 until October 2014 he was President of the European Association for Cardio-thoracic Surgery (EACTS). In 2017 he became a member of the Board of Directors of the International Association for the Study of Lung Cancer (IASLC) and Chair of the lung cancer domain of its Staging and Prognostic Factors Committee (SPFC). In September 2021 he was appointed President-elect of IASLC, and in September 2023, President of this worldwide organisation.

**Professor Martie van Tongeren** is an Occupational and Environmental Health Epidemiologist with nearly 35 years of experience in researching causes of occupational diseases. He leads an interdisciplinary team at the Centre for Occupational and Environmental Health at the University of Manchester. He has been Principal Investigator for The Health and Occupation Research Network (THOR), which collects data on occupational diseases, including occupational lung diseases, from across the UK since 1989. Data are used to determine trends in occupational disease and identify potential sentinel cases.

**Pamela Vaughn** is a Principal Physiotherapist in Respiratory Medicine in the North of Glasgow, with over three decades of experience in the field. She qualified in Glasgow, where she began her career and quickly developed a passion for respiratory care. When the West of Scotland Adult Cystic Fibrosis Unit was established, she was the sole physiotherapist along with being a founding member of the Association of Chartered Physiotherapists in Cystic Fibrosis.
Her career includes time spent working in the United States, after which she returned to Glasgow to further her clinical and academic focus on respiratory medicine. Pamela has held prescribing qualifications since 2013 and has advanced practice qualifications. She runs autonomous outpatient clinics with a specialist focus on bronchiectasis, airway clearance, chronic cough and

breathing pattern disorders.
Pamela has played an active role in national guideline development through her involvement in several British Thoracic Society Guideline Groups. She currently serves on the BTS Bronchiectasis Specialist Advisory Group and is an expert member of the West of Scotland Research Ethics Committee. Her academic contributions include journal publications and a forthcoming student handbook.

**Dr J Christian Virchow,** MD, FRCP, FCCP, FAAAAI, FERS, is a Professor of Medicine and Pneumology and Head of the Clinic for Pulmonary Medicine, Allergology and Intensive Care Medicine in the Department of Internal Medicine at the University Clinic at the University of Rostock, Germany. He received his doctoral degree from the University of Tübingen, Germany after attending Medical School in Marburg and Giessen, Germany, and Zürich, Switzerland. Dr Virchow completed his residency in internal medicine at the University Medical Clinic in Freiburg, Germany, and his fellowship in pulmonary medicine at the Department of Pneumology at the University of Freiburg, Germany under Professor H Matthys.

Dr Virchow is Past Editor-in-Chief of *Respiratory Medicine* and Visiting Professor of the China-Japan-Friendship Hospital in Beijing, PR China. He is a fellow of the Royal College of Physicians in London (FRCP), the American College of Chest Physicians (FCCP), the American Academy of Allergy Asthma and Immunology (FAAAAI) and the European Respiratory Society (FERS) and a member of several medical organisations. Dr Virchow serves as Associate Editor for several journals in pulmonology and allergology and has served as an ad hoc reviewer for *The Lancet*, *The Lancet Respiratory Medicine*, the *European Respiratory Journal*, *Chest*, the *American Journal of Respiratory and Critical Care Medicine*, the *American Journal of Respiratory Cell and Molecular Biology*, *Allergy*, and many others. He is the author or co-author of more than 280 peer reviewed articles and author of several chapters for text books.

His special interests are research into the mechanisms of inflammation in chronic obstructive lung diseases such as asthma and COPD as well as the treatment of these conditions.

**Professor Louise V Wain** is a non-clinical Professor in Respiratory Research at the University of Leicester, UK and previously held a GSK / Asthma + Lung UK Chair. She has a keen research interest in utilising large-scale genetic epidemiological studies to drive mechanistic understanding of disease to underpin drug target discovery. Louise has led some of the largest genetic association studies of idiopathic pulmonary fibrosis and lung function to date, co-leads a national clinical study of long COVID (PHOSP-COVID), and enjoys multiple national and international multi-disciplinary collaborations to translate the findings for patient benefit. Louise is Deputy Chair of a Medical Research Council Board, an Associate Editor for the *European Respiratory Journal* and leads PhD training programmes.

**Dr Steve Walker** is Associate Professor and NIHR Consultant Senior Lecturer in Respiratory Medicine with a specialist interest in pleural disease at the University of Bristol. He co-chaired the 2024 European Respiratory Society (ERS) guideline on pneumothorax and was a member of the guideline committee for the 2023 British Thoracic Society Pleural Disease Guidelines. He is co-chair of the ERS Pleural Clinical Research Collaboration.

**Helen Ward** has been a Consultant Respiratory Physician at the Royal Wolverhampton NHS Trust since February 2013. She has a particular interest in integrated care, the assessment and management of chronic breathlessness and end of life care in non-malignant respiratory disease. She was a Trustee for the British Thoracic Society (BTS) and a member of the BTS Board for four years until June 2023, and is a current Board member of the Association of Respiratory Technology and Physiology (ARTP) Council. She also represents the BTS on the NHS England Respiratory Delivery Board which has oversight of the delivery of the Long Term Plan (LTP) for Respiratory. She is the West Midlands Respiratory Clinical Director for the Respiratory Network and chairs the new Midlands Breathlessness Forum.

**Dr Tom Ward** is an Academic Clinical Lecturer in Respiratory Medicine at the University of Leicester with research interests in COPD, integrated care, exercise physiology and pulmonary rehabilitation. He graduated from Imperial College London in 2012 and completed his PhD at Loughborough University in 2020 investigating the impact of novel exercise modalities for people with COPD. His current research focuses on improving access to specialist interventions for people living with COPD and enhancing the benefits of pulmonary rehabilitation. He is a member of the BTS Specialist Advisory Group on COPD.

**Dr Lindsay Welch** is an Associate Professor in Nursing Practice at the University Hospitals Dorset and

# SPEAKERS' BIOGRAPHICAL DETAILS

Bournemouth University. This senior clinical academic role is concerned with the development of a research skilled, curious, and quality orientated nursing workforce within UHD, simultaneously conceptualising and developing the Breathing and Fundamental Respiratory Care Research Group within the Department of Nursing at Bournemouth University.

Lindsay's research, education, and clinical activity are all centred around concept of fundamental respiratory nursing care, with a focus on health inequalities, and inclusion health. This includes self-management in respiratory nursing, respiratory diagnostics and the development and inclusion and access to digital technology in respiratory practice to enable timely patient centred care and treatment.

**Dr Daniel Wootton** is an NIHR Advanced Fellow and Senior Clinical Lecturer at the University of Liverpool and an Honorary Consultant Respiratory Physician at Aintree Hospital in Liverpool. He works on respiratory tract infections. Current grants and collaborations focus on the syndromes of community and hospital acquired pneumonia (CAP and HAP) and on the use of novel diagnostic strategies to improve patient outcomes and antimicrobial stewardship. Dan is a member of the NICE Pneumonia Guideline Committee, advisor to the EPIC 4 Guidelines for Hospital Acquired Infections, the NRAP Pneumonia Audit and the James Lind Alliance CAP Priority Setting Partnership. He is Clinical Lead for the Advancing Quality HAP Programme, delivering data-driven improvement in HAP management across hospitals in the Northwest.

**Dr Julie Worrell** is a Stromal Immunologist based at University College Dublin and an Affiliate Researcher at the University of Glasgow. Her work is focused on understanding the contributions of lung stromalimmune cell interactions to tissue homeostasis, injury, and repair. Dr Worrell is actively involved in several projects profiling epithelial cells, fibroblasts and immune cells and their role(s) in the development/pathogenesis of rare human lung diseases. She has a particular interest in the expression of immune molecules (chemokines and transcription factors) by lung fibroblasts, with a specific emphasis on their contributions to lung health (e.g., chronic fibrotic conditions, and viral infections).

**Dr Freda Yang** is an Academic Clinical Lecturer at the National Heart and Lung Institute and a Specialty Trainee in Respiratory and Internal Medicine in Northwest London. She holds an MBChB and MD from the University of Glasgow. As a RASP-UK Research Fellow, she studied exacerbations and corticosteroid effects in severe eosinophilic asthma post-IL-5 inhibition. In 2023, she joined Dr Chloë Bloom's group at Imperial to study asthma using large clinical datasets. Her research focuses on phenotyping severe asthma and identifying factors driving disease progression.

**Action for Pulmonary Fibrosis** **Stand D**

APF is a national UK charity. We bring people together to drive change so more people affected by pulmonary fibrosis (or lung scarring) can live well for longer. People living with lung scarring, their loved ones and the professionals caring for them are at the heart of everything we do.

We provide expert support, information, education, help a growing network of support groups and raise awareness of pulmonary fibrosis. We collaborate to drive change that improves health and care, and we provide vital resources to researchers, bringing hope for new and future treatments for this devastating disease.

Email: info@actionpf.org
Website: https://www.actionpf.org

**APR Medtech** **Stand 15**

At APR Medtech we work closely with healthcare professionals to understand their needs and provide tailored solutions that enhance patient care and drive efficiency in healthcare delivery.

Our team is committed to exceeding our customers' expectations, providing ongoing training, support, and guidance to ensure the seamless integration and optimal use of our products. We are proud to play a role in supporting the NHS and contributing to the betterment of patient outcomes.

At our core, we are driven by a mission to improve the quality of healthcare services using innovative medical technology. We believe that every patient deserves access to the best possible care, and we are dedicated to empowering healthcare providers with the tools they need to deliver exceptional care.

We are delighted to be exhibiting at BTS 2025 where we will be showcasing the Passio Pump Drainage System, a digital IPC used for the home management of recurrent pleural effusion. Please visit us at stand 15 to find out more about how this product can help your patients.

Email: passio@aprmedtech.com
Website: https://www.aprmedtech.com

**Aquilant Endoscopy** **Stand 16**

Aquilant Endoscopy, in partnership with Fujifilm Healthcare Europe, delivers innovative endoscopy and pulmonology solutions to support earlier diagnosis and improved patient outcomes. Our collaboration brings world-class imaging and minimally invasive technologies to clinicians across the UK and Ireland.

Expanding our pulmonology portfolio, we are proud to introduce Fujifilm's latest EBUS scope, offering enhanced ultrasound clarity and precision for lung cancer diagnosis and staging. Together, Aquilant and Fujifilm are committed to advancing clinical practice through trusted partnerships, education, and the launch of cutting-edge technologies that enable healthcare providers to deliver the highest standards of care.

Website: https://www.healthcare21.eu

**The Association for Respiratory Technology & Physiology (ARTP)** **Stand J**

The Association for Respiratory Technology & Physiology (ARTP) are the professional society focused on physiological measurement and interpretation within the field of respiratory medicine for the UK. We work alongside partner organisations and societies to produce position papers, national guidelines and standards for good practice. Our primary focus is the performance of respiratory/sleep physiological measurement, and the delivery of lung function and sleep services.

Email: admin@artp.org.uk
Website: https://www.artp.org.uk

**The Association of Chartered Physiotherapists in Respiratory Care (ACPRC)** **Stand F**

The Association of Chartered Physiotherapists in Respiratory Care promotes health and best practice in respiratory physiotherapy for the benefit of all. With over 1800 members the ACPRC is the largest national body of Physiotherapists interested in all aspects of Respiratory Care. Connecting with our members is at the heart of our organisation, and in addition to our ACPRC Conference which took place in April 2025 we also engage with members via:

- Regular short courses, including our new monthly listening events and lunchtime webinars
- Monthly e-Newsletters with latest updates for our members
- Dedicated social media pages via Facebook, Instagram, Bluesky and LinkedIn with regular updates, opportunities to network, access to a high number of followers and links to key resources
- A website that is packed with resources and also contains subspecialty networks such as the UK ECMO Physiotherapy network www.acprc.org.uk

- Our Journal of the Association of Chartered Physiotherapists in Respiratory Care, published bi-annually, peer reviewed and a member of crossref.
- Support with publishing your research
- Education grants

Furthermore, we support the development of National Guidelines related to cardio-respiratory care and are key stakeholders in many professional networks and special interest groups.

Email: secretary@acprc.org.uk
Website: https://www.acprc.org.uk

**The Association of Respiratory Nurses (ARNS)** **Stand I**

The Association of Respiratory Nurses (ARNS) was established in 1997 as a nursing forum to champion the specialty respiratory nursing community, promote excellence in practice, and influence respiratory health policy. ARNS also works to influence the direction of respiratory nursing care. ARNS has built its reputation through respiratory nurse leadership, evidence based practice, discussion and networking, empowering its members and collaborating with other organisations.

Email: info@arns.co.uk
Website: https://arns.co.uk

**Asthma + Lung UK** **Stand 47**

**AstraZeneca** **Stands 2, 45 & 46**

AstraZeneca is a global, science-led biopharmaceutical company, headquartered in the UK, that focuses on the discovery, development, and commercialisation of prescription medicines in Oncology, Rare Diseases, and BioPharmaceuticals. For more information, please visit www.astrazeneca.co.uk and follow us on Twitter @AstraZenecaUK.

Website: https://astrazeneca.co.uk

**BD** **Stand 34**

BD is a global medical technology company that is advancing the world of health by improving medical discovery, diagnostics, and care delivery. BD leads in patient and healthcare worker safety, providing technologies for medical research and clinical laboratories. The company provides innovative solutions that help enhance medical research, genomics, diagnosis of infectious diseases and cancer, medication management, infection prevention, surgical and interventional procedures, and diabetes management. BD Interventional – Peripheral Intervention develops innovative medical devices to address healthcare challenges and improving the quality of patients' lives. We are committed to pursuing technological innovations offering superior clinical benefits while reducing healthcare costs.

Email: olivia.broderick@bd.com
Website: https://www.bd.com/en-uk

**The British Association for Lung Research (BALR)** **Stand G**

The British Association for Lung Research (BALR) provides a focus for exchange of ideas between all manner of respiratory researchers, basic scientists and clinicians alike, to ferment collaboration and to further fundamental pulmonary research. Active for over twenty years, the main aim of the society is to promote respiratory research throughout the UK. We also support early career researchers in the field. The BALR is a registered charity (SC010151).

Email: admin@balr.co.uk
Website: https://www.balr.co.uk

**British Thoracic Society** **Stand A**

British Thoracic Society (BTS) is the largest, most authoritative, and inclusive respiratory professional society in the UK, working to achieve better lung health for all. We have over 4,500 members, including respiratory doctors, nurses, physiotherapists, physiologists, pharmacists, scientists, and other professionals with a respiratory interest. We aim to raise awareness of the impact of lung disease and champion the respiratory workforce.

BTS strongly believes in working collaboratively to influence policy and services to help reduce the health and economic burden of lung disease. We have comprehensive education and clinical workstreams with the goal of developing and promoting evidence-based care. We publish Guidelines, Quality Standards, Clinical Statements, and run national audits. Our members all have a drive to improve patient outcomes, and supporting our members to improve the health of respiratory patients is central to all that we do.

The Society's vision is 'Better Lung Health for all' and BTS aims to:

- influence the provision of the optimum respiratory workforce and the development of services that promote sustainable solutions and reduce health inequalities

- educate professionals to advance knowledge and share learning in the prevention, diagnosis and treatment of lung disease
- support all members of the respiratory team to improve standards of care

Email: bts@brit-thoracic.org.uk
Website: https://www.brit-thoracic.org.uk

**Broncus Medical, Inc** **Stand 21**

Broncus Medical, Inc is a publicly listed company based in San Jose, CA. The company's mission is to deliver navigation, diagnostic, and therapeutic technologies to treat patients with lung disease. Archimedes® Virtual Bronchoscopic Navigation System supports bronchoscopic procedures and to quickly and accurately navigate to tumors in the lungs. The Archimedes™ system supports a comprehensive lung access platform, including BTPNA (Bronchoscopic Trans-Parenchymal Nodule Access), which combines state-of-the-art nodule, vessel, and airway mapping, pathway guidance, and intra-operative fluoroscopy imaging – allowing for previously impossible biopsy and therapeutic access to virtually any location in the lung.

Email: info@broncus.com
Website: https://www.broncus.com

**Celltrion Healthcare UK Ltd** **Stand 9**

Celltrion Healthcare is an established global biopharmaceutical company based in South Korea that focuses on biosimilar innovation.

CELLTRION HEALTHCARE COMMITMENT TO BIOSIMILARS

Founded in 2002, Celltrion Healthcare offers an expanding portfolio of high-quality and competitively priced biosimilars supporting patient access across a broad range of therapy areas.

Celltrion's supply capability is based on a production capacity of 250,000L and a facility with a filling capacity of up to 18 million syringes per year.

UK--25-00146 Aug 2025

Email: UKMedical@celltrionhc.com
Website: https://www.celltrionhealthcare.co.uk

**Chiesi** **Stand 4**

Headquartered in Parma, Italy, Chiesi Farmaceutici is an international research-oriented biopharmaceuticals group with over 85 years' experience in the pharmaceutical sector with 31 affiliates worldwide, employing over 7,000 people. Chiesi develops and markets innovative therapeutic solutions in respiratory health, rare diseases, and specialty care. The company's mission is to improve people's quality of life and act responsibly towards both the community and the environment. As a certified B Corp since 2019, Chiesi is part of a global community of businesses that meet high standards of social and environmental impact. Chiesi Limited is headquartered in Manchester employing over 400 people.

Email: info.uk@chiesi.com
Website: https://www.chiesi.uk.com

**Cipla** **Stand 5**

Cipla has been developing respiratory medicines for patients for more than 40 years and we are the second largest global manufacturer of respiratory inhalers, committed to becoming a global leader in respiratory care. We have a range of high-quality respiratory products, designed with similar instructions and features to the market-leading originator brands. At Cipla, we have priced our range to be more sustainable and affordable so more NHS patients can receive optimal care. We are dedicated to minimising our environmental footprint in support of the NHS goal of becoming carbon net zero by 2040.

CIPCOR20240038 October 2024

Email: uk.info@cipla.com
Website: https://uk.cipla.com

**Creo Medical** **Stand 12**

Creo Medical is dedicated to enhancing diagnostic and therapeutic outcomes in pulmonology. Visit our booth to discuss our broad portfolio of bronchoscopy and navigational technology and devices.

Email: customerservice@creomedical.com
Website: https://www.creomedical.com

**Erbe Medical Ltd** **Stand 6**

Founded in 1851, Erbe Medical Ltd is a family-owned company shaping medical innovation worldwide. Erbe develops, manufactures, and markets advanced systems for electrosurgery, dissection, vessel sealing, plasma surgery, cryosurgery, hydrosurgery, and imaging. Its solutions support a wide range of disciplines, including gastroenterology, general surgery, gynaecology, pulmonology, and urology. With representation in 110 countries, the group has grown sales by 75% and its workforce by 85% in the past decade, now employing over 1,800 people globally. Backed by 260 R&D specialists and collaborations with leading physicians,

# EXHIBITORS' INFORMATION

Erbe continues to drive forward pioneering medical technologies that improve patient outcomes.
Email: sales@erbe-uk.com
Website: https://uk.erbe-med.com/uk-en

**Essential Pharma Ltd** **Stand 10**

At Essential Pharma, we believe that every patient should have access to the medicines they need. We work to develop and deliver medicines for patients in small, underserved or rare disease populations.
From manufacturing therapies otherwise at risk of discontinuation, to developing treatments for patient groups with few to no alternatives, we apply our expertise and find creative ways to deliver reliable, high-quality medicines that can transform lives.
With a growing portfolio of high-quality products reaching patients in approximately 70 countries, our medicines cover a range of therapeutic areas with a particular focus on rare disease, CNS, ophthalmology and gastroenterology.
Phone: +44 (0)1784 477 167
Email: info@essentialpharmagroup.com
Website: https://essentialpharmagroup.com

**Fannin** **Stand 18**

Fannin offers innovative, high-quality single-use endoscopic devices for pulmonologists across the UK. Our EBUS portfolio includes both FNA and FNB needles, available in nitinol and stainless steel, with advanced features such as the Twist-Lock mechanism to provide enhanced control, stability, and precision during endobronchial ultrasound-guided procedures.
Email: serviceuk@fannin.eu
Tel: +44 1453 874130
Website: https://www.fannin.eu
Social media: https://www.linkedin.com/company/fannin-ltd
https://www.youtube.com/channel/UCkqa0v0A6UjzmfgWfmFNoQw
https://x.com/fanninl?s=21

**Fisher & Paykel Healthcare** **Stand 24**

Fisher & Paykel Healthcare is a leading designer and manufacturer of products and systems for use in acute and chronic respiratory care, surgery and the treatment of obstructive sleep apnea. Driven by a strong sense of purpose, we are working to improve patient care and outcomes through inspired and world-leading healthcare solutions. The needs of our customers and their patients drive everything we do. We call this commitment Care by Design.
Website: https://www.fphcare.co.uk

**GSK** **Stands 3, 37 & 38**

GSK are a global biopharma company with a purpose to unite science, technology and talent to get ahead of disease together. We aim to positively impact the health of 2.5 billion people by the end of 2030. Our bold ambitions for patients are reflected in new commitments to growth and a step-change in performance.
NP-GB-RS-COCO-230002 | January 2024
Website: https://www.gsk.com/en-gb

**ILD IN** **Stand 49**

**Insmed** **Stands 26, 30 & 31**

Insmed is a global biopharmaceutical company on a mission to transform the lives of patients with serious and rare diseases. We are powered by purpose, a purpose to serve patients and their families with unwavering dedication. A purpose to find solutions where there were none before. A purpose to do what's right, even when it isn't easy.
A biotech company that empowers great people to deliver with a profound sense of urgency and compassion, life-altering therapies to small patient populations experiencing big health problems, transforming the lives of patients living with serious and rare diseases.
At Insmed, we are powered by our shared sense of purpose to serve patients. We don't always have a defined play book, but we operate with passion and creativity to find the best path forward. We take pride in our ability to challenge the status quo. Team members are comfortable operating outside their traditional roles and comfort zones, using what we already know to uncover what we don't.
Email: christopher.annis@insmed.com
Website: https://www.insmed.com

**Inspire Medical Systems** **Stand 17**

Inspire Medical Systems is a company involved in the treatment of moderate to severe Obstructive Sleep Apnoea with a hypoglossal nerve stimulation device that sits under the skin. The company was founded in 2007 and over 100,000 patients have been treated around

the world. Patient satisfaction is 94%. This was the world's first fully implantable device approved by the FDA for the treatment of OSA. Robust clinical data proving safety, efficacy and effectiveness is now out to 5 years and usage has commenced in the NHS. It is specifically for patients suffering from OSA who have become intolerant to CPAP.

Email: info@inspiresleep.com
Website: https://www.inspiresleep.co.uk

**Its Interventional** **Stand 32**

Putting the patient first is at the centre of everything we do at Its Interventional. With symptom management increasingly the primary focus for patients with recurrent pleural effusion, managing drainage at home and delivering optimal quality of life has never been more important. The Aspira Siphon Drainage system provides gentler drainage, ease of use and 'packability', meaning it's never been easier to mitigate drainage discomfort. Whilst facilitating easier travel for those patients wishing to take that last holiday or visit family. Gone are the days worrying about acquiring drainage materials at the patient's destination.

Why not visit us at our stand 32 to find out more.

Email: hello@itsinterventional.com
Website: https://www.itsinterventional.com

**LifeArc** **Stand 42**

LifeArc is a not-for-profit medical research organisation that turns promising scientific research into impact for people living with rare diseases and Global Health infections.

We form partnerships and provide scientific expertise and funding to help break down the barriers preventing scientific breakthroughs from becoming life-transforming treatments and cures. We have been doing this for more than 30 years and our work has resulted in five licensed medicines, including cancer drug pembrolizumab and lecanemab for Alzheimer's disease. Our goal is a world where no one with a rare disease or a global infectious disease misses out on life-changing innovation because of complexity, cost or risk.

Email: info@lifearc.org
Website: https://www.lifearc.org

**Mesothelioma UK** **Stand K**

Mesothelioma UK is a national charity dedicated to supporting people affected by mesothelioma, an asbestos-related cancer. Through expert nursing care, accredited information, and advocacy, the charity improves lives and promotes equitable access to treatment and clinical trials. Embedded within NHS hospitals, its network of Clinical Nurse Specialists ensures specialist support is available at the point of need. Mesothelioma UK also funds research, offers benefits advice, and raises awareness to help prevent future cases.

Email: info@mesothelioma.uk.com
Website: https://www.mesothelioma.uk.com

**MSD** **Stand 25**

At MSD, known as Merck & Co., Inc., Rahway, NJ, USA in the United States and Canada, we are unified around our purpose: We use the power of leading-edge science to save and improve lives around the world. For more than a century, we've been at the forefront of research, bringing forward medicines, vaccines and innovative health solutions for the world's most challenging diseases.

GB-NON-11119 | March 2025

Email: corporateaffairsuk@msd.com
Website: https://www.msd-uk.com

**The National Respiratory Audit Programme (NRAP)** **Stand B**

The National Respiratory Audit Programme (NRAP) is a suite of continuous clinical audits. There are five audits covering the following workstreams – COPD, adult asthma, children and young people's asthma, primary care (Wales only) and pulmonary rehabilitation.

NRAP aims to improve the quality of care, services and clinical outcomes for people with respiratory conditions, and healthcare improvement is central to the programme. NRAP's approach involves providing meaningful real-time data to participating services, engaging with local, regional and national stakeholders, supporting a comprehensive quality improvement programme, and using data insights from the data collected to inform national policy to support population-level change.

Email: NRAPinbox@rcp.ac.uk
Website: https://rcp.ac.uk/improving-care/national-clinical-audits/the-national-respiratory-audit-programme-nrap

**NeilMed Pharmaceuticals** **Stand 22**

NeilMed Pharmaceuticals, is a global leader in nasal and sinus care, offering a wide range of safe, effective, and

user-friendly products including sinus rinse kits, saline sprays, pediatric solutions, ear care, and wound wash. Founded with a mission to improve quality of life through better breathing and sinus health, NeilMed products are trusted by healthcare professionals and millions of users worldwide. With innovation, safety, and customer care at the core, NeilMed continues to set the standard in drug-free nasal and sinus hygiene.
Email: questions@neilmed.com
Website: https://www.neilmed.com

**NIOX Group plc** **Stands 43 & 44**
NIOX Group plc is the global leader in FeNO testing, with a strong reputation for innovation and excellence in the development of medical devices for the diagnosis and management of asthma. With NIOX VERO®, healthcare professionals have a reliable and non-invasive tool to help them better diagnose and manage asthma.
Email: sales.uk@niox.com
Website: https://www.niox.com

**NTM Patient Care UK and The NTM Network** **Stand M**
NTM Patient Care UK aims to improve the lives of people living with non-tuberculous mycobacterial (NTM) infection in the UK, by providing education and information to increase understanding of NTM for both patient and clinical communities. Leaflets and posters are provided on request or via our website.
The NTM Network for health care professionals, along with input from patients from NTM Patient Care have produced a Standards of Care document this year.
We hold regular Zoom support meetings which include various speakers followed by a questions and answers session.
Email: info@ntmpatientcare.uk
Website: https://ntmpatientcare.uk
Email: admin@ntmnetwork.uk.com
Website: https://www.ntmnetworkuk.com

**Olympus** **Stand 13**
At Olympus, we are committed to our purpose of making people's lives healthier, safer and more fulfilling. As a global medical technology company, we partner with healthcare professionals to provide best-in-class solutions and services for **early detection**, **diagnosis** and **minimally invasive treatment**, aiming to **improve patient outcomes** by elevating the standard of care in targeted disease states.
Olympus offers a variety of products and system solutions for **Respiratory Endoscopy**, constantly seeking to improve **lung cancer outcomes**, among other diseases. Olympus is committed to developing new technologies, products, services and solutions that comply with the toughest industry standards.
Proud to sponsor BTS once again, Olympus welcomes you to **stand 13** to explore our latest technology.
Email: info@olympus.co.uk
Website: https://www.olympus.co.uk/pulmonology

**OmniaMed Communications** **Stand 19**
GPnotebook is a clinical reference tool for primary care professionals (general practitioners, practice nurses and pharmacists), providing the information most relevant to primary care professionals. Over 30,000 concise articles cover specific clinical topics, while a growing library of videos, podcasts and educational modules support your learning and professional development.
GPnotebook is a subsidiary of OmniaMed Communications, who also publish a range of other resources for healthcare professionals including *Practice Nurse* journal, the *British Journal of Cardiology*, and DiabetesontheNet.com.
Email: jhall@omniamed.com
Website: https://gpnotebook.com
https://omniamedcommunications.com

**Orion Pharma (UK) Ltd** **Stand 14**
Orion Pharma (UK) Ltd is a subsidiary of Orion Corporation, a pharmaceutical company based in Finland. We are continuously developing new drugs and treatment methods. The core therapy areas of our pharmaceutical R&D are oncology and pain. UK therapy areas include Respiratory, Women's Health & Neurological disorders.
July 2025/CORP-233(4)
Email: uk.medicalinformation@orionpharma.com
Website: https://www.orionpharma.co.uk

**PCD Support Group** **Stand 50**
We are the UK's dedicated charity supporting those affected by Primary Ciliary Dyskinesia (PCD).

PCD is a rare genetic disease affecting approximately 1 in 7,500 people in the UK and is caused by abnormal motile cilia. Many of our patients develop bronchiectasis before they are diagnosed and require intense daily physiotherapy regimes.
At PCD Support UK, we put our patient community first. We are a team of volunteers whose mission it is to support families and individuals affected by PCD. We also work closely with our clinical professionals, championing research to improve the lives of people with PCD.
Email: chair@pcdsupport.org.uk
Website: https://www.pcdsupport.org.uk

**Pfizer Ltd** **Stand 28**
Website: https://www.pfizer.co.uk

**Pharming** **Stand 27**
Pharming is a global biopharmaceutical company dedicated to transforming the lives of patients with rare, debilitating, and life threatening diseases. Pharming is commercialising and developing an innovative portfolio of protein replacement therapies and precision medicines to serve the unserved rare disease patient.
Email: info@pharming.com
Website: https://www.pharming.com

**The Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS)** **Stand C**
The Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS), developed and run by the Royal College of Physicians, supports and accredits pulmonary rehabilitation services across the UK. Since its launch in 2018, PRSAS has aimed to improve the quality, consistency, and accessibility of care for people with chronic respiratory conditions. Through a structured accreditation process, PRSAS promotes clinical excellence, encourages continuous service improvement, and supports patient-centred care.
Email: pulmrehab@rcp.ac.uk
Website: https://www.prsas.org

**Rocket Medical** **Stand 20**
Rocket Medical has partnered the NHS for over 50 years, with our aim to help improve patient's lives. Come and visit us on our stand, where we can demonstrate how we can support your patient's treatment journey for pleural effusion or pneumothorax; including Rocket homecare for supporting patient's care from hospital into the home. For information about any of Rocket Medical's products please contact 0191 419 6949 or homecaresupport@rocketmedical.com or www.rocketmedical.com.
Email: customerservices@rocketmedical.com
Website: https://www.rocketmedical.com

**The Royal College of Speech and Language Therapists** **Stand H**
The Royal College of Speech and Language Therapists is the professional body for speech and language therapists and assistant practitioners. We promote excellence in practice and influence health, education and social care policies to achieve the best possible outcomes for people with communication and swallowing difficulties.
Email: info@rcslt.org
Website: https://www.rcslt.org

**Sanofi / Regeneron** **Stands 1, 35, 36, 39 & 40**
**Sanofi** is an innovative global healthcare company, driven by one purpose: we chase the miracles of science to improve people's lives. Our team, across some 100 countries, is dedicated to transforming the practice of medicine by working to turn the impossible into the possible. We provide potentially life-changing treatment options and potentially life-saving vaccine protection to millions of people globally, while putting sustainability and social responsibility at the centre of our ambitions.

**Regeneron** is a leading biotechnology company that invents, develops and commercializes life-transforming medicines for people with serious diseases. Founded and led by physician-scientists, our unique ability to repeatedly and consistently translate science into medicine has led to numerous approved treatments and product candidates in development, most of which were homegrown in our laboratories.
Email: uk-medicalinformation@sanofi.com
Website: https://www.sanofi.co.uk

**SarcoidosisUK** **Stand L**
SarcoidosisUK is the UK's charity dedicated to sarcoidosis. It is a rare disease, with poor-quality information, low levels of support and awareness and very little funding for a cure.

SarcoidosisUK is working to change this.
The charity has four main goals:

- to provide accurate, detailed information to people with sarcoidosis, their carers, and medical professionals
- to provide emotional support for those affected by sarcoidosis by telephone, email, social media and through regional support groups
- to spread awareness and understanding of sarcoidosis among medical professionals and more widely
- to raise funds for focused medical research to improve outcomes for sarcoidosis patients

Email: info@sarcoidosisuk.org
Website: https://www.sarcoidosisuk.org

**STADA Group, Thornton & Ross Ltd** **Stands 7 & 8**

Thornton & Ross, part of the STADA group, focuses on caring for people's health through a broad portfolio including generics, biosimilars and non-prescription consumer healthcare products as a trusted partner supplying the NHS. STADA sells its products in more than 120 countries, employing over 12,000 people worldwide.
Email: hcpsupport@thorntonross.com
Website: https://www.thorntonross.com

**Stirling Anglian Pharmaceuticals** **Stand 33**

Based in the UK, Stirling Anglian is committed to medicines optimisation. It has sourced and developed a portfolio of medicines to help the NHS curb waste – across a range of conditions that currently place unnecessary and avoidable pressure on NHS resources. At a time when there is such pressure on the NHS to reduce costs we believe we offer a real and practical solution.
We work closely with stakeholders across the NHS to identify real-world problems, and develop value-based solutions that support the delivery of efficient and cost-effective healthcare.
Website: https://www.stirlinganglianpharmaceuticals.com

**Sunvou** **Stand 11**

Sunvou: Leading the Way in FeNO Technology
Sunvou is recognized worldwide as a pioneer in FeNO innovation, consistently setting the benchmark for precision, reliability, and ease of use in airway inflammation diagnostics.
The HA1101 device perfectly illustrates this leadership, delivering fast, accurate, and reproducible results in a compact, portable format designed for both clinical and point-of-care settings. With streamlined operation, intuitive user experience, and robust engineering, it ensures confidence for healthcare professionals and comfort for patients alike. Offering lower purchase and operating costs than competing systems, the HA1101 provides exceptional long-term value without compromising on performance, making advanced respiratory diagnostics more accessible than ever.
Email: info@diagenics.co.uk
Website: https://www.diagenics.co.uk

**Vapotherm** **Stand 29**

Vapotherm are a global medical technology company focused on the development and promotion of our proprietary High Velocity Therapy products that are clinically indicated for patients of all ages suffering from respiratory distress caused by: Asthma, Bronchitis, CHF, COPD, Dyspnoea, Pneumonia and prolonged mechanical.
Our High Velocity Therapy delivers non-invasive ventilatory support by providing heated, humidified, and oxygenated air at a high velocity to patients through a comfortable small-bore nasal interface.
Our Precision Flow and HVT 2.0 systems are clinically validated alternatives to, and address many limitations of, the current standard of care for the treatment of undifferentiated respiratory distress in a hospital setting.
Randomised controlled trials have shown high velocity therapy to be non-inferior to NiPPV in preventing intubation for spontaneously breathing patients, from premature babies in the NICU to hypercapnic COPD patients experiencing moderate to severe acute exacerbations in the Emergency Department and the ICU.
Email: ukinfo@vtherm.com
Website: http://www.vapotherm.com

**Vitalograph** **Stand 41**

Vitalograph develops, manufactures, and markets respiratory diagnostic solutions for use in the detection, diagnosis and management of respiratory conditions. Our mission is to improve the lives of patients with lung conditions by offering the broadest range of diagnostic solutions, enabling a better understanding of lung health. Vitalograph also enables respiratory research and drug development through our Clinical Trial services, and we have supported many successful clinical trials for some

of the world's leading pharmaceutical companies, biotechs, and contract research organisations. We serve global markets with operations in the USA, UK, Ireland, Germany, and Japan.

Email: contact@vitalograph.com
Website: https://vitalograph.com

# BTS/ BALR/ A+ LUK Early Career Investigator Award Symposium

## T1 RETROSPECTIVE EVALUATION OF ARTIFICIAL INTELLIGENCE (AI) SOFTWARE FOR THE DETECTION OF ACUTE PULMONARY EMBOLISM (PE) IN CT PULMONARY ANGIOGRAMS (CTPA): A PRELIMINARY GROUND-TRUTH ANALYSIS FROM THE AID-PE STUDY

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**Background** Acute PE is a life-threatening condition requiring prompt diagnosis and treatment. Whilst CTPA has improved PE detection, diagnostic accuracy remains limited by interobserver variability and suboptimal image acquisition, which contributes to under- and over-diagnosis. These errors may lead to inappropriate anticoagulation or missed treatment opportunities.

FDA-approved AI software for acute PE detection is increasingly deployed in clinical workflows. However, validation often relies on comparison with initial radiology reports, which may themselves contain diagnostic inaccuracies. The AID-PE study (NCT06093217) addresses this limitation by using expert thoracic radiologists and senior clinicians to establish reference standards and clinical outcomes – believed to be one of the first such study to do so.

**Purpose** To assess whether integrated AI software would improve diagnostic accuracy among general radiologists and reduce PE-related clinical risk in a single-centre DGH.

**Methods** A subset of consecutive CTPAs from the AID-PE comparator cohort was retrospectively assessed using automated PE detection software. A senior respiratory clinician reviewed each initial radiology report determining whether scans were positive or negative for acute PE. The imaging reference standard was determined by independent expert thoracic radiologist review, blinded to the initial report and AI output.

**Results** 664 CTPAs were re-evaluated (mean age 63 ± 19; 310 males, 354 females). Expert review detected acute PE in17.2% (114/664) of the original scans. Compared to expert review, the initial report demonstrated a sensitivity of 88.7% (95% CI 81.7–93.7%) and specificity of 98.7% (95% CI 97.8–99.6%). AI achieved slightly higher sensitivity at 90.4% (95% CI 84.9–95.8%) and comparable specificity at 98.5% (95% CI 97.5–99.5%).

The initial report false negative rate was 12.3% (14/114) with AI correctly identifying 3 of these missed cases. The initial report false positive rate 1.3% (7/550) with AI correctly reclassifying 6 as negative. Two patients with under-reported PE experienced further VTE events, whilst one major bleeding episode occurred following treatment of an initial report false positive.

**Conclusions** Preliminary results from the AID-PE study suggest AI closely aligns with expert interpretation than initial radiology reports. Findings highlight the potential for AI to improve diagnostic accuracy and reduce harm from misdiagnosis in acute PE.

## T2 HAEMOPHILUS COMMENSALS WITHIN THE AIRWAY MICROBIOME MODULATE IMMUNE RESPONSES AND DICTATE VIRAL SUSCEPTIBILITY IN COPD

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10.1136/thorax-2025-BTSabstracts.2

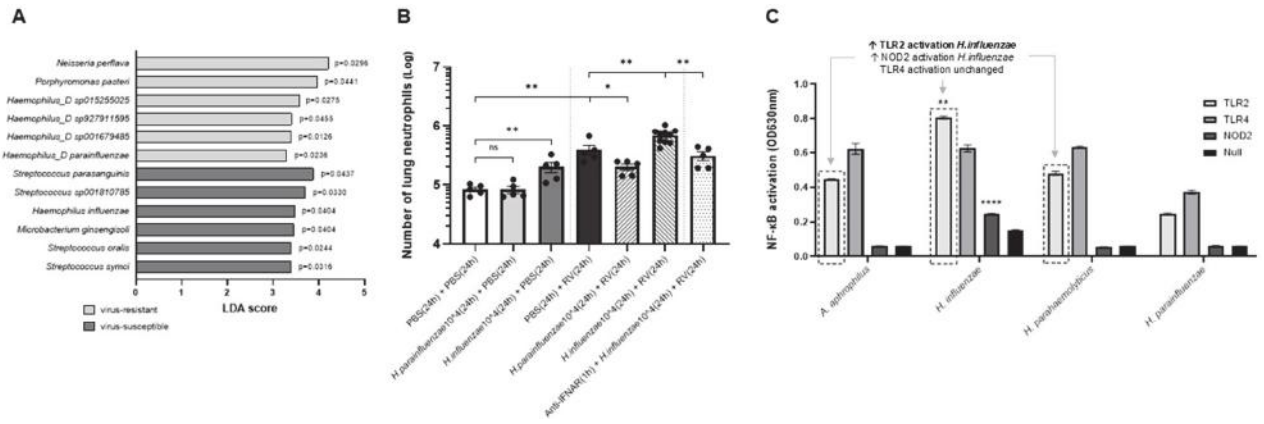
**Introduction** Viral infections trigger exacerbations of chronic obstructive pulmonary disease (COPD) through poorly defined mechanisms. Perturbation of the airway microbiome with expansion of *Haemophilus* genus is reported in COPD. *Haemophilus influenzae* colonisation is strongly associated with exacerbation susceptibility but roles played by other resident *Haemophilus* commensals are unknown. We sought to investigate airway microbiome-immune interactions and define roles played in driving COPD viral susceptibility.

**Methods** We used metagenomics to profile microbiota in subjects with COPD undergoing rhinovirus challenge (n=21) and analysed relationships between commensals and immunological/clinical responses to infection. We employed mouse models to study the specific effects of *Haemophilus* species upon immune responses to rhinovirus infection with experimental manipulations to define functional relationships.

**Results** Sputum metagenomics indicated that baseline *Haemophilus* genus abundance correlated with rhinovirus induction of antiviral mediators (sputum interferon-λ1, r=0.4846, $P<0.05$), and other immunological readouts including sputum % neutrophils (r=0.8108, $P<0.05$). Linear discriminant analysis effect size (LEfSe) analysis comparing susceptible (i.e. developed exacerbation) versus resistant (i.e. no clinical/immunological response to rhinovirus challenge) COPD indicated that *H.influenzae* and several *Streptococci* were associated with viral susceptibility whilst *H.parainfluenzae* and other *Haemophilus_D* species were associated with resistance.

Using mouse models of *Haemophilus* dysbiosis, we found that *H.influenzae* potently exacerbated early airway antiviral (type-I/III interferon) and subsequent pro-inflammatory responses (neutrophilic inflammation, exhibiting more activated/pathogenic phenotype) to rhinovirus. Conversely, *H.parainfluenzae* administration attenuated neutrophilic responses to rhinovirus whilst other *Haemophilus* species tested had minimal immunomodulatory effects.

Blockade of immune signalling using an anti-IFNAR1 antibody abrogated *H.influenzae*-mediated pro-inflammatory responses to rhinovirus, indicating that the exacerbating effect is dependent on type-I interferon. This effect was retained when heat-killed *H.influenzae* was administered, suggesting a mechanism via pattern-recognition receptor (PRR) stimulation. Comparison of relative PRR stimulation using a HEK293 cell reporter assay indicated that *H.influenzae* more potently activated TLR2/NOD2 than other *Haemophilus* species. Given that TLR2 activation can promote type-I interferon, ongoing




**Abstract T2 Figure 1 (A) Sputum bacterial species associated with susceptibility to or resistance from rhinovirus challenge in COPD.** Linear Discriminant Analysis Effect Size (LEfSe) of shotgun metagenomics, calculated with LDA alpha score 0.05 and LDA threshold 2.0. **(B) *Haemophilus* species-specific augmentation of rhinovirus-induced neutrophil recruitment in mouse lung is driven by type-I interferon.** Flow cytometry showing numbers of neutrophils in lung from BALB/c mice treated with intranasal *H.influenzael H.parainfluenzae* ($1x10^4$ CFU) and subsequently infected with RV-A1B (MOI1) for 24 hours; anti-IFNAR1 antibody administered intranasally one hour prior to *Haemophilus* treatment. Data generated from two independent experiments (n=5–10), represented as mean ± SEM. Statistical significance analysed by Mann-Whitney U, where $^{*}P<0.05$ and $^{**}P<0.01$. **(C) Comparison of PRR stimulation by *Haemophilus* species in HEK293-reporter assay.** NF-κB activation (OD630nm) of HEK derived reporter cell lines, TLR2/TLR4/NOD2 or Null (control) by *Haemophilus* species (MOI1). Statistical significance analysed by Mann-Whitney U, where $^{**}P<0.01$ and $^{****}P<0.0001$ difference between *H.influenzae* and other species

studies are examining effects of TLR2 inhibition within these models.

**Conclusion** *Haemophilus* commensals within the airway microbiome exhibit differing pro-inflammatory and anti-inflammatory effects which may influence COPD viral susceptibility. Further studies are needed to define the optimum approaches for therapeutically targeting *Haemophilus* dysbiosis in COPD.

## T3 LUNG EOSINOPHILS IN EOSINOPHILIC COPD PATIENTS DIFFER FUNCTIONALLY BUT NOT PHENOTYPICALLY

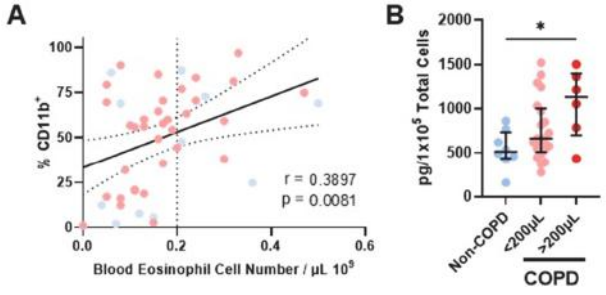
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10.1136/thorax-2025-BTSabstracts.3

**Introduction** Chronic obstructive pulmonary disease (COPD) is a common respiratory disease, associated with inflammation and irreversible lung damage leading to breathlessness. Up to 40% of COPD patients display elevated blood eosinophil counts which are associated with higher exacerbation risks. However, the role eosinophils perform in COPD and heighten exacerbation risk is not understood.

**Methods** Resected background lung tissue was collected from COPD patients (n=26) and non-COPD controls (n=7). COPD samples were stratified based on blood eosinophil count into <200 cells/μL (low) and ≥200 cells/μL (high) COPD subgroups. Eosinophils were phenotyped by flow cytometry using CD62L (resident eosinophil) and CD123 (inflammatory eosinophil) subset markers plus CD63 (cell degranulation), HLA-DR (antigen presentation), and CD11b (cell adhesion) activation markers. Eosinophil peroxidase (EPO) was quantified in cell supernatants by ELISA. Transcriptional analysis of isolated blood eosinophils from non-COPD (n=4) and COPD (n=5) was performed by bulk RNA-sequencing.

**Results** Patient characteristics for non-COPD patients (age: 70.5 ±10.0,%$FEV1_{pred}$: 99.2% ±15.8, smoking park years (PY): 34.0 ±12.9, 53.3% male), low eosinophil COPD (age: 68.0 ±9.2,%$FEV1_{pred}$: 71.0% ±25.9, PY: 36.0 ±24.3, 41.4% male) and high eosinophil COPD (age: 68.5 ±13.0,% $FEV1_{pred}$: 61.0% ±26.1, PY: 44.0 ±18.2, 69.2% male) showed no statistical differences between COPD subgroups. While eosinophil subset CD62L/CD123 and activation markers CD63/HLA-DR were unaltered, CD11b expression positively correlated with blood eosinophil number (r=0.39, p=0.008; figure 1A). Normalised for cell number, EPO release increased 2.2-fold in eosinophils derived from high eosinophil COPD compared to non-COPD (p=0.037) (figure 1B). Moreover, transcriptional blood eosinophil data demonstrated enrichment for degranulation ($p_{adj}$=2.06E-07), metabolic pathways ($p_{adj}$=0.0002), and DNA repair ($p_{adj}$=0.0005) in COPD blood-derived eosinophils.




**Abstract T3 Figure 1** COPD eosinophil phenotype and function. **(A)** Flow cytometry analysis of lung-derived eosinophils $CD11b^{+}$ cellular expression associated with clinical blood eosinophil count (Spearman correlation, blue dots-non-COPD, red dots-COPD). **(B)** Lung-derived eosinophils normalised to cell number eosinophil peroxide release (Kruskal-Wallis test). Data show median ± IQR. *P ≤ 0.037

**Conclusion** Overall, data suggest primed COPD blood-derived eosinophils enter the lung via more efficient contact-dependant adhesion and migration via CD11b. In high eosinophil COPD patients this facilitates focused localisation of excessive EPO degranulation in the lung.

## T4 SEX DIFFERENCES IN ASTHMA: A LARGE-SCALE MULTI-OMICS STUDY

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10.1136/thorax-2025-BTSabstracts.4

**Background** Adult women have a higher prevalence and greater severity of asthma, along with increased asthma-related mortality compared to men, yet the underlying molecular and genetic mechanisms remain poorly understood.

**Methods** To investigate sex-specific characteristics and mechanisms in asthma, we analysed clinical, transcriptomic, proteomic, metabolomic, and metagenomic data from 568 adult participants in the pan-European U-BIOPRED cohort. Differential expression analysis, functional enrichment analysis, and

**Abstract T4 Table 1** Sex differences in clinical characteristics of mild/moderate asthma, severe asthma, and healthy controls

| Characteristics | Healthy controls | | | Mild/moderate asthma | | | Severe asthma | | |
|---|---|---|---|---|---|---|---|---|---|
| | Female | Male | p-value* | Female | Male | p-value# | Female | Male | p-value& |
| Subjects, n | 39 | 62 | | 44 | 44 | | 237 | 142 | |
| Age, years | 40.3 ± 2.2 | 38.0 ± 1.7 | 0.399 | 41.5 ± 2.2 | 41.8 ± 2.5 | 0.924 | 50.4 ± 0.9 | 54.6 ± 1.1 | 0.004 |
| Age at diagnosis, years | NA | NA | NA | 16 (5, 34) | 13 (6, 28) | 0.956 | 23 (9, 40) | 30 (8, 44) | 0.139 |
| Race, Caucasian | 3/39 (7.7%) | 4/62 (6.5%) | 1.000 | 41/44 (93.2%) | 41/44 (93.2%) | 1.000 | 213/237 (89.9%) | 129/142 (90.8%) | 0.897 |
| BMI, $kg \cdot m^{-2}$ | 23.8 ± 0.54 | 26.3 ± 0.44 | 0.001 | 25.2 ± 0.71 | 26.3 ± 0.63 | 0.255 | 30.0 ± 0.45 | 28.5 ± 0.44 | 0.026 |
| Ex-smokers/non-smokers, n | 11/28 | 9/53 | 0.154 | 7/37 | 6/38 | 1.000 | 62/175 | 53/89 | 0.030 |
| Smoking history, pack-years | 1.0 [0.6, 3.5] | 0.7 [0.2, 3.0] | 0.447 | 3.5 [1.4, 4.1] | 4.3 [1.5, 4.9] | 0.352 | 5.2 [2.0, 14.8] | 13.0 [4.0, 23.0] | 0.018 |
| Spirometry | | | | | | | | | |
| Pre-bronchodilator $FEV_1$,% pred | 98.4 [92.7, 107.2] | 104.3 [95.7, 111.2] | 0.180 | 92.3 [81.8, 101.8] | 91.6 [73.1, 100.6] | 0.387 | 70.2 [52.4, 86.4] | 62.7 [48.3, 77.3] | 0.010 |
| Pre-bronchodilator FVC,% pred | 108.1 [99.7, 120.1] | 108.7 [100.8, 114.5] | 0.594 | 108.3 [97.3, 113.2] | 104.2 [86.8, 118.5] | 0.325 | 88.8 [75.9, 103.2] | 84.8 [73.5, 97.1] | 0.043 |
| Pre-bronchodilator $FEV_1$/FVC,% | 79.8 [74.9, 83.3] | 80.1 [75.3, 82.8] | 0.810 | 75.5 [70.1, 78.8] | 71.3 [63.7, 77.4] | 0.094 | 66.7 [56.2, 75.9] | 61.3 [49.2, 68.6] | <0.001 |
| Pre-bronchodilator $FEF_{25-75\%}$,% pred | 70.9 [62.4, 95.4] | 85.80 [73.1, 104.7] | 0.034 | 60.9 [43.1, 70.1] | 59.7 [44.2, 69.4] | 0.932 | 32.1 [19.1, 52.7] | 29.6 [17.6, 45.9] | 0.370 |
| Change in FEV1, post-salbutamol, % | NA | NA | NA | 7.9 [4.5, 13.4] | 12.3 [6.7, 21.1] | 0.009 | 14.0 [6.3, 22.7] | 13.4 [5.2, 19.7] | 0.201 |
| Body plethysmography | | | | | | | | | |
| Airflow limitation,% | 0/39 (100.0%) | 0/62 (100.0%) | NA | 5/44 (11.4%) | 10/44 (22.7%) | 0.257 | 113/237 (47.7%) | 79/142 (55.6%) | 0.164 |
| TLC,% pred | 107 [96, 114] | 101 [95, 107] | 0.051 | 106 [97, 123] | 106 [97, 117] | 0.581 | 110 [100, 122] | 106 [94, 112] | 0.003 |
| RV/TLC,% pred | 1.6 [1.5, 1.8] | 1.7 [1.4, 2.1] | 0.467 | 1.6 [1.4, 1.9] | 2.0 [1.8, 2.5] | 0.001 | 2.2 [1.8, 2.5] | 2.9 [2.5, 3.3] | <0.001 |
| $sG_{aw}$, $kPa^{-1} \cdot s^{-1}$ | 1.5 [1.1, 2.0] | 1.7 [1.1, 2.3] | 0.438 | 1.4 [1.1, 2.6] | 1.1 [0.7, 1.8] | 0.118 | 0.7 [0.4, 1.1] | 0.8 [0.4, 1.1] | 0.383 |
| Biomarkers | | | | | | | | | |
| Blood eosinophils, $10^9$/L | 0.1 [0.06, 0.2] | 0.1 [0.1, 0.2] | 0.521 | 0.1 [0.1, 0.3] | 0.2 [0.1, 0.3] | 0.004 | 0.2 [0.1, 0.4] | 0.2 [0.1, 0.4] | 0.686 |
| Blood neutrophils, $10^9$/L | 3.3 [2.7, 5.2] | 2.9 [2.3, 3.9] | 0.032 | 3.2 [2.9, 4.5] | 3.3 [2.7, 4.3] | 0.720 | 5.0 [3.7, 6.7] | 4.7 [3.7, 7.1] | 0.868 |
| Sputum eosinophils,% | 0.0 [0.0, 0.4] | 0.2 [0.0, 0.2] | 0.640 | 0.7 [0.2, 3.4] | 1.1 [0.2, 3.0] | 0.477 | 3.3 [0.6, 19.4] | 2.4 [0.4, 16.1] | 0.827 |
| Sputum neutrophils,% | 29.3 [16.5, 41.3] | 42.6 [21.2, 68.7] | 0.129 | 44.5 [27.5, 68.7] | 39.1 [23.4, 60.8] | 0.509 | 52.5 [32.2, 71.0] | 57.4 [39.0, 84.8] | 0.112 |
| FeNO, ppb | 16.3 [12.4, 21.6] | 21.8 [15.0, 32.3] | 0.017 | 20.0 [15.0, 28.4] | 39.0 [22.0, 63.0] | <0.001 | 24.0 [14.0, 47.3] | 29.0 [20.5, 53.0] | 0.013 |
| IgE, IU/ml | 16.0 [7.0, 45.2] | 31.0 [10.0, 85.0] | 0.143 | 100.0 [45.0, 188.0] | 88.7 [61.2, 307.0] | 0.230 | 108.0 [41.0, 313.1] | 128.0 [51.8, 387.0] | 0.055 |
| CRP, mg/L | 1.0 [0.4, 2.2] | 0.6 [0.3, 1.4] | 0.156 | 1.1 [0.5, 2.6] | 0.6 [0.3, 1.1] | 0.042 | 2.5 [1.1, 5.1] | 1.9 [0.9, 4.6] | 0.100 |
| Symptoms | | | | | | | | | |
| ACQ5 | NA | NA | NA | 4.0 [2.0, 5.0] | 4.5 [1.1, 7.8] | 0.434 | 12.0 [8.0, 16.0] | 10.0 [5.0, 14.0] | 0.005 |
| AQLQ | NA | NA | NA | 196.0 [172.0, 207.0] | 198.0 [172.5, 209.0] | 0.918 | 142.0 [112.3, 166.8] | 153.0 [121.0, 183.5] | 0.013 |
| Exacerbations | | | | | | | | | |
| Exacerbations (past year), n | NA | NA | NA | 0.0 [0.0, 1.0] | 0.0 [0.0, 0.0] | 0.163 | 2.0 [1.0, 3.0] | 2.0 [0.0, 3.0] | 0.005 |
| Severe exacerbations (past year), n | NA | NA | NA | 0.0 [0.0, 0.0] | 0.0 [0.0, 0.0] | 0.24 | 2.0 [1.0, 3.0] | 1.0 [0.0, 2.0] | 0.011 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Medical histories | | | | | | | | | |
| Pneumonia or bronchitis | 0/39 (0.0%) | 0/62 (0.0%) | NA | 13/41 (31.7%) | 4/42 (9.5%) | 0.026 | 147/226 (65.0%) | 78/134 (41.8%) | 0.237 |
| Emphysema or COPD | 0/39 (0.0%) | 0/62 (0.0%) | NA | 1/44 (2.3%) | 2/44 (4.5%) | 1.000 | 13/234 (5.6%) | 6/141 (4.3%) | 0.754 |
| Comorbidities | | | | | | | | | |
| Allergic rhinitis diagnosed | 1/38 (2.6%) | 4/58 (6.9%) | 0.653 | 22/43 (51.2%) | 20/39 (51.3%) | 1.000 | 129/224 (57.6%) | 57/134 (42.5%) | 0.008 |
| Eczema diagnosed | 3/39 (7.7%) | 2/62 (3.2%) | 0.592 | 15/43 (34.9%) | 10/44 (22.7%) | 0.310 | 79/234 (33.8%) | 43/142 (30.3%) | 0.559 |
| GORD diagnosed | 1/39 (2.6%) | 3/62 (4.8%) | 0.963 | 10/44 (22.7%) | 6/43 (14.0%) | 0.436 | 121/229 (52.8%) | 54/140 (38.6%) | 0.011 |
| Hay fever diagnosed | 1/38 (2.6%) | 9/61 (14.8%) | 0.109 | 25/44 (56.8%) | 21/41 (51.2%) | 0.764 | 107/224 (47.8%) | 55/140 (39.3%) | 0.140 |
| Nasal polyps diagnosed | 1/39 (2.6%) | 2/61 (3.3%) | 1.000 | 4/44 (9.1%) | 3/44 (6.8%) | 1.000 | 59/233 (25.3%) | 69/139 (49.6%) | <0.001 |
| Non-allergic rhinitis diagnosed | 0/39 (0.0%) | 1/61 (1.6%) | 1.000 | 5/42 (11.9%) | 3/42 (7.1%) | 0.710 | 32/229 (14.0%) | 22/137 (16.1%) | 0.695 |
| Psychiatric disease diagnosed | 3/39 (7.7%) | 2/62 (3.2%) | 0.592 | 7/44 (15.9%) | 0/44 (0.0%) | 0.018 | 30/237 (12.7%) | 5/142 (3.5%) | 0.005 |
| Medications | | | | | | | | | |
| ICS | NA | NA | NA | 44/44 (100.0%) | 43/44 (97.7%) | 1.000 | 229/229 (100.0%) | 138/139 (99.3%) | 1.000 |
| OCS | NA | NA | NA | 0/44 (0.0%) | 0/44 (0.0%) | NA | 121/234 (51.7%) | 78/141 (55.3%) | 0.568 |
| OCS dose, prednisolone (equ.) mg | NA | NA | NA | NA | NA | NA | 16.4±11.0 | 15.7±10.7 | 0.667 |
| SABA | NA | NA | NA | 30/44 (68.2%) | 37/44 (84.1%) | 0.132 | 197/232 (84.9%) | 103/139 (74.1%) | 0.015 |
| LABA | NA | NA | NA | 2/44 (4.5%) | 2/44 (4.5%) | 1.000 | 226/229 (98.7%) | 137/139 (98.6%) | 1.000 |
| Antibiotic | NA | NA | NA | 0/44 (0.0%) | 0/44 (0.0%) | NA | 53/234 (22.6%) | 27/142 (19.0%) | 0.481 |
| LAMA | NA | NA | NA | 0/44 (0.0%) | 0/44 (0.0%) | NA | 50/230 (21.7%) | 36/141 (25.5%) | 0.475 |
| Omalizumab | NA | NA | NA | 0/44 (0.0%) | 0/44 (0.0%) | NA | 36/236 (15.3%) | 16/141 (11.3%) | 0.363 |
| LTM | NA | NA | NA | 0/44 (0.0%) | 0/44 (0.0%) | NA | 100/232 (43.1%) | 63/142 (44.4%) | 0.895 |

*Compare between female and male healthy controls; #Compare between female and male mild/moderate asthma; &Compare between female and male severe asthma.
**Abbreviations:** ACQ5: Asthma Control Questionnaire-5; AQLQ: Asthma Quality of Life Questionnaire; BMI: body mass index; COPD: chronic obstructive pulmonary disease; CRP: C-reactive protein; $FEF_{25-75\%}$: forced expiratory flow at 25% and 75% of the pulmonary volume; FeNO: fractional exhaled nitric oxide; $FEV_1$: forced expiratory volume in 1 second; FVC: forced vital capacity; GORD: Gastro-oesophageal reflux disease; LABA: long-acting β-agonist; LAMA: long-acting muscarinic antagonist; IgE: immunoglobulin E; ICS: Inhaled corticosteroids; LTM: Leukotriene modifier; NA: not applicable; OCS: oral corticosteroids; RV: residual volume; SABA: short-acting β-agonist; sGAW: specific airway conductance; TLC: total lung capacity.

weighted gene co-expression network analysis (WGCNA) were conducted.

**Results** In mild/moderate asthma, clinical characteristics among women and men were largely comparable, but women exhibited a higher prevalence of type 2-low phenotypes ($p = 0.013$), characterised by lower FeNO levels ($p < 0.001$) and reduced blood eosinophil counts ($p = 0.004$). Women also showed higher CRP levels ($p = 0.042$) and prevalence of a history of lung infections ($p = 0.026$). In severe asthma, sex-related differences were more pronounced. Men with severe asthma displayed more pronounced pulmonary dysfunction, including greater declines in spirometry values ($p < 0.001$), increased residual volume ($p < 0.001$), and elevated mucin production. In contrast, women with severe asthma experienced a greater symptom burden ($p = 0.005$) and more frequent exacerbations ($p = 0.005$), along with an increased prevalence of comorbidities, including allergic rhinitis and psychiatric disorders. In women with severe asthma, bronchial biopsy transcriptomic analysis revealed enhanced airway remodelling and impaired mitochondrial and ciliary function, while blood transcriptomics indicated stronger innate and adaptive immune responses with enhanced T-cell activation. Moreover, proteomic analysis showed heightened type 2 immune activity in female patients with severe asthma, evidenced by increased expression of TSLP, CCL11, and increased GZMB, suggesting increased cytotoxicity. Sputum metagenomic profiling further revealed a higher prevalence of airway infections in women with severe asthma, with a higher prevalence of *Haemophilus*, *Moraxella*, and *Pseudomonas* species. Clinical data and metabolomic analyses showed comparable medication usage and corticosteroid adherence between sexes. Interestingly, circulating androgen levels were inversely associated with asthma severity in both sexes.

**Conclusions** This large-scale multi-omics study reveals significant sex differences in asthma pathophysiology. Sex specific immunopathological differences suggest sex may be relevant to the choice of biologic.

On behalf of the UBIOPRED collaboration.

## T5 REDUCED AIRWAY REMODELLING AND INCREASED EPITHELIAL BARRIER INTEGRITY IN BRONCHIAL BIOPSIES FROM PATIENTS WITH T2 HIGH SEVERE ASTHMA FOLLOWING MEPOLIZUMAB TREATMENT

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**Background** Mepolizumab, an anti-IL-5 biologic used to treat T2 high severe asthma (T2 high SA), reduces exacerbation and improves asthma control. Airway remodelling is associated with persistent symptoms and lung function decline. Limited evidence exists investigating the effect of mepolizumab on airway remodelling.

**Objective** We aimed to investigate the effect of mepolizumab treatment on airway remodelling in patients with T2 high SA, hypothesising a reduction in airway remodelling markers and an increase in epithelial barrier repair following 6 months of treatment.

**Methods** Bronchial biopsies from 20 patients with T2 high SA were collected by bronchoscopy at baseline (T0) and 6 months (T6) post mepolizumab treatment (NCT05144087, GSK funded, investigator led study). Fourteen (70%) paired biopsies were analysed using immunohistochemistry (IHC) and movat's pentachrome staining. Clinical and lung function data were also collected.

**Results** At T6, there was a significant reduction in exacerbations (median AER: T0 5 [IQR 4–7], T6 0 [0,0]) and significant improvement in asthma control (ACQ: T0 2.8 [IQR 2.1,3.1], T6 1.0 [0.3,2.1]) and lung function (FEV1/FVC: T0 0.60 [IQR 0.57, 0.66], T6 0.64 [IQR 0.58,0.75]). In bronchial biopsies, submucosal eosinophil counts were significantly reduced ($P \leq 0.05$) along with markers of remodelling including extracellular matrix deposition of collagen and elastin (both $P \leq 0.001$, by movat's pentachrome) and collagen I ($P \leq 0.05$, by IHC). Basement membrane thickness was also significantly reduced (mean T0 6.6 mm [0.3], T6 5.7 mm [1.1], p=0.04). Finally, in the bronchial epithelium there was a reduction in goblet cells ($P \leq 0.01$) and a significant increase in e-cadherin (a component of adherens junctions) ($P \leq 0.0001$).

**Conclusions** In patients with T2 high SA, mepolizumab treatment significantly reduced airway inflammation and structural markers of airway remodelling including submucosal eosinophils, extracellular matrix components and basement membrane thickness. Notably, our study is the first to demonstrate improved epithelial barrier integrity with biologic therapy in T2 high SA, as evidenced by increased e-cadherin expression. These findings suggest that, beyond clinical improvements, mepolizumab reverses key features of airway remodelling in patients with T2 high SA.

## T6 GENETIC CHARACTERISATION OF PSEUDOMONAS AERUGINOSA ISOLATES FROM BRONCHIECTASIS PATIENTS IN A RANDOMIZED, DOUBLE-BLIND PLACEBO-CONTROLLED TRIAL OF A BISPECIFIC MONOCLONAL ANTIBODY TARGETING PSL AND PCRV (GREAT-2)

[1]RC Hull, [1]MB Long, [1]A Gilmour, [1]I Petrou, [1]R Galloway, [1]K Viligorska, [1]H Richardson, [1]JM New, [1]C Hennayake, [1]E Johnson, [1]E McIntosh, [1]Z Eke, [2]M Band, [2]H Bandara, [2]G Martin, [3]S Aliberti, [4]M Vendrell, [5]E Polverino, [6]G Suarez-Cuartin, [7]MR Loebinger, [8]M Tunney, [9]K Cartwright, [9]R Hughes, [10]A DiGiandomenico, [11]MG Belvisi, [11]W Brailsford, [12]C Haworth, [13]O Sibila, [1]J Stobo, [1]JD Chalmers. *[1]Division of Respiratory Medicine and Gastroenterology, University of Dundee, Dundee, UK*; *[2]Tayside Clinical Trials Unit, University of Dundee, Dundee, UK*; *[3]Department of Biomedical Sciences, Humanitas University, Milano, Italy*; *[4]Department of Pulmonology, Dr. Trueta University Hospital, Girona Biomedical Research Institute, University of Girona, Girona, Spain*; *[5]Pneumology Department, Hospital Universitari Vall d'Hebron, Barcelona, Spain*; *[6]Pneumology Department, Hospital Universitari de Bellvitge, Hospitalet de Llobregat, Barcelona, Spain*; *[7]Royal Brompton Hospital, Guy's and St. Thomas' NHS Foundation Trust, London, UK*; *[8]School of Pharmacy, Queen's University Belfast, Belfast, UK*; *[9]Respiratory and Immunology, AstraZeneca, Cambridge, UK*; *[10]Vaccine and Immune Therapies, AstraZeneca, Gaithersburg, USA*; *[11]Respiratory and Immunology, AstraZeneca, Gothenburg, Sweden*; *[12]Cambridge Centre for Lung Infection, Royal Papworth Hospital NHS Foundation Trust, Cambridge, UK*; *[13]Pneumology Service, Hospital Clinic, Barcelona, Spain*

10.1136/thorax-2025-BTSabstracts.6

**Introduction** *Pseudomonas aeruginosa* (PA) airway infections in bronchiectasis are associated with increased disease severity and exacerbations. New treatment options are needed due to inconsistent results with inhaled antibiotics and increasing concerns over antimicrobial resistance. The GREAT-2 trial of gremubamab, a bispecific monoclonal antibody targeting PA Psl and PcrV, demonstrated reduced bacterial load at the end-of-treatment (EoT) (500 mg), and significantly prolonged time-to-first exacerbation (1500 mg) in patients chronically infected with PA. Whether PA adapts to evade monoclonal antibody treatment is unknown. This study aimed to characterise PA genetic variation in patients receiving treatment with gremubamab.

**Methods** 37 patients were enrolled in the multinational (UK and Spain), randomized, double-blind, placebo-controlled trial of gremubamab for people with CT-confirmed bronchiectasis and chronic PA infection, randomised 1:1:1 to 1500 mg or 500 mg gremubamab, or placebo, once every four weeks for 12 weeks. 233 PA clones were isolated from sputum on days 1, 7, 14, 28, 56, 84 (EoT), and day 168 (12-week follow-up), with all clone morphologies stored. Whole-genome Illumina sequencing was followed by analysis of phylogenetic distances, sequence types, SNPs, and deletions relative to PAO1/baseline clone.

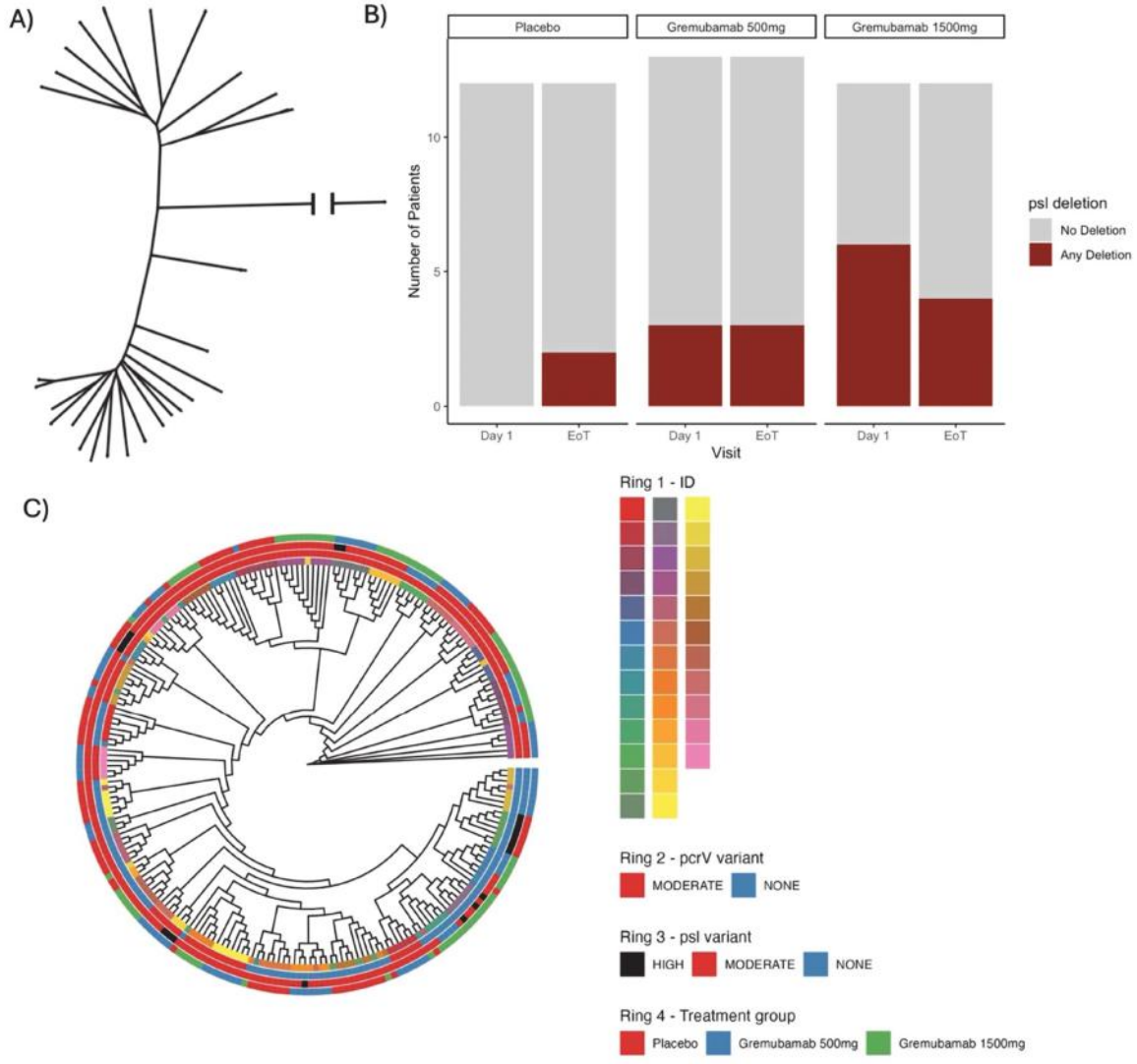
**Results** The 233 PA clones are genetically diverse, comparable to global collections of bronchiectasis isolates. In a rooted phylogenetic tree, 163 patient-specific clades with $\geq 3$ tips were identified (figure 1A). 8 clones, from 2 patients in the placebo group, had a *pcrV* deletion. There was no significant difference in the frequency of *psl* deletions between treatment

groups over the trial (placebo p=0.48, 500 mg p=NA, 1500 mg p= 0.62, figure 1B). When mapping variants to patient's baseline clone, SNPs were not significantly changed between treatment groups.

There were no significant changes in phylogenetic distance interactions between visit number and treatment group over the trial ($\chi^2(12) = 13.9$, p=0.307), suggesting little evolutionary adaptation (figure 1C).

In all 233 clones, 23 unique sequence types were identified, plus novel sequence types in 8 patients. 25/37 patients had the same sequence type at all visits.

**Conclusion** PA isolates showed stability through the 6-month treatment period in the GREAT-2 trial. There were no changes or adaptations in the PcrV and Psl targets, suggesting no resistance development.




**Abstract T6 Figure 1**

# Emerging clinical trial data

## S1 RANDOMISED, PLACEBO-CONTROLLED TRIAL OF ASTEGOLIMAB FOR COPD WITH FREQUENT EXACERBATIONS: ALIENTO

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10.1136/thorax-2025-BTSabstracts.7

**Rationale** The randomised, double-blind, placebo-controlled Phase IIb ALIENTO trial (NCT05037929) evaluated efficacy and safety of astegolimab (anti-ST2) in COPD.

**Methods** Participants (current/former smokers, aged 40–90 years, history of frequent exacerbations) were randomised 1:1:1 to astegolimab SC 476 mg Q2W or Q4W, or placebo, for 52 weeks. Efficacy and safety endpoints were evaluated.

**Results** 1301 participants were treated (Q2W, n=433; Q4W, n=437; placebo, n=431; 79% on ICS/LABA/LAMA at baseline). Annualised rate of moderate/severe COPD exacerbations (primary endpoint) was significantly lower with astegolimab Q2W vs placebo (adjusted rate reduction 15%; $P$=0.0494), but not Q4W vs placebo (figure 1). There were numerical decreases with astegolimab Q2W in moderate/severe COPD exacerbation rate in participants with baseline EOS <300 cells/μL (RR 0.87 [95% CI 0.73, 1.04]; $P$=0.1210), severe exacerbation rate (RR 0.71 [95% CI 0.49, 1.05]; $P$=0.0883), and change from baseline to Week 52 in SGRQ-C (difference in adjusted mean –1.87 [95% CI –4.01, 0.26]; $P$=0.0857). No differences were seen in other secondary endpoints (Week 52). Adverse events were balanced between treatment arms.

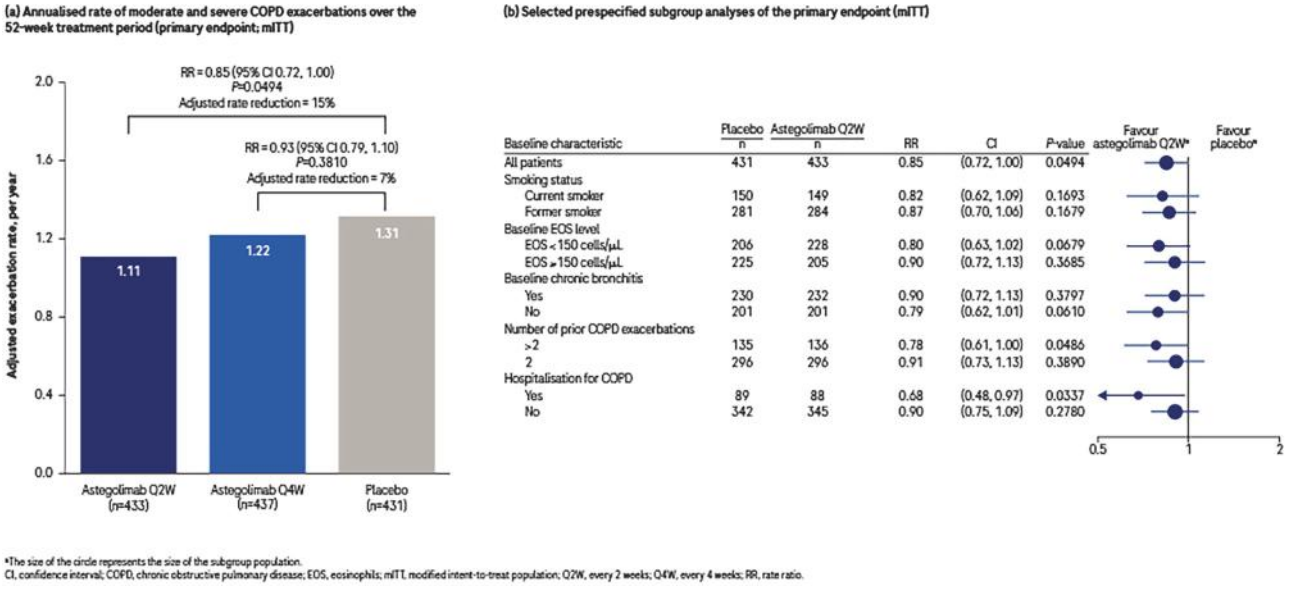
**Conclusions** Astegolimab Q2W decreased annualised COPD exacerbation rate by 15% ($P$=0.0494) vs placebo in the overall ALIENTO population (irrespective of smoking status or EOS level), with trends for improvement in some other clinical outcomes.

## S2 WHAT IS THE CLINICAL EFFECTIVENESS AND COST-EFFECTIVENESS OF NEBULISED 7% SALINE IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)- (MUCACT COPD TRIAL)

[1]AT Hill, [2]S Lewis, [2]S Tuck, [2,3]A Skeikh, [2]A Stoddart, [3,4]M Gibbons, [5]L Gibbons, [6]F Harris, [7]H Bayes, [8]M Patel, [8]A Smith, [9]L Murray, [10]D Saralaya, [11]J Suntharalingam, [12]A de Soyza, [13]A Turner, [14]A Usher, [13]D Dosanjh, [13]A Dev Vellore, [15]M Crooks, [16]L Linkson, [8]P Bedi, [17]S Finch, [18]R Mustafa, [19]T Nagarajan, [20]K Ibrahim, [21]D Miller, [22]R Aul, [7]D Anderson, [2]E Ward, [2]B Selby, [2]K Oatey, [21]J Miller, [15]S Hart, [23]J Wildgoose, [24]S Ramalingam, [2]S Donaldson, [2]J Crowe, [24]A Clarke, [25]R Milroy, [26]P Hodkinson, [24]R Frangulyan. *[1]The University of Edinburgh Centre for Inflammation Research, Edinburgh, UK; [2]The University of Edinburgh, Edinburgh, UK; [3]Academic Department of Respiratory Medicine, Royal Devon University Healthcare Foundation NHS Trust, Exeter, UK; [4]NIHR Exeter Biomedical Research Centre, University of Exeter, UK; [5]NIHR Clinical Research Network, South West Peninsula, UK; [6]University of Stirling, Stirling, UK; [7]NHS Greater Glasgow and Clyde, UK; [8]NHS Lanarkshire, UK; [9]NHS Highland, UK; [10]Bradford Teaching Hospitals NHS Foundation Trust, Bradford, UK; [11]Royal United Hospitals Bath NHS Foundation Trust, Bath, UK; [12]The Newcastle upon Tyne Hospitals NHS Foundation Trust, Newcastle, UK; [13]University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK; [14]Gloucestershire Hospitals NHS Foundation Trust, Gloucestershire, UK; [15]Hull University Teaching Hospitals NHS Trust, Hull, UK; [16]King's College Hospital NHS Foundation Trust, London, UK; [17]NHS Fife, Kirkcaldy, UK; [18]South Tees Hospitals NHS Foundation Trust, Middlesborough, UK; [19]NHS East Cheshire NHS Trust, Macclesfield, UK; [20]Walsall Healthcare NHS Trust, Walsall, UK; [21]Aberdeen Royal Infirmary, Aberdeen, UK; [22]St George's University Hospitals NHS Foundation Trust, London, UK; [23]PPI Member, UK; [24]NHS Lothian, UK; [25]Retired, UK; [26]University Hospital Crosshouse, UK*

10.1136/thorax-2025-BTSabstracts.8

This was an open pragmatic UK multi-centre parallel RCT in participants with COPD that have either chronic bronchitis




**Abstract S1 Figure 1**

and/or associated bronchiectasis that have self-reported difficulty self-expectorating. Participants were randomised to receive either nebulised 7% saline plus active cycle breathing technique (ACBT) twice daily (intervention arm) or 750 mg carbocisteine plus ACBT twice daily for 1 year (control arm).

The Primary objective was to assess whether the intervention arm was superior compared with the controlled arm when comparing the change in COPD Assessment Test (CAT) over 1 y.

Secondary objectives: health related quality of life; exacerbations; time to first exacerbation; lung function; health economic benefit; safety.

The aim was to recruit 860 participants. Randomisation was on a 1:1 basis and included stratification for current smoking status and COPD severity (GOLD class 1 and 2 versus 3 and 4).

Due to difficulty in recruitment post COVID, the funding was stopped after 206 participants completed the study. 104 participants were in the control arm and 102 participants in the intervention arm.

The demographics were well matched between groups. There was no significant difference in the improvement of CAT scores at 1 y comparing the control and intervention arms from baseline (control arm n=56, -2.5 (-7.5 to 1.5), intervention arm n=50, -1 (-6 to 3), p=0.37).

There was no significant difference in quality-of-life questionnaires, lung function tests, or serious adverse events at 1 y.

In the intervention arm there were fewer exacerbations than the control arm (p=0.017) (in the intervention arm 70% had no exacerbations and in the control arm 56% had no exacerbations); hazard ratio was 0.628 (95% CI 0.399–0.988, p= 0.044). There was a trend to fewer upper respiratory tract infections assessed using the WURSS24 questionnaire in the intervention arm (83% no upper respiratory tract infections) compared with the control arm (72%, p=0.053). There was a prolonged time to first exacerbation requiring antibiotic therapy and/or systemic steroids (p=0.044) in the intervention arm.

**Conclusion** Although hypertonic saline compared with carbocisteine treatment led to no significant difference in the CAT questionnaire, there was a prolonged time to first exacerbation and reduced exacerbations.

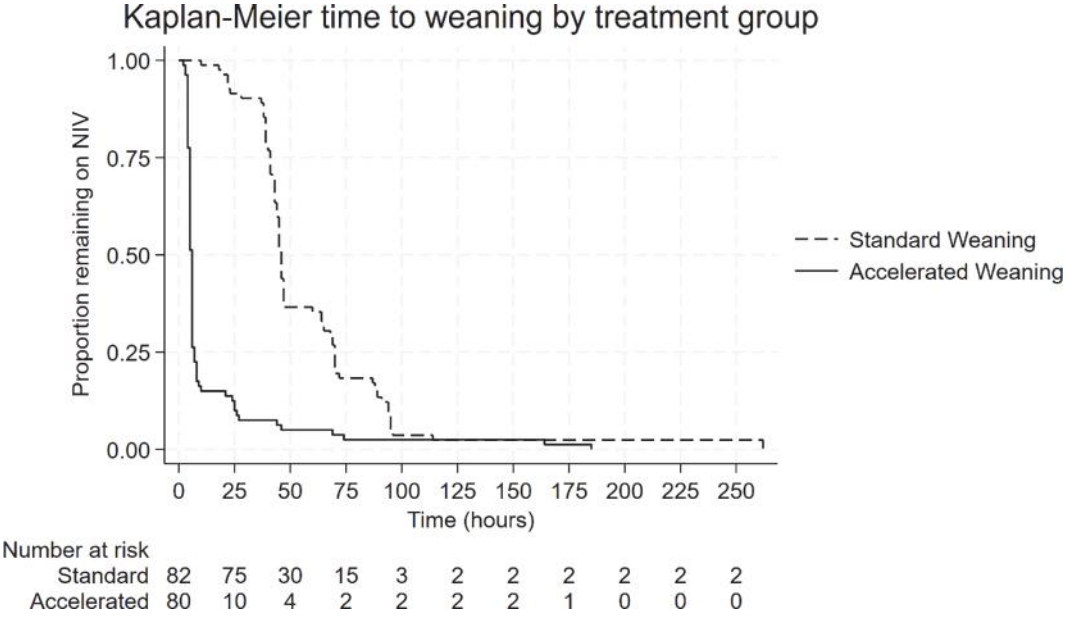
## S3 STANDARD VERSUS ACCELERATED WEANING FROM NON-INVASIVE VENTILATION (NIV) IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE DIRECTED BY THE NIV OUTCOMES SCORE: A RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2025-BTSabstracts.9

**Introduction** Acute NIV for exacerbation of chronic obstructive pulmonary disease (ECOPD) is one of the most effective interventions in emergency medicine, but it is intrusive and complications are common. NIV weaning guidance is limited and inconsistently followed. Our 10-centre NIV Outcomes study[1] showed marked variation in duration of NIV, relapse (respiratory acidaemia requiring NIV more than 48 hours after successful weaning), inpatient and 90-day mortality.

**Methods** We conducted a 7-centre, open-label, parallel, randomised controlled trial with embedded economic evaluation to compare time to successful weaning from NIV in ECOPD. Patients meeting a combination of clinical risk stratification (low or medium risk NIVO score) and objective weaning criteria were randomised to standard stepwise (SW) or accelerated (AW) weaning protocols (ISRCTN:64639614). SW




**Abstract S3 Figure 1** Kaplan-Meier plot for time to successful weaning from NIV in patients randomised to standard and accelerated weaning

reflected current BTS guidance. AW involved a 4-hour trial off NIV and arterial blood gas to exclude recurrent respiratory acidaemia. Not replacing NIV within 48 hours was considered successful weaning.

**Results** Between October 2022 and April 2025, participants were randomised to SW (n=83) or AW (n=81): medium risk NIVO score= 61 (37.2%); female= 99 (60.4%); mean (SD) age= 68.6 (8.7); FEV1% predicted= 39.3 (15.2); duration of NIV prior to meeting weaning criteria= 37.8 (13.7) hours. Both groups were well matched. Median (IQR) hours to successful weaning was: SW= 46 hours (41–70), AW= 6 (5–7), p<0.0001. Competing risks precluding weaning, death during weaning (n=0) and failure to wean requiring transition to home NIV (n=2), were similar between groups.

There was no difference in relapse (SW n=5, AW n=3), median (IQR) length of hospital stay after meeting weaning criteria (SW n=5 (3–7), AW n= 5 (2–8) days), one or more readmissions within 30 days of discharge (SW n=20, AW n=14), in-hospital mortality (SW n=2, AW n=3), or 90-day mortality (SW n=10, AW n=8).

**Conclusions** In patients requiring NIV for ECOPD with a low or medium risk NIVO score and meeting objective weaning criteria, NIV was successfully removed 40 hours earlier using an accelerated weaning protocol with no increase in mortality or readmission. This releases NHS resources and may reduce complications for patients. Further trial analysis will be available for the winter BTS.

## REFERENCE

1. Hartley T, Lane ND, Steer J, *et al*. The noninvasive ventilation outcomes (NIVO) score: prediction of in-hospital mortality in exacerbations of COPD requiring assisted ventilation. *Eur Respir J.* 2021;**58**(2).

## S4 EFFECTS OF HYPEROXIA ON PULMONARY INFLAMMATION AND ORGAN INJURY IN A HUMAN *IN VIVO* MODEL (HIPI): A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

[1,2]DA Linden, [1]D Dorrian, [1]M Glenn, [3]K Welzenbach, [3]MJ Rowland, [3]M Yang, [3]L Klein, [3]E Randazzo, [3]M Sindelar, [3]J Schlotterbeck, [3]A Cattini, [1]S Tandel, [4]A Dushianthan, [1]MC McKelvey, [2]M Bailey, [1]J Conlon, [2]D Moore, [5]S Carr, [5]JM Bradley, [1]D Simpson, [1]CC Taggart, [2]JC Kidney, [1]CM O'Kane, [1,6]DF McAuley. *[1]Wellcome-Wolfson Institute for Experimental Medicine, School of Medicine, Dentistry and Biomedical Sciences, Queen's University Belfast, UK; [2]Mater Hospital Belfast, Belfast Health and Social Care Trust, Belfast, UK; [3]Novartis Biomedical research, CH-4002, Basel, Switzerland; [4]NIHR Biomedical Research Centre, University Hospital Southampton NHS Foundation Trust, Southampton, UK; [5]Wellcome-Wolfson Institute for Experimental Medicine, Northern Ireland Clinical Research Facility, Queen's University Belfast, UK; [6]Regional Intensive Care Unit, Royal Victoria Hospital, Belfast Health and Social Care Trust, Belfast, UK*

10.1136/thorax-2025-BTSabstracts.10

**Rationale** Hyperoxia may be associated with adverse ICU outcomes but despite numerous large-scale randomised clinical trials (RCTs) there is ongoing lack of consensus regarding optimal oxygenation strategies. Importantly, detailed mechanistic understanding of the potential pro-inflammatory effects of hyperoxia remain to be defined.

**Objective** To test the hypothesis that hyperoxia will modify pulmonary and systemic inflammation and cellular injury in the setting of inhaled lipopolysaccharide (LPS) challenge.

**Methods** In this single-centre, randomised, double-blind, allocation-concealed, placebo-controlled trial (NCT05414370), healthy non-smoking adult volunteers (<45 years of age) were randomised (1:1) to receive hyperoxia administered via high-flow nasal oxygen delivery system ($FiO_2$ 1.0, 60L/min flow rate) or synthetic medical air for a duration of 6-hours. Bronchoalveolar lavage (BAL) was performed at 6-hours following inhalation of 50µg of LPS. The primary outcome measure was BAL IL-8. Secondary outcome measures included markers of pulmonary and systemic inflammation, as well as epithelial cell injury. Untargeted metabolomic and lipidomic profiling of BAL fluid and plasma was conducted using liquid chromatography-tandem mass spectrometry to investigate oxidative stress. Transcriptomic analysis was performed on whole blood.

**Results** Between January 2023 and March 2024, a total of 22 participants were recruited and completed all study procedures. Exposure to hyperoxia for a duration of 6-hours did not significantly increase the pre-specified primary outcome measure BAL IL-8 (hyperoxia 375.7 pg/ml; placebo 331.9 pg/ml; p=0.33). There were no between-group differences in BAL cytokines or plasma markers of systemic inflammation. However, untargeted metabolomics analysis found hyperoxia was associated with differentially altered BAL metabolites including upregulation of reduced glutathione (p=0.02), glycerophosphoglycerol (p=0.04) and downregulation of cysteinylglycine disulfide (p=0.04). Furthermore, hyperoxia induced distinct transcriptional response at 24h with 247 DEGs (p<0.05). Enrichment analysis indicated activation of platelet-related pathways and ECM-receptor interactions.

**Conclusions** In a human model of pulmonary inflammation induced by inhaled lipopolysaccharide, 6 hours of hyperoxia does not accentuate the innate pulmonary or systemic inflammatory response nor cause overt tissue damage.

## S5 HAP-FAST: A PILOT SEQUENTIAL MULTIPLE ASSIGNMENT RANDOMISED TRIAL (SMART) COMPARING CHEST X-RAY TO LOW-DOSE CT SCAN AND EMPIRICAL ANTIBIOTICS TO ANTIBIOTICS GUIDED BY THE BIOFIRE® FILM ARRAY® PNEUMONIA PLUS PANEL IN ADULTS WITH SUSPECTED NON-VENTILATOR-ASSOCIATED HOSPITAL-ACQUIRED PNEUMONIA

[1,2]D Wootton, [3]I Powell, [3]S Willshaw, [3]A Tao, [2]N Shafiqa, [4]E Adegbenro, [5]K Cook, [6]A Cross, [4]D Mclaughlan, [7]H Singh, [7]K Kalapurakal, [7]N Senior, [1,8]L Turtle, [8,9]S Aston, [9,10]AL Howard, [11]L Pilling, [12]R Bazaz, [13]S Trewick, [14]I Poku, [14]A Alvarez-Nishio, [14]E Lam, [1]S Abrams, [1]A Collins, [15]F Sherratt, [15]B Young, [3]A Jones. *[1]Department of Clinical Infection Microbiology and Immunology, University of Liverpool, Liverpool, UK; [2]Department of Respiratory Medicine, Aintree University Hospital, Liverpool, UK; University Hospitals of Liverpool Group, Liverpool, UK; [3]Liverpool Clinical Trials Centre, University of Liverpool, Liverpool, UK; [4]NHS University Hospitals of Liverpool Group, Liverpool, UK; [5]The Emergency Department, NHS University Hospitals of Liverpool Group, Liverpool, UK; [6]Institute of Life course and Medical Sciences, University of Liverpool, Liverpool, UK; [7]Lancashire Teaching Hospitals NHS Foundation Trust, Preston, UK; [8]Tropical and Infectious Diseases Unit, Royal Liverpool Hospital, NHS University Hospitals of Liverpool Group, Liverpool, UK; [9]Department of Pharmacology and Therapeutics, Institute of Systems, Molecular and Integrative Biology, University of Liverpool, Liverpool, UK; [10]Department of Microbiology, Royal Liverpool Hospital, NHS University Hospitals of Liverpool Group, Liverpool, UK; [11]Department of Respiratory Medicine, Royal Liverpool Hospital, NHS University Hospitals of Liverpool Group, Liverpool, UK; [12]Department of Infectious Diseases, Wythenshawe Hospital, Manchester University NHS Foundation Trust, Manchester, UK; [13]Department of Respiratory Medicine, Wythenshawe Hospital, Manchester University NHS Foundation Trust, Manchester, UK; [14]Independent PPI advisor, UK; [15]Institute of Population Health, University of Liverpool, Liverpool, UK*

10.1136/thorax-2025-BTSabstracts.11

**Background** HAP is treated with broad spectrum antibiotics but a third of patients have no radiological evidence of a lung infiltrate and most are treated empirically. Therefore,

opportunities exist to enhance antibiotic stewardship by improving the radiological and microbiological diagnosis of HAP.

**Aims**

1. Investigate the feasibility of a fully powered diagnostic trial in suspected HAP comparing: a) low-dose thoracic CT scan to chest x-ray b) BIOFIRE® FILMARRAY® Pneumonia Panel (FAPP) to standard microbiology
2. Evaluate trial endpoints and look for signals of efficacy

**Methods**

- Funding: UK National Institute of Health and Care Research (NIHR300669)
- NHS combined Research Ethics/Health Research Authority approval: 22/WA/0315
- Trial registration: www.ClinicalTrials.gov/study/NCT05483309

Using a deferred consent model, clinicians at 4 hospitals randomised patients 1:1 to the interventions via a trial website. The research team later sought consent for ongoing participation. Stage one randomisation was between a routine chest x-ray and a low-dose, non-contrast enhanced thoracic CT scan. Then, patients requiring ongoing antibiotics who could produce a sputum sample were randomised for a second time between usual care (empirical antibiotics and standard microbiology) versus antibiotics informed by BIOFIRE® FILM ARRAY® pneumonia *plus* panel analysis of their sputum. Antibiotic prescribing in the FAPP arm was supported by a guideline. Participants were visited for 10 days or until discharge, at 28 and 90 days. Outcomes evaluated were mortality, clinical cure, quality of life, length of stay and antibiotic use.

**Results** Recruitment (n=220) was completed ahead of schedule with a mean of 6 randomisations/site/month. Characteristics were similar between groups with a median age of 77 y (range 20–99). Of those randomised at stage 1 (CT v CXR) 28% were randomised at stage 2 (FAPP v standard micro). 65% of patients gave consent for ongoing participation. Median time to CT was 2 hours. Group differences were seen in antibiotic prescribing, mortality and several other outcome measures.

**Conclusion** A multicentre trial of novel diagnostic pathways for patients suspected of HAP is feasible and there is evidence to support a fully powered trial of these interventions.

## S6 CONSERVATIVE MANAGEMENT IN TRAUMATIC PNEUMOTHORACES IN THE EMERGENCY DEPARTMENT (COMITED): A RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2025-BTSabstracts.12

**Introduction** Current guidelines advise chest drain insertion for most traumatic pneumothoraces, although very small pneumothoraces can be managed with observation at the treating clinician's discretion. There remains a large proportion of patients in whom there is clinical uncertainty as to whether an immediate chest drain is required, with no robust evidence to inform practice. Chest drains carry a high risk of complications such as bleeding and infection. The default to invasive treatment may be causing potentially avoidable pain, distress and complications. We evaluated the clinical and cost-effectiveness of an initial conservative approach to the management of patients with traumatic pneumothoraces.

**Methods** The CoMiTED (Conservative Management in Traumatic Pneumothoraces in the Emergency Department) trial was a multicentre, pragmatic parallel group, individually randomised controlled noninferiority trial to establish whether initial conservative management of significant traumatic pneumothoraces was non-inferior to invasive management in terms of subsequent emergency pleural interventions, complications, pain, breathlessness and quality of life. Adults with traumatic pneumothoraces, in whom treating clinician(s) believed either a chest drain or conservative management was a suitable initial treatment option were eligible for inclusion. The primary outcome was a binary measure of the need for one or more subsequent emergency pleural interventions within 30 days of randomisation (excluding the initial chest drain in that arm).

**Results** Among 400 patients randomised from 41 UK National Health Service hospitals between Aug 2022-Jan 2025, 394 were included in the primary analysis. Randomised patients were predominantly male (74%), with a pneumothorax size of 2–5 cm (57%), with 33% having a pneumothorax >5 cm. Participants were predominantly spontaneously ventilating at the point of randomisation (89%) and had blunt chest wall trauma (92%).

In the conservatively managed group, 49/199 (25%) met the primary outcome, and 15/195 (8%) in the chest drain arm; Risk Difference 0.17 (95% CI 0.10–0.24). However, 112/199 56% of conservatively managed patients required no pleural intervention within 30 days.

**Conclusions** An initial conservative approach to traumatic pneumothorax management is not non-inferior to initial drainage in terms of the need for subsequent emergency pleural intervention. Full results on clinical effectiveness and implications for practice will be discussed.

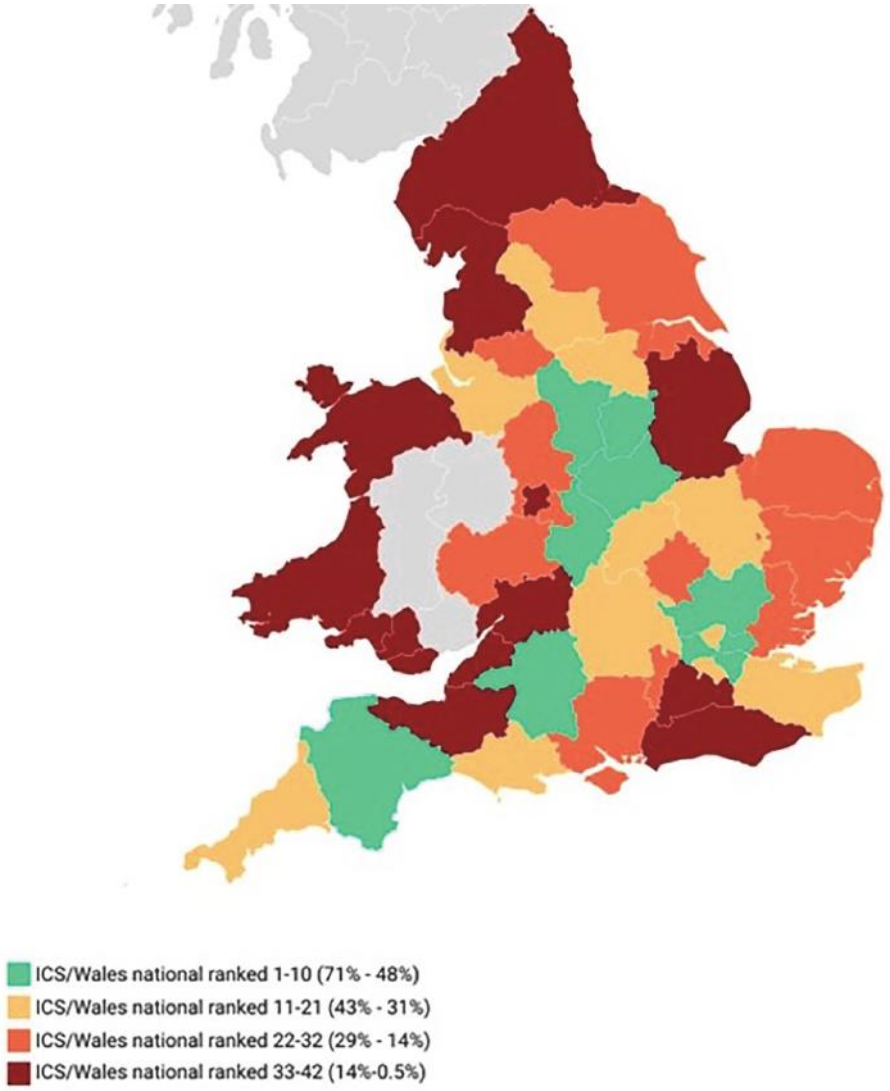
# Addressing inequalities in COPD

## S7 NATIONAL RESPIRATORY AUDIT PROGRAMME (NRAP) HIGHLIGHTS VARIATION IN DELIVERY OF DISCHARGE BUNDLES OF EVIDENCE-BASED INTERVENTIONS IN PATIENTS ADMITTED TO HOSPITAL WITH COPD EXACERBATION

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10.1136/thorax-2025-BTSabstracts.13

**Introduction** Discharge bundles consist of a short list of evidence-based practices to be implemented prior to hospital discharge to optimise patient outcomes, address high COPD readmission rates and ensure consistent provision of high-quality care. However, despite the availability of evidence-based




**Abstract S7 Figure 1** Variation in completion of the discharge bundle elements across England and Wales with integrated care systems (ICS) ranked by % of completed of elements.
**Note**: Wales is shown as a national total rather than broken down by health board due to the small numbers associated with discharge bundle completion. Map created by NRAP team with Datawrapper.

interventions, discharge bundles have been implemented with varying success.

**Methods** The National Respiratory Audit programme (NRAP) is a continuous national audit of secondary care across England and Wales. Data from the 2023–2024 NRAP audit on COPD admissions were evaluated, and patients were included in this analysis if they were admitted with an acute COPD exacerbation and were alive on discharge (n=62,790). All elements of the discharge bundle were assessed for completion, and data were linked to demographic factors and compared nationally.

**Results** The NRAP data report that delivery of the discharge bundle is a key performance indicator (KPI) in the COPD workstream and must be completed in 60% of patients admitted with an exacerbation of COPD to achieve the best practice tariff in England. However, only 28% of admissions nationally receive all elements of the discharge bundle. Performance across 171 hospitals ranges from 0–100%, with a median of 14.5% (IQR 1.3–42.8%). The audit data highlight a discrepancy between reporting delivery of the discharge bundle as a whole and confirmation of the delivery of each individual element. Assessment for pulmonary rehabilitation (PR), one of the most evidence-based interventions in COPD, was completed in only 57.8% (IQR 25.0–80.0). Completion of each discharge bundle element varies nationally, with a trend towards Integrated Care Systems (ICSs) in areas of higher deprivation reporting lower percentages of patients receiving fully completed discharge bundles (figure 1).

**Conclusion** Despite evidence supporting the use of discharge bundles in the care of patients with acute COPD exacerbations admitted to hospital, delivery of these evidence-based interventions remains variable, particularly in areas of high deprivation. Our data mirror findings from the recent *Asthma + Lung UK ICS Respiratory Review 2024/2025*,[1] which highlights the same regions as having some of the highest rates of respiratory admissions and deaths.

## REFERENCE

1. Asthma + Lung UK (2024/2025). ICS Respiratory Review. Retrieved 10th March 2025, http://www.asthmaandlungUK.org.uk/healthcare-professionals/ics-respiratory-review

## S8 ADDRESSING INEQUALITIES IN RESPIRATORY EMERGENCIES THROUGH DATA-DRIVEN INTERVENTIONS

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10.1136/thorax-2025-BTSabstracts.14

**Introduction and Objectives** Ambulance services are often the first point of contact for patients in respiratory distress, disproportionately affecting the most deprived communities. Leveraging this rich and largely untapped reservoir of data offers a powerful opportunity to inform targeted interventions, optimise care pathways, and ultimately improve outcomes for this high-risk population.

This study analysed ambulance service data from 2024 to examine trends in calls related to breathing problems and deprivation.

**Methods** This exploratory retrospective study analysed 83,800 calls categorised as 'breathing difficulties' using Advanced Medical Priority Dispatch System (AMPDS) triage. Additional analysis using integrated care datasets was conducted to understand frequent Emergency Department (ED) attendances and outcomes.

**Results**

- Calls for breathing difficulties increased 78% since 2021, with clear winter spikes, particularly in the most deprived areas.
- 72% of all calls for breathing problems were conveyed to hospital. Under 18s were more likely to be conveyed than adults (82% compared to 71%) and under 5s were most likely to be conveyed (89%), although national guidance may bias crew behaviours.
- Analysis of the data through a health inequalities lens revealed that 41% of calls came from the most deprived quintile, compared to 9% in the least deprived.
- There is significant variation in the rate of calls by local authority. This correlates with index of multiple deprivation scores.
- ED data showed high rates of low-acuity outcomes. Individual patient-level data analysis revealed an increase in ambulance usage over time indicating the potential to work more collaboratively to deliver more appropriate, preventative care for these individuals.

**Conclusion** This study highlights the intersection of respiratory health, deprivation, and urgent care. Better leveraging of ambulance service data to pilot new models of care could realise improved outcomes for patients with breathing difficulties.

A separate study has been conducted evaluating the role of ambulance service specialist practitioners in urgent care in managing category 2 calls for breathing difficulties. Findings aim to inform more equitable and effective care for patients with high-acuity respiratory presentations.

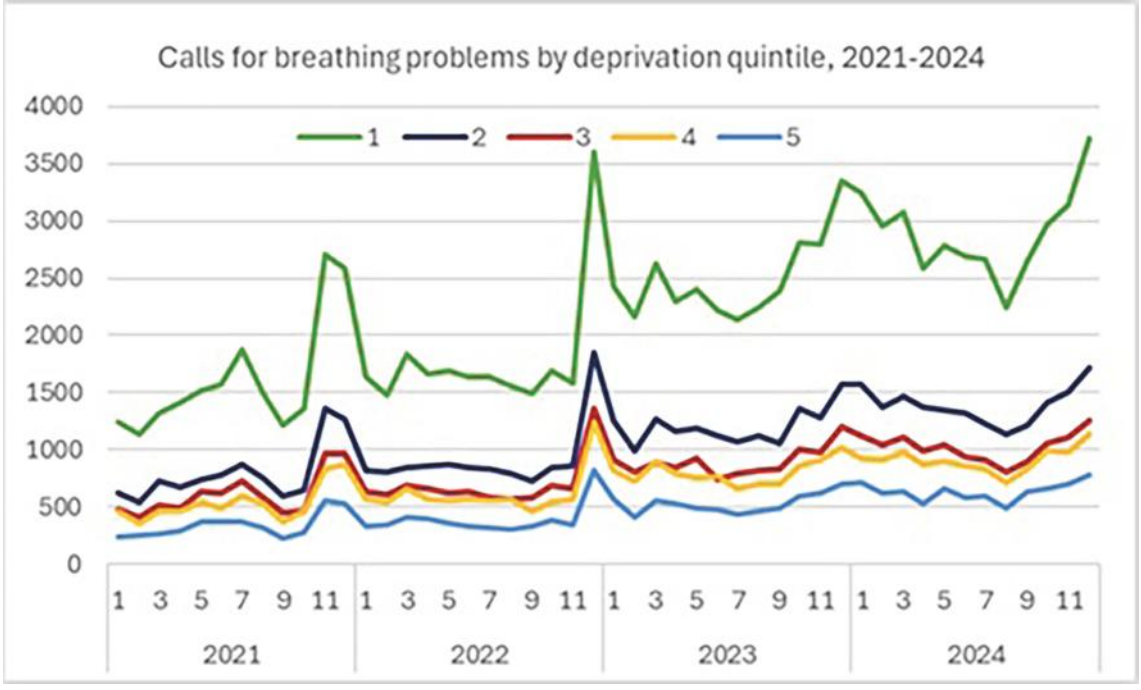
## S9 A MATTER OF LIFE AND BREATH: UNDERSTANDING THE NEEDS OF PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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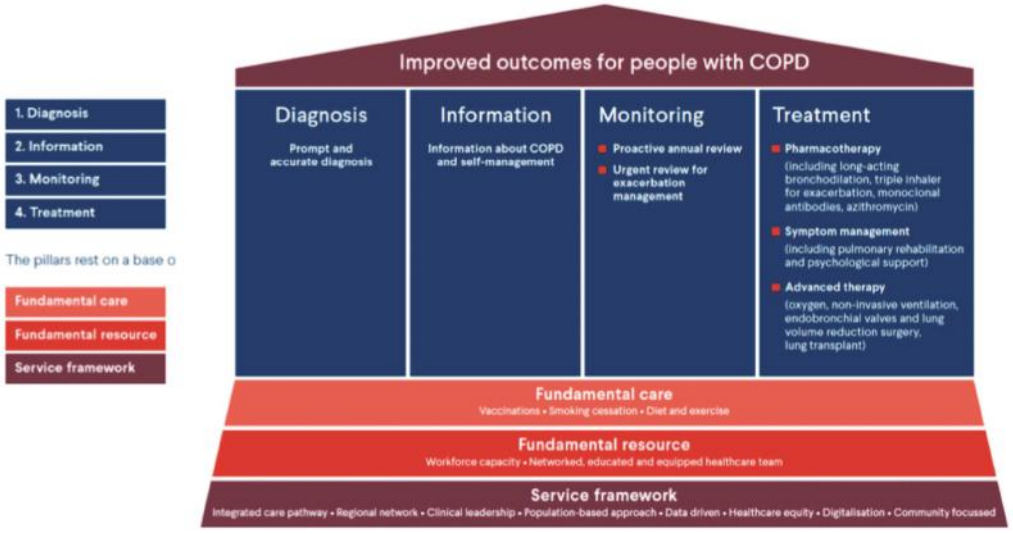
10.1136/thorax-2025-BTSabstracts.15

**Context** 1 in 10 adults over 40 live with COPD costing the NHS over £2billion annually making this a pressing public health challenge. Despite medical advancements, many people face delayed diagnoses, inconsistent care and avoidable hospital admissions.

**Methods** We undertook the largest qualitative analysis of lived experience in people with COPD (PwCOPD) and their carers in England, via focus groups, or one-to-one




**Abstract S8 Figure 1**




**Abstract S9 Figure 1**

interviews, held in local settings (e.g., village halls, seminar rooms and places of religious importance). Questions were around the themes of COPD management, including symptoms, diagnosis and follow-up care. Each interview took approximately 1 hour. PwCOPD were recruited from primary and secondary care and all meetings were recorded, transcribed and analysed.

**Results** 120 people (71 women) from 7 NHS regions participated, including 35 carers. The majority (73%) were over the age of 65. Almost 1 in 3 PwCOPD were widowed. PwCOPD were most likely to be managed by their GP alone (57%). The majority of PwCOPD had had an exacerbation in the last 12 months (65%) and 16% reported being hospitalised with a severe exacerbation on more than 1 occasion.

A consistent picture emerged including gaps in care, poor communication between services and an urgent need for a more integrated, patient-centred approach to reduce unwarranted variation and improve the quality of services and support. PwCOPD expressed a strong interest to hear and take part in clinical research, but almost no-one had ever been offered this. The patient voices described:

- delays in diagnosis and a lack of information about COPD, with a diagnosis occurring after many years of symptoms and illness.
- difficulties accessing essential services including GP appointments, specialist care and pulmonary rehabilitation.
- a lack of support for carers, the majority of whom had little information to access financial support.

**Conclusion** This is a powerful insight into the lived experience of PwCOPD. We produce nine recommendations which map to the three shifts in care proposed in the NHS 10-year plan, refined into four fundamental pillars of COPD care: diagnosis, information, monitoring and treatment, which provide a roadmap for meaningful changes in services (figure 1).

## S10 CARE CLOSER TO HOME: THE ROLE OF AMBULANCE SERVICE SPECIALIST PRACTITIONERS IN URGENT CARE IN MANAGING CATEGORY 2 BREATHING DIFFICULTIES

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10.1136/thorax-2025-BTSabstracts.16

**Introduction and Objectives** Respiratory conditions drive significant demand for ambulance services, disproportionately affecting the most deprived communities.

This study examines the impact of targeted dispatch of Specialist Paramedics and Nurses in Urgent Care (SP/NUCs) on patient conveyance rates. SP/NUCs possess enhanced skillsets facilitating care closer to home for medically complex patients, enhancing patient experience and aligned to national strategy. Outcomes were compared against those of standard double-crewed ambulances (DCA)

**Methods** This exploratory retrospective study analysed data between 22/7/24 to 24/22/24 and 25/11/25 to 31/3/25 from ambulance service computer aided despatch (CAD) data. Included calls were categorised as 'breathing difficulties' using Advanced Medical Priority Dispatch System (AMPDS) triage.

**Results** SPUC attendance is associated with increased care at home, with the mean rate across all regions at 38.4%, compared to 27.2% for DCA crews. Higher rates of care at home were associated with codes relating to ineffective breathing and unresponsiveness, difficulty speaking or visible skin perfusion changes. This suggests that SP/NUCs can support alternative care pathways, for high-acuity respiratory presentations.

Regional variation persisted. The effect was most notable in two ICS areas, whilst the third showed lower care at home where SP/NUCs attended (28.6% vs. 33.7%). This divergence may reflect local differences in acuity, dispatch profiles, or the

implementation of clinical pathways, underscoring the complexity of system-level influence.

An estimated mean cost of £417 per conveyed patient and £287 per non-conveyed patient, produces an estimated saving of £14,255 per 1,000 SPUC- attended incidents.

The comparison between two reference periods revealed a strengthening of the SP/NUC effect. In contrast, conveyance rates for DCA-attended incidents either remained stable or increased, suggesting that the observed change is linked to changes in SP/NUC clinical practice or operating environment.

**Conclusions** These findings reinforce the value of SP/NUCs in providing care closer to home for patients with high acuity breathing difficulties and offers notable financial savings. The variation across regions and dispatch profiles highlights the importance of local context, including case mix, operational models, and clinical decision-making culture. Further health economic evaluation is recommended, and multivariate analysis of other factors may be necessary to inform service development.

## S11 SPIRO-AID: A RANDOMIZED CONTROLLED TRIAL OF AI-ASSISTED SPIROMETRY INTERPRETATION IN PRIMARY CARE

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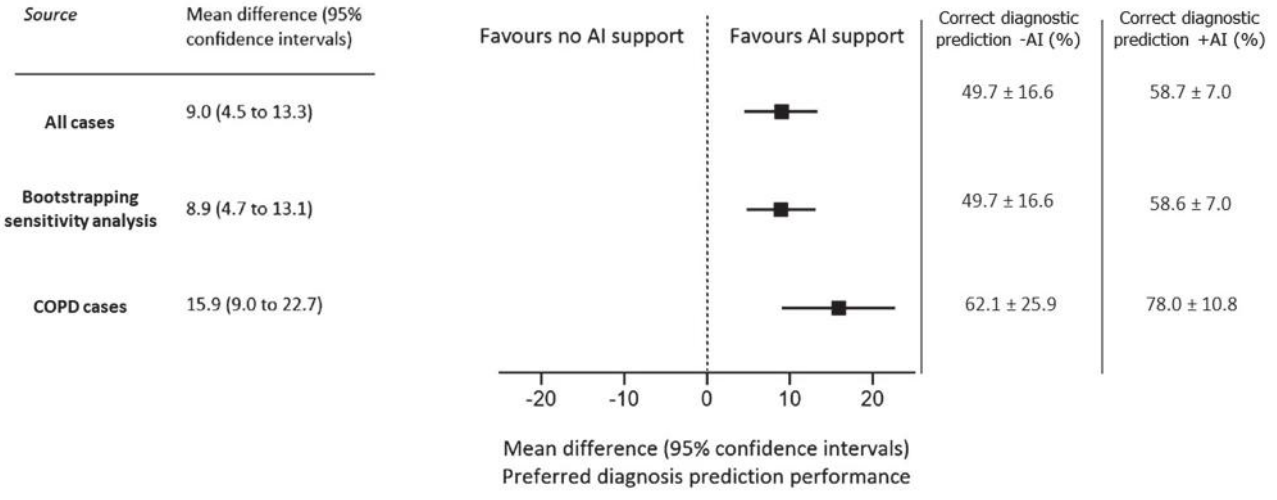
10.1136/thorax-2025-BTSabstracts.17

**Background** The quality of spirometry interpretation is highly variable in primary care, contributing to delayed diagnosis of chronic respiratory diseases. We hypothesised that Artificial intelligence (AI) decision support software would improve spirometry interpretation performed by primary care clinicians.

**Methods** We conducted a parallel group, randomised controlled superiority trial. Clinicians working in primary care who routinely refer for, perform or interpret spirometry assessed fifty real-world patient spirometry records cases, providing 'preferred diagnosis' (most likely diagnosis) through an online platform either with (Intervention) or without (Control) AI decision support software. The primary outcome was the percentage of cases where 'preferred diagnosis' agreed with the reference diagnosis pre-determined by expert pulmonologists with access to full patient records. Sensitivity analysis for missing data, and subgroup analysis in cases with a reference diagnosis of chronic obstructive pulmonary disease (COPD) was performed. Secondary outcomes included performance in differential diagnosis prediction, technical quality assessment, pattern interpretation and self-rated confidence in spirometry interpretation.

**Results** Between June 2023 and April 2024, 234 participants were randomised with 133 (57%) completing (Intervention n=67, Control n=66): 73% female, 42% were general practitioners, 50% nurses. The addition of AI decision support software improved the mean 'preferred diagnosis' prediction performance from 49.7% (Control) to 58.7% (Intervention) in all cases (difference 9.0 [95% confidence interval 4.5 to 13.3]%, P=0.001) and in COPD cases (15.9 [9.0 to 22.7]%, p<0.001, [figure 1]). Sensitivity analysis showed the results were robust to adjusting for job role, sex, spirometry role and registration for spirometry accreditation, and assessing the potential impact of missing data. Differential diagnosis prediction performance and technical quality assessment performance also improved with intervention, but not pattern interpretation nor clinician confidence levels.

**Conclusion** In primary care clinicians, AI spirometry decision support software significantly improved diagnosis prediction performance. AI decision support can help to address suboptimal interpretation of spirometry in primary care.




**Abstract S11 Figure 1** Estimates of study group effects on primary outcome: preferred diagnosis prediction performance. Data expressed as mean difference (95% confidence interval)

## S12 IMPROVING SAFETY AND TREATMENT PRECISION FOR ACUTE COPD EXACERBATIONS (TRACE): FEASIBILITY OF POINT-OF-CARE TESTING IN COMMUNITY PHARMACIES. A QUALITATIVE STUDY

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10.1136/thorax-2025-BTSabstracts.18

**Introduction and Objectives** Acute COPD exacerbations (AECOPDs) account for 11% of primary care antibiotic prescriptions & up to 40% of patients receive repeated oral corticosteroids (OCS). Without objective markers, prescribing decisions rely on symptoms and examination, without test corroboration. Drug rescue packs are usually issued without clinical assessment and obtained directly from pharmacists. Use of point-of-care blood tests (POCTs) of CRP and blood eosinophil count can decrease prescriptions of antibiotics & OCS by 22% and 33% respectively, without harm.

This study aimed to explore the opinions of community pharmacists, GPs and practice nurses of the feasibility and acceptability of POCT COPD assessment in pharmacies.

**Methods** Semi-structured interviews (pharmacists, GPs, practice nurses) to undertaken to determine the facilitators & barriers to implementing POCT COPD assessment within community pharmacies. Interviews were audio-recorded and transcribed verbatim for coding and inductive thematic analysis.

**Results** 20 participants (11 pharmacists, 8 GPs, 1 nurse) in London and Kent were invited and agreed to being interviewed. Participants were recruited via Integrated Care Boards, Local Pharmaceutical Committees and support groups for community pharmacists. All supported the intervention and its feasibility. Pharmacists were enthusiastic about expanding their clinical scope and felt confident it fitted their current role. POCTs are established in community pharmacies. GPs and nurses were willing to support the pharmacists. Proposed benefits of the intervention included a decrease in unnecessary prescribing, the reassurance for patients and clinicians that would be obtained from objective results, the empowerment of patients and improvement in self-management of COPD, professional recognition of pharmacists' clinical role, and an increased accuracy in the recording and monitoring of drug rescue packs. Key context implementation factors: Patient education (required patient behavioural changes), Clinical demand (time & pharmacist reimbursement), Communication (data sharing/storage, safety-netting).

**Conclusions** Pharmacists, GPs and practice nurses support the development and implementation of a POCT COPD assessment pathway in community pharmacies, potentially improving patient medication stewardship.

# Approaches to predict survival and increased mortality in ILD

## S13 QUANTITATIVE CT IS ASSOCIATED WITH PROGRESSION FREE SURVIVAL IN IDIOPATHIC PULMONARY FIBROSIS AND FIBROTIC HYPERSENSITIVITY PNEUMONITIS

[1,2,3,4,5,6,7]G Dixon, [2]HI Adamali, [2]J Hardy, [2]M Wells, [2,6]S Mulholland, [2]G Ruffino, [8]P Croxford, [9]N Kennedy, [10]H Emms, [5]T Hartley, [11]W Bou-Zeid, [11]C Rennison-Jones, [11,12]A Devaraj, [11,12,13]PM George, [1,3,6,7]P Rivera-Ortega, [1,3,7,10]S Stanel, [5,14]JCL Rodrigues, [1,3]CJ Scotton, [15,16,17]K Tsaneva-Atanasova, [2,4,6]SL Barratt, [1,3,7,10]MA Gibbons. *[1]Department of Clinical and Biomedical Sciences, University of Exeter, Exeter, UK; [2]Bristol Interstitial Lung Disease Service, North Bristol NHS Trust, Bristol, UK; [3]NIHR Exeter Biomedical Research Centre, University of Exeter, Exeter, UK; [4]NIHR Bristol Biomedical Research Centre, University of Bristol, Bristol, UK; [5]Royal United Hospitals Bath NHS Foundation Trust, Bath, UK; [6]Academic Respiratory Unit, School of Health Sciences, University of Bristol, Bristol, UK; [7]South West Peninsula Interstitial Lung Disease Service, Exeter, UK; [8]Department of Pathology, North Bristol NHS Trust, Bristol, UK; [9]Royal Devon University Hospitals NHS Foundation Trust, Exeter, UK; [10]Academic Department of Respiratory Medicine, Royal Devon University Healthcare NHS Foundation Trust, Exeter, UK; [11]Brainomix, Oxford, UK; [12]Royal Brompton Hospital, London, UK; [13]National Heart and Lung Institute, Imperial College London, London, UK; [14]Department of Health, University of Bath, Bath, UK; [15]Living Systems Institute, University of Exeter, Exeter, UK; [16]EPSRC Hub for Quantitative Modelling in Healthcare, University of Exeter, Exeter, UK; [17]Department of Mathematics and Statistics, Faculty of Environment, Science and Economy, University of Exeter, UK*

10.1136/thorax-2025-BTSabstracts.19

**Introduction** Idiopathic pulmonary fibrosis (IPF) and fibrotic hypersensitivity pneumonitis (fHP) cause significant morbidity and mortality. Quantitative computed tomography (qCT) may

**Abstract S13 Table 1** Patients included in Cox proportional hazards model of progression free survival in idiopathic pulmonary fibrosis and fibrotic hypersensitivity pneumonitis

| | Fibrotic hypersensitivity pneumonitis | Idiopathic pulmonary fibrosis |
|---|---|---|
| Patient (n) | 107 | 113 |
| Follow-up median (months) (IQR) | 22.5 (12.8–38.6) | 21.1 (11.9–32.6) |
| Sex - Male | 45 (42%) | 100 (88.4%) |
| Mean age at baseline (±SD) | 71.0 (8.8) | 73.4 (9.7) |
| Ever smoker | 60 (56.1%) | 84 (74.3%) |
| Charlson comorbidity index median (IQR) | 4 (3–5) | 4 (4–5) |
| Mean FVC% predicted at baseline (±SD) | 77.5% (16.7%) | 82.8% (16.1%) |
| Mean TLCO% predicted at baseline (±SD) | 61.2% (17.4%) | 57.5% (15.1%) |

Legend: IQR=interquartile range; SD=standard deviation

provide radiomic biomarkers to assist in the prognostication and disease monitoring of these conditions. We aimed to investigate a prognostic role for qCT in fHP and IPF.

**Methods** This retrospective, observational, multicentre study analysed electronic records of patients with an ILD multidisciplinary diagnosis of fHP or IPF. Patients required ≥1 HRCT and ≥2 pulmonary function tests (PFTs) between 2015 and 2022. Data were censored on 30/01/2025. Demographics, PFTs, bronchoalveolar lavage (BAL), CT scans, and survival data were recorded. Brainomix e-Lung used deep-learning to derive radiomic biomarkers from anonymised scans.

Correlations between PFTs and qCT was assessed using Pearsons correlation. For patients with baseline paired PFTs and qCT and ≥12 months follow-up Cox proportional hazards models were used to assess association of qCT, age, sex, smoking status, Charlson comorbidity index and baseline PFTs with progression-free survival (time to 10% FVC decline, 15% TLCO decline, or death).

**Results** 155 patients with fHP and 216 with IPF were included. Of the CT scans, 485/558 (fHP) and 388/456 (IPF) had successful quantitative analysis, and 304/558 (fHP) and 338/456 (IPF) had paired PFTs (within 90 days of scan acquisition). Forced vital capacity (FVC)% predicted and transfer factor (TLCO)% predicted correlated with e-Lung's radiomic weighted reticulovascular score (WRVS) in both fHP (r=-0.56 and r=-0.49 respectively, both p<0.05) and IPF (r=-0.52 and r=-0.57 respectively, both p<0.05) across all paired scans.

In patients with fHP, BAL lymphocytosis was negatively correlated with WRVS (n=45, r=-0.352, p=0.02).

Table 1 summarises patients included in the Cox proportional hazards analysis, WRVS was independently associated with progression-free survival in both IPF and fHP (fHP: HR 1.08, 95%CI 1.03–1.12, p<0.05; IPF: HR 1.08, 95%CI 1.02–1.15, p<0.05).

**Conclusions** WRVS correlates with PFTs and is negatively associated with lymphocytosis on BAL, highlighting the potential for WRVS to assess non-fibrotic burden in fHP patients. Baseline WRVS is associated with progression-free survival in both IPF and fHP and may aid prognostication in these patient groups.

## S14 ABSENCE OF CT IMAGING IN AEILD ADMISSIONS ASSOCIATED WITH HIGHER MORTALITY RISK

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10.1136/thorax-2025-BTSabstracts.20

**Background** Acute exacerbations of interstitial lung disease (AEILD) are devastating events with high mortality. A definite AEILD is defined by <30-day deterioration in respiratory symptoms, not due to cardiac, thrombotic or pneumothorax causes, with new bilateral ground-glass changes on a background of ILD.[1] High-resolution CT imaging is central to this definition, yet its use during AEILD admissions is variable and understudied. We evaluated whether CT imaging during AEILD admission was associated with survival and differences in care.

**Methods** We conducted a multicentre retrospective observational study of ICD-10-coded primary ILD admissions in patients ≥18 years, across 11 NHS hospitals in North West England between 01.01.2017 and 31.12.2019. AEILD events were identified using clinical criteria via manual review. Events were grouped by whether thoracic CT imaging was performed. Primary outcome was time to death from admission commencement.

**Results** 511 AEILD admissions were identified; 192 (37.6%) had CT imaging. Median survival was significantly longer in the CT group (144 vs. 100 days; p=0.027); mean survival was also higher (493.8 vs. 394.7 days). Respiratory team involvement was more frequent in the CT group (79.2% vs. 62.1%; p<0.001), as was high-dose intravenous methylprednisolone use (11.5% vs. 3.8%; p=0.002). Mean length of stay was longer in the CT group (11.5 vs. 6.6 days; p<0.001), while palliative care input was more common in the non-CT group (20.1% vs. 14.6%). Baseline demographics, lung function and comorbidity scores were similar.

**Discussion** CT imaging during AEILD admissions was associated with improved survival, more respiratory specialist input and greater use of high-dose steroid treatment. This may reflect diagnostic certainty influencing care escalation, or that in the non-CT group some patients were too unstable to undergo imaging and further management.

**Conclusion** Hospitalised AEILD patients face high mortality. Our findings suggest variation in CT use may influence care and outcomes. Further prospective studies are needed to determine whether CT imaging contributes directly to improved survival or is a proxy for more intensive management.

### REFERENCE

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## S15 THE ROLE OF QUANTITATIVE CT IN THE PROGNOSTICATION AND MONITORING OF SYSTEMIC AUTOIMMUNE RHEUMATIC DISEASE RELATED INTERSTITIAL LUNG DISEASE

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**Background** Interstitial lung disease (ILD) is a significant cause of morbidity and mortality in people with systemic autoimmune rheumatic disease (SARD). Effective screening, accurate prognosis and monitoring are essential components of patient

**Abstract S15 Table 1** Demographics of 140 patients with systemic autoimmune rheumatic disease - interstitial lung disease

| MDT Diagnosis | Number of Patients | Age (mean ± SD) | Male | Ever smoker | Median CCI | FVC% predicted (± SD) | TLCO% predicted (± SD) |
|---|---|---|---|---|---|---|---|
| Polymyositis and systemic sclerosis overlap (Anti-PMSCL) | 19 | 62.7 (9.5) | 7 (37%) | 8 (42.1%) | 4 (1) | 80.1 (20.2) | 69.3 (25.2) |
| Anti-synthetase syndrome | 46 | 56.7 (13.2) | 21 (46%) | 19 (41.3%) | 3 (1) | 72.4 (19.9) | 61.4 (19.1) |
| Rheumatoid-arthritis associated ILD | 23 | 65.2 (8.6) | 17 (74%) | 14 (60.9%) | 4 (1.5) | 88.0 (21.4) | 65.7 (14.9) |
| Sjogrens Syndrome | 1 | 52.6 (n/a) | 0 | 1 (100%) | 3 (0) | 66.6 | 75.5 |
| Systemic sclerosis | 51 | 55.3 (14.7) | 13 (26%) | 21 (41.2%) | 3 (3) | 76.4 (17.2) | 61.8 (22.8) |
| Total | 140 | 58.4 (13.2) | 58 (41%) | 63 (45%) | 4 (2) | 77.4 (19.7) | 63.4 (20.7) |

Legend: CCI= Charlsons comorbidity, FVC= Forced vital capacity, ILD= Interstitial lung disease, MDT= Multi-disciplinary team, PMSCL= Polymyositis-Scleroderma, TLCO= Transfer capacity for carbon monoxide

management. High-resolution computed tomography (HRCT) plays a role in all these aspects but is currently reliant upon expert radiological interpretation. Quantitative CT (qCT) using deep-learning may provide a powerful adjunct tool. We aimed to assess this in patients with SARD-ILD.

**Methods** We conducted a retrospective, observational cohort study across 3 centres in England. Patients required a multidisciplinary team diagnosis of SARD-ILD, ≥1 HRCT and ≥2 pulmonary function tests (PFTs) between 2015 and 2022. Data were censored on 30/01/2025. Demographics, PFTs, pathology and mortality data were obtained from electronic patient records. Anonymised HRCT scans were analysed using deep-learning (Qureight Ltd), to quantify airway, fibrosis, ground glass and vascular abnormalities. qCT and PFTs within 90 days of each other were paired for baseline and serial Pearsons correlation assessment.

For patients with ≥12 months follow-up Cox proportional hazards modelling identified baseline factors (age, gender, disease sub-type, ethnicity, PFTs and qCT) associated with progression free survival (PFS).

**Results** 140 patients were included (table 1). 533/580 (91.9%) scans had successful quantitative analysis. 378/533 and 359/533 had paired forced vital capacity (FVC) and transfer capacity (TLCO). FVC and TLCO correlated significantly with all qCT biomarkers even when adjusted for per patient differences ($p<0.05$). Strongest correlations were seen between fibrosis extent score (Fibr8) and FVC and TLCO ($r=-0.52$ and $r=-0.56$, $p<0.05$).

When assessed longitudinally only vessel quantification (Vascul8) remained significant after adjustment for per patient differences ($r=-0.42$ and $r=-0.52$, $p<0.05$).

On multivariate Cox proportional hazards analysis of 111/140 eligible patients (median follow-up 4.83 years, IQR 2.14–7.98) extent of ground glass abnormality (Glass8) at baseline was associated with reduced risk of PFS (HR 0.91; CI95% 0.84–0.98) and Fibr8 was associated with disease progression (HR 1.09; CI95% 1.03–1.15).

**Conclusions** QCT correlates significantly with changes in pulmonary physiology and appears useful in predicting disease progression in SARD-ILD. An adjunct role for qCT in identification, prognostication and disease monitoring of ILD in patients with SARD warrants prospective evaluation.

## S16 IMPACT OF HOSPITALISATION IN ILD SUBTYPES AND PREDICTORS OF OUTCOME

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10.1136/thorax-2025-BTSabstracts.22

**Background** Predicting the clinical course of Interstitial Lung Disease (ILD) remains challenging. Hospitalisations are frequent across all subtypes, altering the trajectory and resulting in significant morbidity and mortality. Outside IPF there is scarce literature regarding the impact of hospitalisation. We aim to further understand the significance of hospital admissions in ILD patients.

**Methods** We undertook a retrospective observational study of all ILD medical admissions between January 2022 and December 2023. The cohort was stratified by ILD subtype, data collection included demographics, admissions investigations (FBP, CRP, Trop, BNP, Neutrophil Lymphocyte Ratio [NLR]), and measures of disease burden at clinic review. Outcome measures included inpatient mortality, length of stay (LOS), readmission within 30 and 90 days and survival. We interrogated the

**Abstract S16 Table 1** Admission baseline characteristics and measures of disease burden in ILD subtypes

| | N | Age* | FVC% | TF%** | 6MWT* | LTOT | Charleston** | GAP | Survival** |
|---|---|---|---|---|---|---|---|---|---|
| Unclassifiable | 66 | 79.1 (8.8) | 73 (17) | 52 (16) | 215 | 39% | 6.2 (1.5) | 4.48 (1.5) | 10.6 |
| CPFE | 19 | 69.6 (10.7) | 86 (25) | 32 (11) | 284 | 63% | 4.21 (1.9) | 4.82 (1.6) | 3.2 |
| Fibrotic HP | 13 | 73.5 (7.7) | 66 (17) | 46 (13) | 232 | 46% | 4.69 (1.1) | 5 (1.5) | 6.8 |
| IPF | 12 | 79.9 (8.2) | 79 (13) | 38 (17) | 184 | 50% | 6.08 (6.08) | 4.25 (1.0) | 6.7 |
| Sarcoid | 4 | 65.3 (7.4) | 79 (20) | 45 (15) | 140 | 50% | 3.25 (0.5) | 3.5 (1.0) | 6.1 |
| Total | 114 | 76.5 (9.8) | 75 (19) | 45 (17) | 224 | 46% | 5.56 (5.6) | 4.54 (1.6) | 7.1 |

**Notes**: Results presented as mean (SD), Survival in months. Difference between groups * $p < 0.05$, **$p < 0.001$. CPFE (Combined Pulmonary Fibrosis & Emphysema), IPF (Idiopathic Pulmonary Fibrosis), HP (Hypersensitivity Pneumonitis)

relationship of combinations of admission investigations and ILD characteristics to outcome measures.

**Results** During the study period there were 114 admissions events (86 individual patients). Baseline characteristics per subgroup are presented in table 1. Overall: mean age 76.5 (SD 9.8), 54% male, median Charlestown Comorbidity Index (CCI) 6 (IQR 4.25–7), median GAP score 5 (IQR 3–6), 46% required LTOT, and 19% were treated with anticoagulation pre-admission. Treatment: 82% received antibiotics, 57% received corticosteroids, 19% received diuretics, and 3% received Respiratory Support. Median LOS 6 days (IQR 4–9). The inpatient mortality rate was 8%, with readmission within 30 days 36% and 90 days 52%. The median survival following admission was 7 months, 12% died in the community. IP mortality correlated significantly with admission CRP (r 0.275), there was no relationship with NLR, Trop, BNP, or anticoagulation use. Age (r 0.207) and 6MWT (r -0.438) were significantly correlated with readmission, however CCI was not. LTOT use preadmission was associated with reduced survival (r -0.257) whereas preserved Transfer factor (r 0.257) was associated with survival.

**Conclusion** Predictors of increased healthcare utilisation include age and decreased functional capacity (6MWT) but not comorbidity. Overall, there is a poor prognosis in all ILD subtypes following hospital admission, this may provide a window of opportunity to proactively engage with palliative care services.

## S17 LOW SERUM TESTOSTERONE PREDICTS SURVIVAL IN PATIENTS IN FIBROTIC INTERSTITIAL LUNG DISEASE: LONGITUDINAL FOLLOW-UP OF THE STARSHIP COHORT

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10.1136/thorax-2025-BTSabstracts.23

**Introduction** Fibrotic interstitial lung disease (f-ILD) has a high mortality, yet no cure. The causal role of short telomeres in f-ILD is well-established.[1] Shorter telomere length is associated with lower serum sex hormone concentrations. STARSHIP study demonstrated lower bioavailable oestrogen and testosterone in patients with f-ILD compared with age/sex-matched controls, and an association of lung function with sex hormone levels.[2] We sought to understand the impact of low testosterone on survival outcomes in patients with f-ILD.

**Methods** 102 patients with f-ILD attending a regional ILD clinic were recruited between March 2022-February 2023. Serum testosterone levels were measured at baseline. Date of death was recorded to June 2025.

Kaplan-Meier and Cox proportional hazard models were used to compare male patients with f-ILD grouped by normal versus low free testosterone (<225pmol/L) and female patients grouped by free androgen index (FAI). Cox regressions were adjusted for age, forced vital capacity (FVC) and lung diffusion capacity (DLCO).

**Results** Mean free testosterone concentration was lower in male patients (N=80; 211pmol/L [95% CI: 192–230]) than age/sex-matched controls (265pmol/L [95% CI: 243–287]). By June 2025, 31/50 (62%) male patients with low testosterone and 9/30 (30%) with normal testosterone had died. Kaplan-Meier analysis demonstrated a significant difference in survival between the two groups (log-rank test $Chi^2=6.6$, p=0.0092).

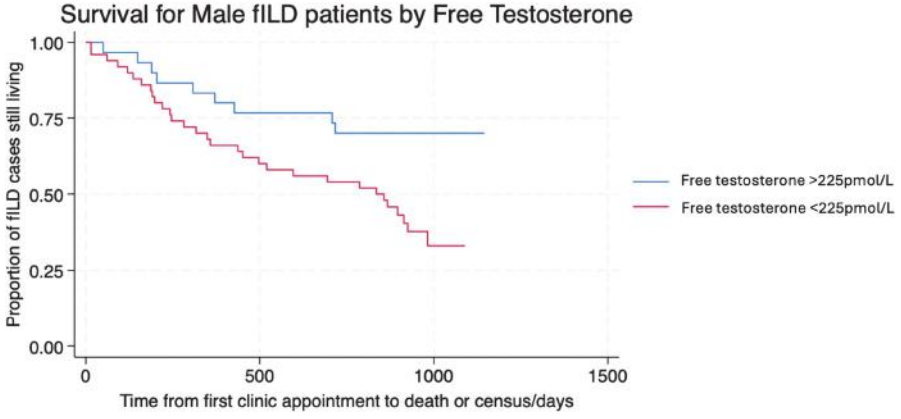
In Cox proportional hazard modelling, low testosterone increased risk of death (HR=2.23, p=0.038) when accounting for expected variables (baseline age, FVC and DLCO). Survival was influenced by an interaction between DLCO and low testosterone that varied with time (p=0.027).

The study was under-powered to analyse female patients (N=22); however, dichotomising by group mean FAI, 8/12 (67%) female patients below vs 3/10 (30%) above the mean had died, although did not reach statistical significance.

**Conclusions** Low testosterone in males with f-ILD increases risk of mortality compared to male patients with normal testosterone, including when adjusting for age, FVC and DLCO. Similar findings are suggested in female patients; more data is required for confirmation. These findings may inform future research into new treatment options for f-ILD.

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**Abstract S17 Figure 1** Kaplan-Meier survival estimates for male patients with f-ILD compared by free testosterone from trail recruitment to census date June 2025

## S18 STREPTOCOCCUS IS ENRICHED IN THE GUT MICROBIOME IN IDIOPATHIC PULMONARY FIBROSIS AND ASSOCIATED WITH WORSE SURVIVAL

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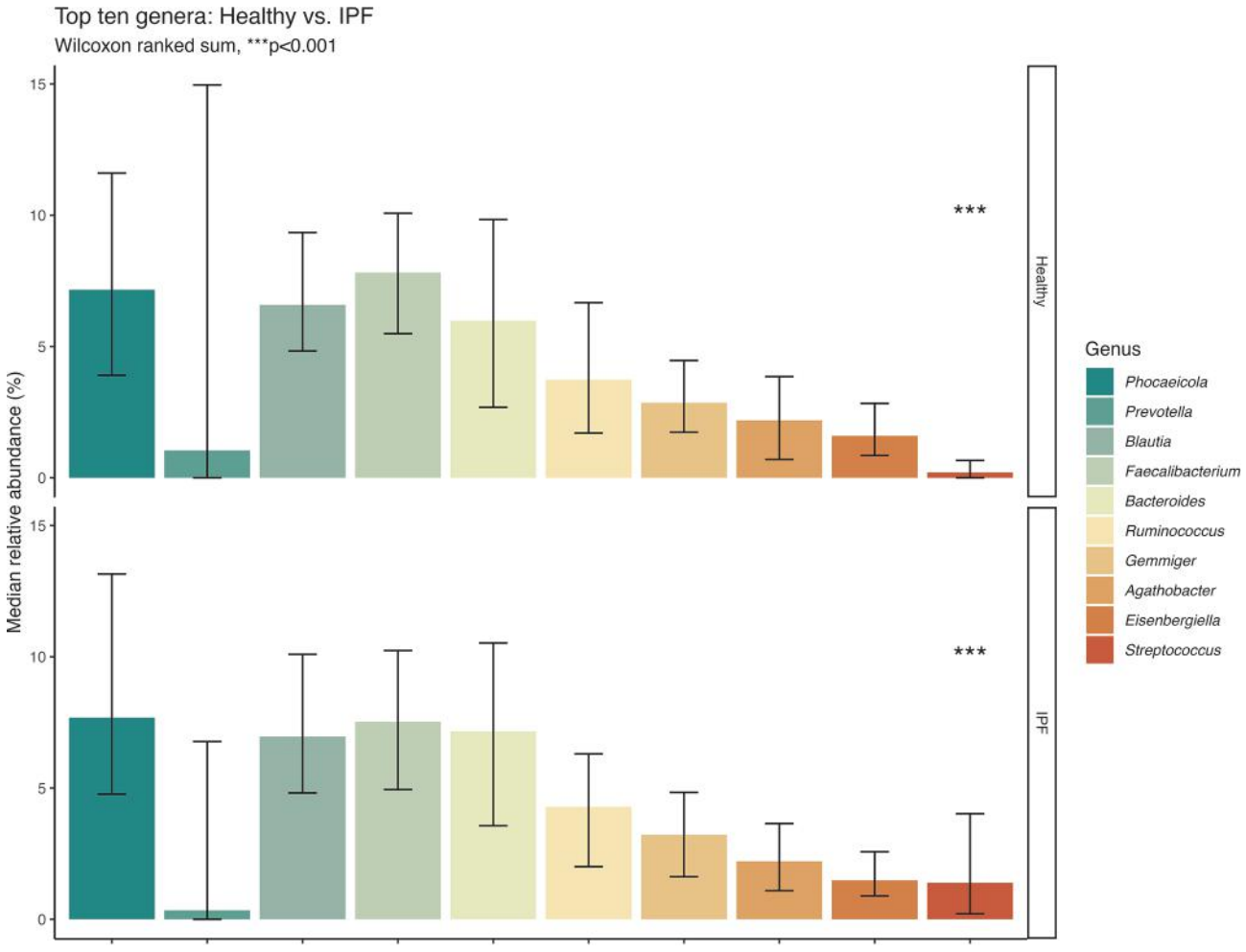
10.1136/thorax-2025-BTSabstracts.24

**Introduction** Idiopathic pulmonary fibrosis (IPF) is a progressive and incurable lung disease with a median survival of less than four years. Disturbance of the lung microbiome has been identified as a potential risk factor for the development and progression of IPF, and may play a mechanistic role in fibrogenesis. Despite growing evidence that changes to the gut microbiome can contribute to the pathogenesis of respiratory conditions, its role in IPF is poorly understood. We therefore aimed to investigate the associations between the gut microbiome and disease behaviour in IPF.

**Methods** Incident cases of IPF and healthy controls were recruited prospectively. OMNIgene GUT sample collection kits were sent to participants, returned by post, and frozen prior to processing. 16S rRNA gene sequencing of the v4 hypervariable region was performed on DNA extracted from stool samples. Following comparisons with healthy controls, the inferred functional potential of the IPF microbiota and associations with progression-free survival were explored.

**Results** Stool samples were collected from 184 patients with IPF and 146 healthy controls. The IPF cohort was predominantly male (86.8%) with a median age of 75.1 years. The majority were either current or former smokers (74.2%). Lung function was impaired, with a mean forced vital capacity (FVC) of 78.2% predicted (±SD 14.3), and a diffusing capacity of the lung for carbon monoxide (DLCO) of 49.4% predicted (±SD 13.5). The gut microbiome in IPF was significantly different to that of healthy controls, driven by the enrichment of *Streptococcus*. Age and reflux treatment also influenced microbial composition, with therapy for reflux driving changes in both health and disease. In patients with IPF, a higher abundance of *Streptococcus* was associated with significantly worse progression-free survival. Inferred functional analysis showed a higher abundance of microbial pathways involved in host invasion and infection in IPF, including endocytosis, bacterial invasion of epithelial cells, and *Staphylococcus aureus* infection.

**Conclusions** There are significant alterations in the gut microbiome in patients with IPF, driven by an increased abundance of *Streptococcus*. These changes may contribute to pathogenesis and disease progression.




**Abstract S18 Figure 1**

# Sleep matters

## S19 IMPACT OF VIRTUAL CPAP SET-UP ON LONG-TERM COMPLIANCE

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10.1136/thorax-2025-BTSabstracts.25

**Introduction** Continuous positive airway pressure (CPAP) therapy, for obstructive sleep apnoea (OSA), transitioned from a face-to-face (F2F) to virtual (VR) set-up during the COVID-19 pandemic. Appleby et al reported no effect on short-term CPAP compliance, however little literature exists for long-term adherence. Consistent CPAP usage is essential for symptom reduction and prevention of health complications. This retrospective service evaluation compares long-term CPAP compliance and symptomatic relief between both groups to ascertain whether VR set-up produces equally effective patient outcomes.

**Method** 100 patients using CPAP were sequentially sampled from April 2019 (F2F) and a further 100 from April 2020 (VR). Demographic data and OSA severity levels were collected from EPR. Current Epworth Sleepiness Scale (ESS) scores and compliance data were collected in 2024, at a median of 3.4 years post CPAP issuance, via EPR and the remote CPAP monitoring platform. Statistical analysis was conducted using STATA software.

**Results** No significant differences were found between the proportion of patients currently using CPAP (F2F 89.7% vs VR 80.6%, p=0.20), or in the improvement of ESS scores (F2F 7.5 vs VR 8.6, p= 0.28). Statistically significant differences were found in the average CPAP usage per night (F2F 6.83 hours vs VR 6.45 hours, p= 0.012) illustrated in figure 1, and the number of days of CPAP usage in the last month (F2F 30 days vs VR 27.5 days, p= 0.0037).

**Conclusion** Currently, a similar proportion of both patient groups continue to use CPAP. Since both groups greatly exceeded the threshold for effective CPAP compliance (≥4 hours/night on ≥70% of nights over 30 days), the F2F group's additional 20 minutes and 2.5 days of CPAP usage is unlikely to be of clinical significance. Furthermore, both groups reported similar ESS score improvements reflecting comparable symptomatic benefit. Hence, VR CPAP set-up can be confidently continued by this sleep service.

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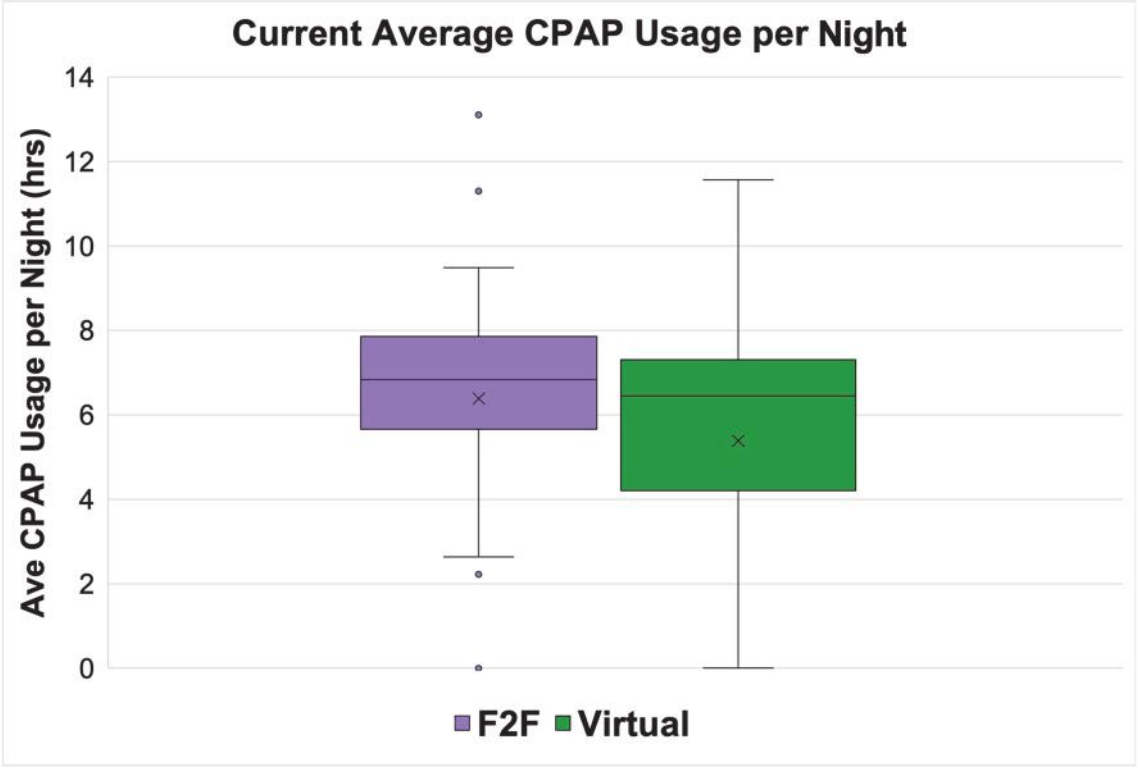
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## S20 FROM CLINIC TO CLICK – DIGITAL OBSTRUCTIVE SLEEP APNOEA IDENTIFIER PATHWAY

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10.1136/thorax-2025-BTSabstracts.26

Obstructive sleep apnoea (OSA) is the most common chronic respiratory disorder, affecting an estimated 1 billion people worldwide. Current OSA models of care are cumbersome and characterised by unacceptable waiting times. Digital innovation of OSA care, leveraging home-based sleep studies and remote




**Abstract S19 Figure 1** Boxplots displaying the distribution of current average hourly CPAP usage per night in both set-up groups

data analysis, could expedite diagnosis and treatment, potentially enhancing patient experience and outcomes.

This pilot study sought to streamline the diagnosis and management of obstructive sleep apnoea in a tertiary hospital in Ireland. Through a fully remote digital pathway, patients were invited to download a bespoke app and complete a series of symptom questionnaires. Thereafter, they underwent a home sleep study, followed by a video consultation with a sleep specialist.

This pilot study took place over 17-month period. 187 appointments were offered. Of 109 home sleep studies completed; 85 (78%) revealed an AHI of $\geq$ 5 events per hour [24 patients with AHI 5–15 events per hour, 28 patients with AHI 15–30 per hour, 33 patients with AHI $\geq$30 events per hour]. Of these 85 participants, 56 (65.8%) were referred for CPAP therapy. Overall average nightly usage was 5.8 hours, 48 patients (56%) remain adherent to therapy at 6 months. Average time from referral to virtual review was 5.4 months.

The results from this pilot study are promising, with significant reduction in waiting times and expedited diagnoses of OSA in the majority of patients reviewed. To strengthen the findings, future studies should include assessment of patient-related outcomes such as patient satisfaction, experience and adherence and CPAP therapy when compared to standard of care pathways.

## S21 POSTOPERATIVE OUTCOMES IN PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA: A RETROSPECTIVE STUDY COMPARING OUTCOMES OF BARIATRIC SURGERY IN BLACK AND WHITE PATIENTS

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10.1136/thorax-2025-BTSabstracts.27

**Introduction** Obstructive sleep apnoea (OSA) is highly prevalent in the general population, this is in parts due to a rise in obesity. It remains difficult to achieve substantial weight loss with conventional measures, however, bariatric surgery has been shown to reliably reduce significant weight. Ethnicity is an important factor that has been described to impact healthcare delivery and clinical outcomes. The objective of this study was to compare immediate and perioperative outcomes between black and white patients diagnosed with OSA undergoing bariatric surgery.

**Methods and Patients** We reviewed the data of patients who were screened for OSA using sleep studies as part of the pre-bariatric surgical pathway between 2017–2025 in a large tertiary referral centre (NHS; Guys and St Thomas). Data were collected from the electronic patient records (EPIC); we recorded demographics, ethnicity, and postoperative outcomes (length of stay, complication rates and weight loss at 12 months). Differences were tested using Shapiro-Wilk, Independent T-test and Chi-square and presented as mean (SD) or median (IQR 25th – 75th). Level of significance was chosen as $p<0.05$.

**Results** We screened 831 patients and due to incomplete datasets, we included 245 patients in the analysis. Of those 174 were white (54 ± 12 Years, 121Females and 49 Males, 49.2 ± 8.3 Kgxm$^2$) and 71 Black (52 ± 10 Years (p=0.22), 67Females and 4 Males (p=0.001) and 46.7 ± 7.0 Kgxm$^2$(p=0.02)). The length of postoperative hospital stay was shorter (2 IQR 2–3 *vs* 4 IQR 2–5 p=0.001). Although the absolute rate of complications was similar between the groups (9.8% *vs* 9.8% p=0.97), white patients experienced less severe complications (e.g., infection) while black patients had more severe complications (e.g, retained needle in situ). At one year follow up, more White patients attended the appointment (46.0 *vs* 33.8% p=0.19)),had more significant weight-loss (-30.1% IQR -22.4- -38.7% *vs* -26.3% IQR -20.1- -34.9%, p= 0.04), and lost 3.4% more of their baseline weight on average.

**Conclusion** Black patients experienced less favourable peri and postoperative outcomes following bariatric surgery. These findings highlight potential disparities in the operative care of patients with different ethnicities and raises questions how to improve on these outcomes.

## S22 INTERDISCIPLINARY DISCUSSION OF NON-CONTINUOUS POSITIVE AIRWAY PRESSURE THERAPIES FOR OBSTRUCTIVE SLEEP APNOEA: A RETROSPECTIVE REVIEW OF DIFFICULT-TO-TREAT CASES

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10.1136/thorax-2025-BTSabstracts.28

**Introduction** Continuous Positive Airway Pressure (CPAP) therapy is the first-line treatment for Obstructive Sleep Apnoea (OSA), but long-term adherence to CPAP is limited. This study evaluates the implementation and outcomes of a multidisciplinary team (MDT) approach to manage CPAP-intolerant patients using alternative non-CPAP therapies.

**Methods** A retrospective review was conducted on 'difficult-to-treat' OSA cases discussed at multidisciplinary team meetings at the Sleep Disorders Centre at Guy's and St Thomas' NHS Foundation Trust [project no. 17160]. Patient demographics, comorbidities, OSA severity (Apnoea-Hypopnoea Index [AHI], Epworth Sleepiness Scale [ESS], 3% Oxygen Desaturation Index [ODI]), management plans and outcomes were extracted from the meeting minutes and clinical records. Statistical analysis was performed using ANOVA to compare baseline characteristics (AHI, age, Body Mass Index [BMI]) across treatment groups, and paired t-tests to assess pre- and post-treatment changes in AHI, ESS, and ODI, with significance set at $p<0.05$.

**Results** We reviewed a cohort of 138 cases of patients (103/138 male, age 51 (17) years, BMI 29.5 (5.1) kg/m$^2$) with severe OSA (AHI 37.7 (21.4) hour$^{-1}$). Common comorbidities included hypertension (29.0%), anxiety/depression (25.4%) and cardiac disease (16.7%). 17.4% had a history of previous ENT intervention. Treatment selection was guided by predefined criteria including OSA severity, BMI, patient preferences, anatomical abnormalities, and comorbid conditions. The recommended management options were tailored Mandibular Advancement Devices (MADs, 27.5%), Hypoglossal Nerve Stimulation (HNS, 24.6%), and weight management (23.9%). Other options included BiPAP/ASV (10.1%), other ENT surgery (10.9%), and CPAP retrial (13.8%) for patients with higher AHI. In comparison, less invasive options such as MAD and specific postural advice (15.9%) were preferred for those with milder OSA (AHI<30). Follow-up data from a

subset of patients demonstrated significant reductions in AHI (-44.1%, n=29), ESS (-32.9%, n=13), and ODI (-67.2%, n=12) post-treatment ($p<0.05$, respectively).

**Conclusion** An interdisciplinary approach enables the delivery of clinical and evidence-based recommendations for OSA patients who have failed conventional treatments, leading to symptomatic improvements in selected individuals through personalised treatment strategies.

## S23 OBSTRUCTIVE SLEEP APNOEA AND METABOLIC SYNDROME IN ADULTS: EVIDENCE FROM CASE-CONTROL STUDIES OF A SYSTEMATIC REVIEW AND META-ANALYSIS

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10.1136/thorax-2025-BTSabstracts.29

Adults with Obstructive Sleep Apnoea (OSA) are considered to exhibit a higher risk of developing metabolic syndrome (MetS), with published data indicating that both conditions frequently co-exist. However, the strength and consistency of these data across epidemiological study designs remain unclear. The present analysis pools the odds ratio of MetS in adults with polysomnography-confirmed OSA compared to controls without OSA, based on case-control studies identified within the context of a broader systematic review.

We conducted a systematic review of peer-reviewed primary research articles published in English until November 2024 from four databases (Ovid Medline, CINAHL, Embase, and Cochrane). A total of 81 primary studies, including case-control, cross-sectional, cohort study designs, and randomised clinical trials, were eligible for inclusion and data extraction. Herein, we meta-analysed the identified seven eligible case-control studies for this interim evidence synthesis. Meta-analysis was conducted using a random-effects model, and the summary effect estimate is reported. Publication bias was assessed using funnel plot and Egger's test. The protocol is registered in PROSPERO 2025 CRD420251073055.

The seven case-control studies comprised 477 OSA cases and 425 controls (predominantly males with age ranging between 45 and 50 years). Adults with OSA had almost 3.5-fold higher odds of having MetS compared to their counterparts without the disease (OR = 3.47; 95% CI: 1.63 - 7.39) (figure 1). Moderate heterogeneity was evident across these studies ($I^2$ = 66.5%, Q (6) = 17.9, p < .01). No publication bias was detected.

The present findings suggest that adults with OSA have a significantly higher risk of MetS. Despite the strength of these findings, interpretation should also be cautious due to the limited number of studies and their observational design. Additional analyses including all identified study designs are planned, and will be presented in a systematic review and three-level meta-analysis.

## S24 PREDICTING DRIVER SLEEPINESS IN OSAHS: TOWARDS A SCREENING TOOL FOR ROAD SAFETY

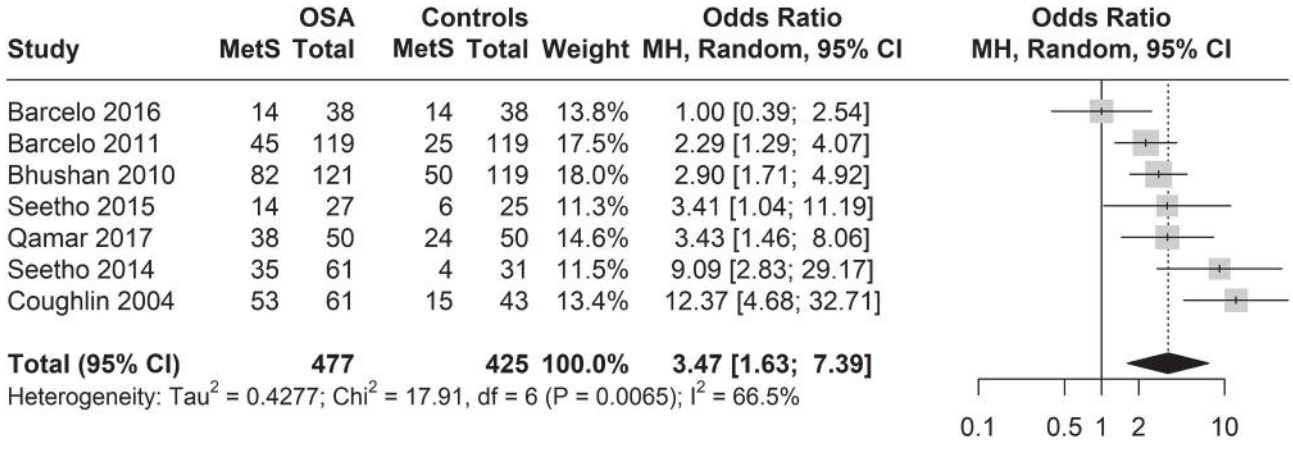
A Kumar, S Craig, B Chakrabarti, A Manuel, G Phillips. *Liverpool University Hospital NHS Foundation Trust, Liverpool, UK*

10.1136/thorax-2025-BTSabstracts.30

**Background** Obstructive Sleep Apnoea Hypopnea Syndrome (OSAHS) is an underdiagnosed condition that contributes to excessive daytime sleepiness. Sleepiness contributes to 20% of road traffic accidents. Prompt treatment of OSAHS has been shown to reduce risk of road traffic accidents as well as work-place accidents. Earlier diagnosis can also help reduce healthcare costs and improve quality of life for our patients.

**Objective** To compare the clinical and historical characteristics of drivers who self-reported sleepiness when driving versus those who did not and to identify characteristics of excessive sleepiness leading to earlier diagnosis and intervention.

**Methods** We retrospectively analysed data from our database looking at all drivers who had completed initial review and CPAP compliance between 2022–2025. 827 drivers were identified. Of these 11% (n=95) admitted to self-reported episodes of excessive sleepiness when driving. We compared demographic, clinical and sleep-related characteristics between




**Abstract S23 Figure 1**

the two groups using data from the initial review, sleep study results and blood tests.

**Results** Sleepy drivers had a significantly higher Epworth Sleepiness Scale (ESS), with a mean ESS of 15.2 vs 9.3 ($p<0.001$) and were more likely to report sleepiness in traffic ($p<0.001$). Seven symptoms were significantly more prevalent in the sleepy group. These were: Morning headaches ($p<0.001$), Depression ($p=0.002$), Difficulty remaining asleep ($p<0.00001$), Daytime sleepiness ($p<0.0001$), Difficulty concentrating ($p=0.0002$), Daytime lethargy ($p=0.00015$), and Heartburn ($p=0.00025$).

A logistic regression model incorporating these seven variables showed that individuals reporting four or more of these were twice as likely (OR = 1.987, p = 0.005) to be classified as sleepy drivers. Drivers positive on six characteristics were over seven times more likely (OR = 7.65, p = 0.052) to be classified as sleepy drivers. Whilst not statistically significant, we aim to re-test on a larger sample size.

**Conclusion** This study highlights a symptom-based profile that may help to identify drivers at risk of excessive sleepiness. Combined score of four or more characteristics shows promise as a screening tool. The findings support development of proactive algorithms or screening protocols to be used in OSAHS clinics to identify our high-risk symptomatic patients to initiate treatment promptly and to improve road safety.

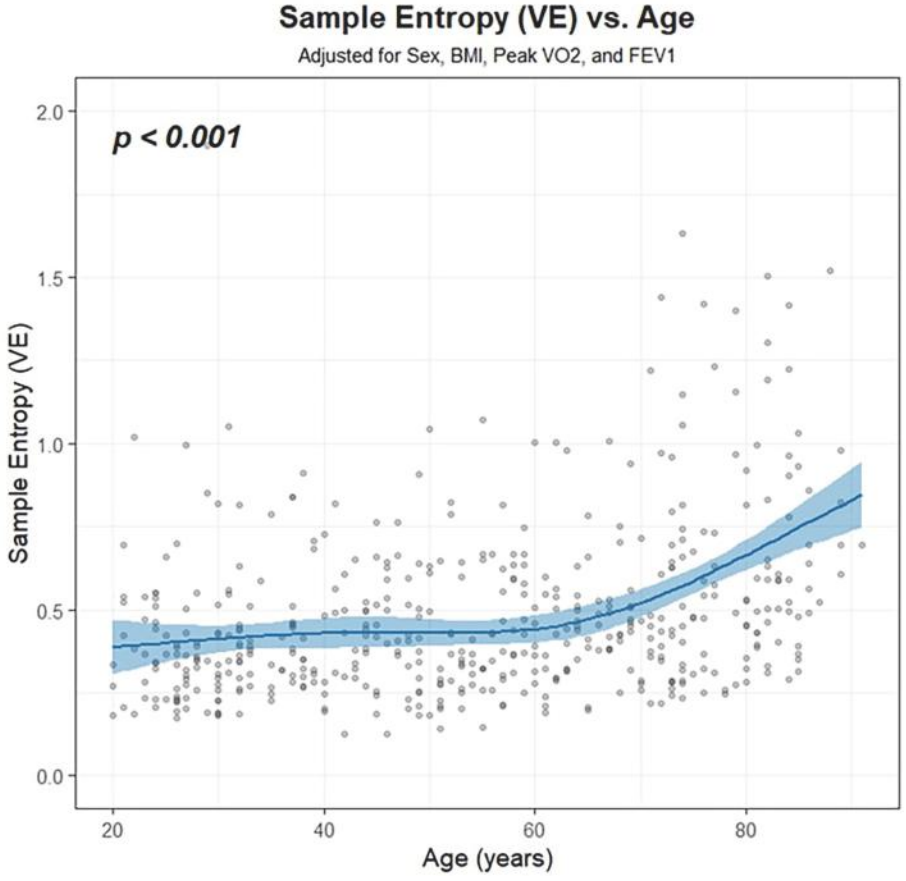
# Physiological approaches to decoding breathlessness

## S25 IS AGEING ASSOCIATED WITH EXERTIONAL VENTILATORY CONSTRAINT?

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10.1136/thorax-2025-BTSabstracts.31

**Introduction** While exertional breathlessness increases with age, its underlying mechanisms remain poorly understood. In many physiological systems, ageing leads to less variable and more constrained patterns. We hypothesized that ageing leads to a more constrained ventilatory pattern during exercise. We utilized non-linear statistical modelling (Sample Entropy – SampEn) to characterize the ventilatory response to exercise in a study of healthy ageing.




**Abstract S25 Figure 1**

**Methods** We analysed data from participants in the COmPLETE study who underwent an incremental cardiopulmonary exercise test to exhaustion. SampEn for minute ventilation (VE), breathing frequency (Bf) and tidal volume (VT) were calculated. The relationship between ventilatory entropy parameters and age was assessed using multiple linear regression models with penalized cubic splines for age, adjusting for sex, BMI, peak oxygen uptake, and FEV1.

**Results** A total of 526 healthy adults (mean age 54 [range 20–92]; 52% female) were included in the analysis.

Contrary to our hypothesis, the relationship between age and ventilatory complexity was significantly non-linear ($p < 0.001$ for VE). The trajectory showed that the SampEn VE remained stable through young adulthood and middle age, before increasing progressively from approximately 60 years onwards.

This pattern of increasing complexity in later life was consistent for both breathing frequency (Bf) ($p=0.023$) and tidal volume (VT) ($p=0.004$), indicating the ventilatory pattern becomes more irregular with advanced age.

**Conclusions** In healthy ageing, the ventilatory pattern during exercise does not become more constrained but rather significantly more complex after the sixth decade of life. Therefore, future studies should focus on validating Sample Entropy as a tool to better assess breathing pattern irregularity.

## S26 EVALUATION OF AN AUTOMATED SOFTWARE APPLICATION VERSUS CLINICIAN INTERPRETATION OF CARDIOPULMONARY EXERCISE TESTS

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10.1136/thorax-2025-BTSabstracts.32

**Introduction** Cardiopulmonary exercise testing (CPET) poses challenges in obtaining consistency in exercise protocol design as well as data processing and presentation. Furthermore, collation and interpretation of results can take a physiologist and a physician up to 40 minutes. We evaluated a novel software application (enhancedCPETanalytics™. Los Angeles, USA) that processes and organises the data from CPET in less than 10 seconds and compared determinations with clinician interpretations.

**Methods** We selected 152 CPET studies performed in Cambridge. They were divided into seven diagnostic categories according to previous clinical interpretations. Data was collected using a metabolic cart (Vyntus™ CPX) and exported via the diagnostic software platform (SentrySuite™, Jaeger Medical GmbH, Hochberg, Germany) as spreadsheets with prespecified format. The data were blinded according to diagnostic category and processed in Los Angeles using the novel software application. Studies were analysed using the two methods by blinded investigators. Key numerical values were compared using Bland Altman statistics. Key categorical data were compared using Cohen's kappa statistics. This investigation was approved under a data sharing agreement between our two universities.[1 2]

**Results** The CPET studies met quality criteria in terms of protocol design and work rate control. Subjects were aged 50.9 (17.6) years and 56% were female. Statistical comparisons are shown in table 1. For numerical data we saw highly significant correlation and small mean bias by Bland Altman analysis between the two analytical methods. Identification of functional impairment, cardiovascular limitation and ventilatory limitation showed substantial to near perfect agreement by Cohen's kappa. enhancedCPETanalytics offered additional quality criteria related to data variability.

**Abstract S26 Table 1**

| | | eCPETa | Clinician | Correlation | Probability | Mean Bias | L95%CI | U95% CI |
|---|---|---|---|---|---|---|---|---|
| Value | | Mean (SEM) | Mean (SEM) | r | P | | | |
| Maximum Work Rate | (watts) | 120.2 (4.5) | 116.3 (4.5) | 0.997 | <0.001 | 3.7 | 11.0 | -3.5 |
| Maximum Oxygen Uptake | (L/min) | 1.60 (0.04) | 1.65 (0.05) | 0.990 | <0.001 | -0.05 | -0.2 | 0.10 |
| Maximum Oxygen Uptake | (ml/kg/min) | 20.4 (0.6) | 21.1 (0.6) | 0.990 | <0.001 | -0.9 | -4.8 | 3.1 |
| Metabolic efficiency | | 9.7 (0.1) | 9.7 (0.1) | 0.755 | <0.001 | 0.0 | -1.8 | 1.8 |
| Metabolic Threshold | (L/min) | 0.89 (0.02) | 0.95 (0.02) | 0.852 | <0.001 | -0.06 | -0.34 | 0.22 |
| Metabolic Threshold | (ml/kg/min) | 11.3 (0.3) | 12.1 (0.3) | 0.791 | <0.001 | -0.8 | -5.4 | 3.8 |
| Maximum Ventilation | (L/min) | 68.1 (1.9) | 68.3 (2.0) | 0.988 | <0.001 | -0.3 | -7.5 | 7.0 |
| Ventilatory Efficiency | | 31.5 (0.6) | 34.8 (0.5) | 0.661 | <0.001 | -3.4 | -14.4 | 7.7 |
| Maximum Heart Rate | (/min) | 148 (2) | 149 (2) | 0.970 | <0.001 | -2 | -14 | 11 |
| Chronotropic Index | (/L) | 62 (2) | 58 (2) | 0.853 | <0.001 | 4 | -23 | 31 |

| | | eCPETa | | Clinician | | Chi-squared | Probability | Kappa |
|---|---|---|---|---|---|---|---|---|
| Category | | 0 | 1 | 0 | 1 | $X^2$ | P | k |
| Techinical Comments: Protocol | | | | | | | | |
| Optimal test duration | 0=no; 1=yes | 53 | 99 | 66 | 86 | 3.596 | 0.058 | 0.356 |
| Appropriate increase in oxygen uptake | 0=no; 1=yes | 18 | 134 | 21 | 131 | 0.600 | 0.439 | 0.559 |
| | | | | | | | | |
| Technical Comments: Measured Variables | | | | | | | | |
| Maximum Work Rate | 0=normal; 1=abnormal | 108 | 44 | 48 | 104 | 58.065 | <0.001 | 0.294 |
| Metabolic efficiency | 0=normal; 1=abnormal | 125 | 27 | 132 | 20 | 3.266 | 0.071 | 0.624 |
| Aerobic Capacity | 0=normal; 1=abnormal | 99 | 53 | 85 | 67 | 5.158 | 0.023 | 0.481 |
| Metabolic Threshold | 0=normal; 1=abnormal | 104 | 44 | 109 | 39 | 0.926 | 0.336 | 0.549 |
| Functional Impairment | 0=no; 1=yes | 39 | 113 | 36 | 116 | 1.286 | 0.257 | 0.876 |
| Ventilatory limitation | 0=no; 1=yes | 16 | 136 | 32 | 120 | 16.000 | <0.001 | 0.612 |
| Ventilatory efficiency | 0=normal; 1=decreased | 107 | 45 | 72 | 80 | 26.064 | <0.001 | 0.395 |
| Cardiovascular limitation | 0=no; 1=yes | 98 | 54 | 88 | 64 | 5.556 | 0.018 | 0.752 |
| Chronotropic index | 0=normal; 1=abnormal | 118 | 34 | 108 | 44 | 2.174 | 0.140 | 0.211 |

**Conclusions** Close agreement was seen for all numerical variables and key categorical variables when comparing the novel software application with clinician interpretation. In addition, the software application has the ability to minimise subjectivity in analysis, standardise testing interpretation and improve reporting time-efficiency. These are qualities much needed for the wider acceptance and recognition of the clinical usefulness of CPET.

## REFERENCES


1. Cooper CB. Exercise testing does not have to be complicated. *Chron Respir Dis*. 2006;**3**:107–108.
2. Ward SA. Exercise testing needs to be comprehensive. *Chron Respir Dis*. 2006;**3**:109.


## S27 THE USE OF CARDIOPULMONARY EXERCISE TESTS IN OBJECTIVELY DIAGNOSING DYSFUNCTIONAL BREATHING

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10.1136/thorax-2025-BTSabstracts.33

**Introduction** Dysfunctional breathing (DB) is a recognised contributor to breathlessness, occurring as the main cause or a contributing cause where other comorbidities exist. Cardiopulmonary exercise testing (CPET) can be used to assess for DB via pattern recognition by a CPET expert, but it has been suggested previously that numerical outputs could be used as an objective measure requiring less expertise.

**Aim** To ascertain if there are objective measures from CPET outcomes that can be used to diagnose DB.

**Methods** Retrospective CPET outcomes for patients referred to Respiratory Physiology between January 2020 and January 2025 were searched for using 'DB', Dys', 'breath' and 'SOB' in the patient query program. Those without peak blood gas measurement or with known cardiac, vascular or ventilatory disorders were removed. Demographic data was taken from the patient's records and CPET outcomes related to ventilation (peak VO2, VE/VCO2 slope, VE/VCO2eq at 40watts, PaCO2, PETCO2, respiratory rate (RR), P(a-ET)CO2, Vd/Vt, P(A-a)O2 gradient and breathing reserve (BR)) were compared to expert opinion using threshold values (30 or 35 for VEVCO slope depending on age, or 35 for VE/VCO2eq) or by analysing continuous data. Mann Whitney U, T-test and Chi square statistics were performed. ROC curve analysis was performed for VE/VCO2eq at 40w.

**Results** 269 patient results were returned and 65 fulfilled inclusion criteria. Cardiovascular impairment was found in 25/65. 59% were diagnosed with DB, 39% of which had no other abnormalities. There were no significant differences in any variable between those with and without DB. Thresholds for VE/VCO2 slope and VE/VCO2eq at 40watts did not identify those with DB. ROC curve analysis gave an AUC of 0.632 for VEVCO2 at 40watts with a sensitivity of 63.2% and specificity of 63.0% using a threshold of 35. When only including those with DB alone, AUC reduced to 0.409 (40% sensitivity and 44% specificity) at a threshold of 35.

**Conclusions** Individual CPET outcomes did not distinguish between those with and without DB in this cohort. Assessment by DB type (hyperventilation or breathing pattern disorder) alongside analysing combinations of CPET outcomes could be useful in a larger cohort.

## S28 FACTORS ASSOCIATED WITH BREATHLESSNESS AND DYSREGULATED BREATHING POST-COVID-19 AMONGST REFERRALS TO A RESPIRATORY CLINIC

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10.1136/thorax-2025-BTSabstracts.34

**Introduction** Persistent respiratory symptoms after SARS-CoV-2 infection are common even in those who were not hospitalised. A local registry for COVID-19 recovery research was created to profile individuals referred to a respiratory clinic with persistent respiratory symptoms and to identify associated factors. This report details a cross-sectional analysis of referred patients who consented.

**Methods** Participants (mean age: 49.4 years, 68% female) were 210 non-hospitalised adults referred to a post COVID-19 respiratory clinic with respiratory symptoms. Demographics, symptoms, and questionnaires to assess breathlessness (MRC dyspnoea scale grade $\geq$ 3), dysregulated breathing (Nijmegen score > 23), fatigue (Chalder Fatigue total score $\geq$ 4), and anxiety and depression (Hospital Anxiety and Depression Scale [HADS] scores $\geq$ 8) were collected. Multivariable logistic regression models examined predictors of breathlessness and dysregulated breathing.

**Results** Shortness of breath was the most commonly reported persistent respiratory symptom (95%), followed by dry and productive cough. Other common persistent symptoms included fatigue, low mood, muscle aches, anxiety and malaise, with many patients reporting multiple symptoms. The majority of participants showed indications of breathing dysregulation (68%), anxiety (64%), depression (53%), and poor sleep quality (85%). Higher body mass index (adjusted odds ratio = 1.10, 95% confidence interval = 1.05 to 1.16) and a HADS-Depression score $\geq$ 8 (2.25, 1.13 to 4.56) were associated with breathlessness. Factors significantly associated with a higher risk of dysregulated breathing (Nijmegen score > 23) were female sex (3.69, 1.79 to 7.78), current smoking (9.96, 1.72 to 191.0), fatigue (8.54, 2.49 to 35.70), a HADS-Anxiety score $\geq$ 8 (3.48, 1.65 to 7.53) and a HADS-Depression score $\geq$ 8 (5.88, 2.67 to 13.80).

**Conclusion** Higher BMI, current smoking, fatigue, anxiety, and depression at assessment were linked with breathing dysregulation and/or breathlessness post-COVID. These findings underscore the need for integrated pathways that use a multidisciplinary and personalised approach to optimise recovery.

## S29 PERIPHERAL MUSCLE OXYGEN EXTRACTION IS COMPROMISED DURING SUBMAXIMAL EXERCISE IN PREVIOUSLY HOSPITALIZED COVID-19 SURVIVORS

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10.1136/thorax-2025-BTSabstracts.35

**Background** Compared to age-matched healthy individuals, people with post-COVID syndrome exhibit profoundly

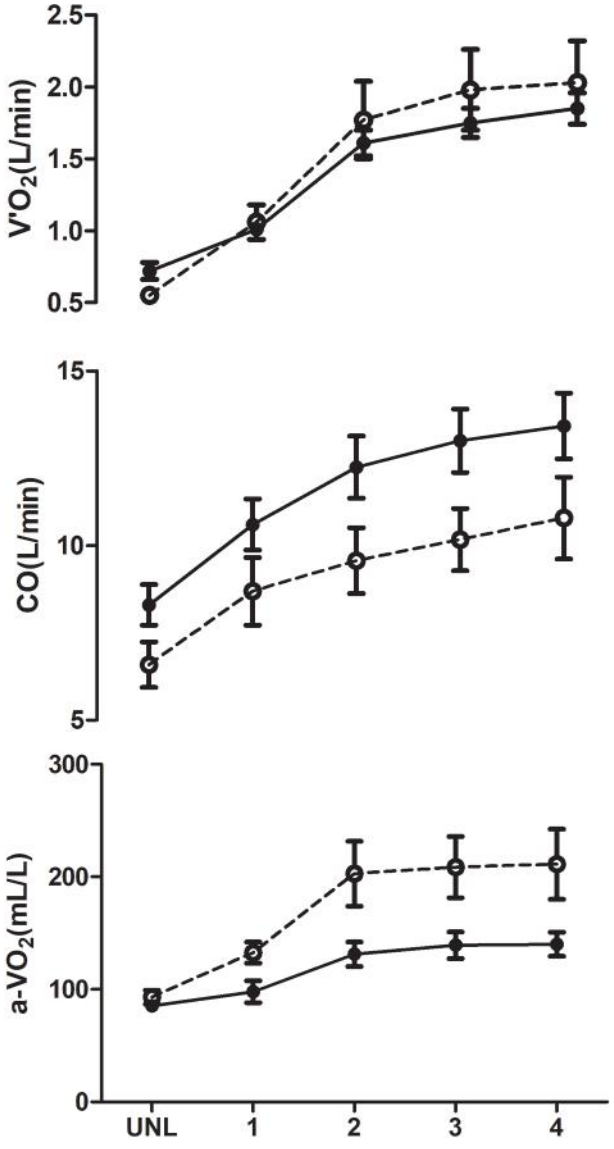
reduced exercise tolerance, secondary to reduced aerobic capacity.

**Aims** To determine whether reduced aerobic capacity in previously hospitalised COVID-19 survivors is due to the diminished central cardiovascular output and/or due to the reduced capacity of the peripheral muscles to extract oxygen from the circulation during exercise.

**Methods** Twelve previously hospitalised COVID-19 survivors (mean age: 54±9 yrs) and seven age-matched healthy controls (58±11 yrs) underwent two exercise testing laboratory visits. First, an incremental cycling test to their maximum tolerable workload to determine peak work rate (PWR). On a subsequent visit, participants undertook a single bout of constant-load exercise (CLE) sustained at a load corresponding to 80% of each participant's PWR. The CLE test was preceded by 3 minutes of unloaded pedalling to warm up. Cardiac output (CO), heart rate, stroke volume (assessed by impedance cardiography), oxygen uptake ($VO_2$), and other respiratory parameters, including indices of ventilatory efficiency ($V_E/VO_2$ and $V_E/VCO_2$), were monitored throughout exercise. Arteriovenous oxygen content difference (a-$vO_2$), which reflects how much oxygen is extracted from the blood by the muscles during exercise, was calculated by rearranging the Fick equation where $VO_2$ = CO x (a-$vO_2$).

**Results** Two-way ANOVA with repeated measures showed that compared to healthy individuals, COVID-19 survivors exhibited lower (p=0.035) $VO_2$ during CLE (figure 1). Whilst CO was greater (p=0.001) in COVID-19 survivors compared to healthy controls, a-$VO_2$ was lower (p=0.01) during exercise in COVID-19 survivors compared to healthy participants (figure 1). Ventilatory efficiency indices were not different between groups.

**Conclusions** Whilst central cardiovascular output and ventilatory efficiency were preserved in COVID-19 survivors during submaximal exercise, peripheral muscle oxygen extraction was diminished compared to healthy participants most likely reflecting compromised muscle vasculature and/or impaired muscle fibre oxidative capacity.




**Abstract S29 Figure 1** Data points (mean ±SEM) are shown for unloading pedalling (UNL) and for each of the 4-min of exercise in people with post-COVID syndrome (closed circles) and healthy participants (open circles)

## S30 OXYGEN REQUIREMENT RISES EARLIER THAN THE TOTAL NATIONAL EARLY WARNING SCORE 2 (NEWS2) SCORE PRIOR TO DETERIORATION

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10.1136/thorax-2025-BTSabstracts.36

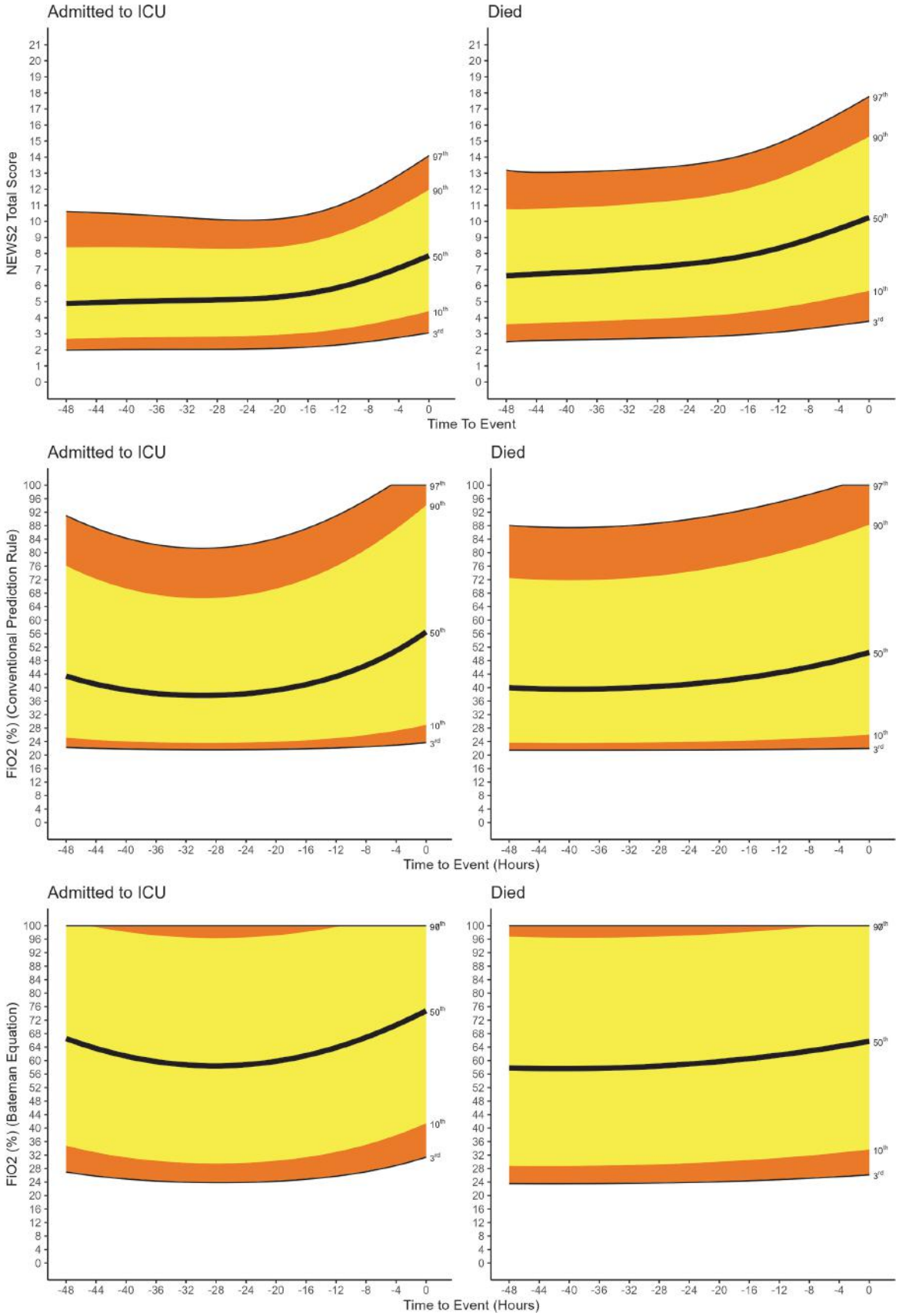
**Introduction** The National Early Warning Score 2 (NEWS2) assigns 0 points for room air and 2 points for any oxygen therapy. This binary scoring may miss deteriorating patients with an escalating oxygen requirement and otherwise stable observations. Although this limitation was recognised during COVID-19, it remains unexplored in general inpatients.

**Aims**

1. Describe the trajectories of fraction of inspired oxygen (FiO2) and NEWS2 in the 48 hours prior to deterioration in adults on oxygen.
2. Describe the trajectories of vital signs (heart rate, respiratory rate, oxygen saturations, temperature, systolic and diastolic blood pressure) 48 hours before discharge or deterioration.

**Methods** A retrospective cohort study using data from a tertiary NHS foundation trust, composed of four hospitals, including a university teaching hospital. We included adults admitted for > 24 hours with at least one full set of vital signs. We estimated FiO2 via the Bateman equation and conventional prediction rule (see[1]). First, we modelled changes in NEWS2 score and FiO2 48 hours prior to unplanned intensive care unit (ICU) admission or death in patients on oxygen. Second, we modelled vital sign changes before discharge or deterioration (unplanned ICU admission or death) in all patients.

**Results** There were 227,812 admissions and 1,532,498 observation sets within 48 hours of discharge or deterioration. 43,662 admissions required oxygen, contributing 178,093 observation sets. 7791 admissions deteriorated.




**Abstract S30 Figure 1** Trajectories of NEWS2 score (top), $FiO_2$ (estimated via the conventional prediction rule) (middle), and $FiO_2$ (estimated via the Bateman equation) (bottom) in the 48 hours prior to unplanned ICU admission or death, in patients on oxygen

In patients on oxygen before ICU admission, FiO2 began rising ~24 hours prior, whereas NEWS2 score rose 12–16 hours before. In those who died, FiO2 rose more gradually ~16–20 hours before death, with NEWS2 increasing 12–16 hours before death (figure 1).

In all patients, all vital signs stabilised before discharge and worsened before deterioration, especially respiratory rate. However, the magnitude of vital sign derangement varied greatly between observations.

**Conclusions** A rise in FiO2 preceded a rise in NEWS2 score by 8–12 hours in patients requiring unplanned ICU admission, and 4–8 hours in those who died. Incorporating FiO2 into NEWS2 may allow for earlier recognition of deterioration.

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# Optimising diagnostics in thoracic malignancy

## S31 EARLY INSIGHTS INTO THE DATA FROM THE ARTIFICIAL INTELLIGENCE AND BIG DATA FOR EARLY LUNG CANCER DIAGNOSIS PROSPECTIVE STUDY (PHASE 2) (IDEAL STUDY)

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10.1136/thorax-2025-BTSabstracts.37

**Introduction** Malignancy risk assessment of incidental pulmonary nodules (IPNs) is key to identifying early-stage lung cancer. **IDEAL**, a multi-centre prospective observational study, assessed the utility of an AI algorithm in patients with solid IPNs.

Here we report the overall diagnostic performance of the AI algorithm and compare its performance to the Brock model.

**Methods** Participants aged ≥18 years with fewer than five 5–15 mm solid IPNs identified on CT were prospectively recruited from four NHS hospitals between 08/2018 and 02/2022. Patients with ground-glass opacities and history of malignancy within five years were excluded.

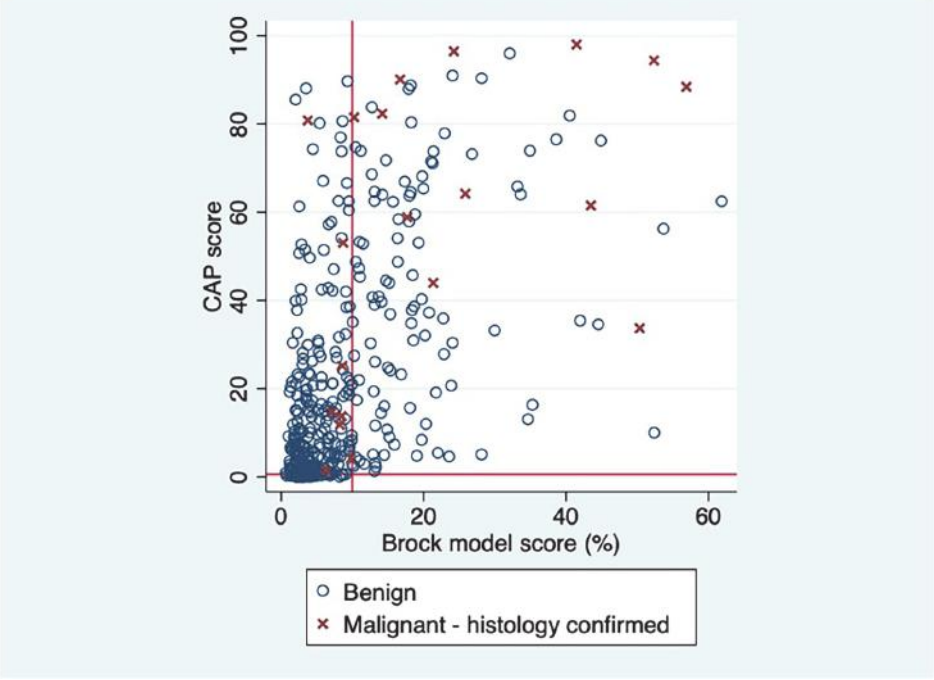
Management of nodules was as per the 2015 BTS Pulmonary Nodule Guidelines. Final diagnosis was benign if the nodule was classified as categorically benign on CT requiring no follow-up or has completed surveillance as per BTS; or malignant if proven histologically. The AI provided a CAP score on selected IPNs.

Brock score threshold of 10% was compared to a predetermined rule out AI threshold risk of 0.56334234.

**Results** 1056 patients with 1594 nodules were included. 24/1056 (2.27%) patients had histologically-proven malignant nodules with one patient having two synchronous malignancies. Of the 25/1594 (1.57%) malignant nodules, 23 were primary lung cancers; adenocarcinoma was the most common.

404 nodules (20 malignant) were selected for analysis: these were solid, ≥8 mm in diameter (as per BTS). The Area Under the Curve (AUC) for the algorithm was 79.3% (95%CI 68.54%–90.05%), compared with 79.56% (95%CI 71.01%–88.08%) for Brock (p=0.995).

For the algorithm, there were 42 nodules (0 malignant) below and 362 nodules (20 malignant) above the predetermined threshold. For Brock, there were 290 nodules (8 malignant) below and 114 nodules (12 malignant) above the 10%




**Abstract S31 Figure 1** Scatterplot illustrating the relationship of CAP score and Brock model score of the 404 nodules included in the analysis. The reference lines are pre-defined rule-out thresholds

threshold. When comparing at equivalent sensitivities, i.e. ruling out the same number of benign nodules as Brock below 10% (282 nodules), CAP would exclude 6 malignant nodules rather than 8 as with Brock.

**Conclusion** This is the first study testing the utility of AI for 5–15 mm IPNs prospectively against current standard of care. Despite a comparable AUC with Brock, AI appears to more reliably identify benign nodules without requiring any clinical information, in comparison to the Brock model.

## S32 CLINICAL READINESS OF AI TUMOUR VOLUME QUANTIFICATION IN MESOTHELIOMA: RESULTS OF THE MESO-Q STUDY

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10.1136/thorax-2025-BTSabstracts.38

**Objectives** We report on the clinical readiness of an AI tool for volumetric tumour quantification in pleural mesothelioma (PM) developed in the multicentre Mesothelioma detection and Quantification study, part of the PREDICT-Meso International Accelerator network.

**Methods** We pre-specified a primary endpoint of 'clinical acceptability', requiring $\geq$90% of AI segmentations to be scored as 'Excellent' or 'Satisfactory' on a 5-point Likert scale and assembled a retrospective cohort of CT scans and clinical data, describing 1851 patients from 9 UK hospitals. 706/1851 had full metadata; 414/1851 had follow-up CT scans following therapy. 3 sites were held out for validation and to assess generalisation. Correlation and agreement (human edited vs. AI) were assessed using Dice overlap, Intra-Class Correlation (ICC) and Bland-Altman limits. AI- and human-segmented tumour volume (TV) were used to stratify patient response and compared using concordance index. Baseline AI-generated TVs were used to stratify overall survival using Cox proportional hazards models.

**Results** Following full annotation of 353 scans (~422 CT slices per scan; >174,000 slices total), the primary objective was met. 90.8% of scans were deemed clinically acceptable in an external validation set of 98 scans from 9 different hospitals, with no performance difference in held-out scans, indicating good generalisation. 98% were considered 'acceptable starting points', needing no more than minor modifications to become clinically acceptable. The optimised network achieved Dice=0.95 and ICC=0.98, with non-significant mean bias of 28 cm3, suggesting edits did not meaningfully change the nature of the segmentation. Using mRECIST response cutoffs ([-30%,20%]), AI-derived TV outperformed human-segmented TV in 36 baseline-response CT pairs (concordance index=0.56 vs. 0.52). AI-derived TV stratified survival (414 paired CT scans) when dichotomised around the median volume (HR=0.68,p<0.001) in Cox proportional hazards models. Agreement on segmentation quality was perfect ($\kappa$=1). Clinical acceptability appeared independent of CT manufacturer and age (p=0.1844,p=0.1335).

**Conclusion** These data demonstrate the clinical readiness of a fully-automated AI tool for PM segmentation. The optimised network generalises well to CT scans acquired on different scanners across multiple centres. AI-generated TV outperforms human-generated TV for response assessment and has important prognostic implications. Ongoing work includes tuning of optimal volumetric thresholds for response assessment.

## S33 HARNESSING EXHALED BREATH FOR LUNG CANCER EARLY DETECTION. THE EXPEL STUDY RESULTS

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10.1136/thorax-2025-BTSabstracts.39

Lung cancer causes more deaths than any other cancer because it is diagnosed late. The NLST and NELSON studies have demonstrated that lung cancer screening by CT scans reduce mortality from lung cancer by introducing a shift in diagnosis towards early-stage disease. However, the service provision of large-scale CT scanning is expensive and therefore can only be offered to people at the very highest risk. This means that only 40% people with lung cancer would have been eligible for CT screening prior to their diagnosis. Therefore, even with perfect regional coverage, lung cancer screening will not reach most people at risk. New tests are needed to expand screening to the wider population. Exhaled breath tests are attractive because they are non-invasive, with potential for community application. To this end, higher levels of exhaled reactive oxygen species (ROS) have been associated with lung cancer. However, translation to clinical practice has been limited by the high prevalence of other chronic diseases in people who smoke, which independently increase ROS. Recently, the VICTORY study[1] demonstrated that using a point-of-care device called Inflammacheck® to measure ROS and multiple breath parameters could distinguish different chronic respiratory conditions including lung cancer by using a machine learning algorithm. The ExPeL study (IRAS 336691) has demonstrated that this approach can be applied to populations relevant to screening. Lung cancer cases (n=23, 83% early stage) were separated from controls (n=11), consisting of ever smokers who were eligible for lung cancer screening, with 100% specificity and 80% sensitivity. Furthermore, ExPeL also included an exploratory mass spec analysis of exhaled breath condensate in lung cancer cases (n=19) and controls (n=23), which has identified four new molecules associated with lung cancer that could be incorporated into Inflammacheck® in the future to improve performance.

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## S34 DOES PROFILING PLEURAL FLUID CIRCULATING TUMOUR DNA (CTDNA) HAVE A ROLE IN IMPROVING DIAGNOSTIC YIELD IN MALIGNANT PLEURAL EFFUSION?

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10.1136/thorax-2025-BTSabstracts.40

**Introduction** Diagnostic yield of pleural fluid (PF) cytology is limited, with an overall sensitivity of 58.2%[1]; often necessitating invasive procedures to obtain histology. Profiling circulating tumour DNA (ctDNA) from PF represents a promising strategy for diagnosing malignant pleural effusion (MPE). This proof-of-concept study evaluated the potential of profiling PF ctDNA to improve diagnostic accuracy using a rapid and cost-effective shallow whole-genome sequencing (sWGS) approach.

**Methods** At time of writing, matched PF and blood samples were collected from five patients with confirmed (n=2) or suspected (n=3) thoracic malignancies. PF samples were also submitted for standard cytological evaluation. Cell-free DNA (cfDNA) was extracted from PF and plasma and subjected to sWGS (300pg) using previously described methods.[2 3] Results were analysed using a tailored ichorCNA pipeline to detect genome-wide copy number (CN) aberrations indicative of ctDNA. Patient recruitment is currently ongoing.

**Results** All 5 patients had a malignant diagnosis, PF cytology confirmed MPE in 2 (40%) patients. Two patients required a biopsy for diagnosis (1 ultrasound guided, 1 thoracoscopy) and 1 patient underwent EBUS (see table 1). sWGS analysis identified ctDNA (CN deletions) in PF in all patients, whereas plasma only showed deletions in 3 patients (60%). Notably, deletions in PF ctDNA involved chromosomal regions harbouring key lung cancer-associated genes, including *RB1*, *SMARCA4* and *CDKN2A*.

**Discussion** We identified presence of ctDNA in PF samples through CN aberrations, potentially offering evidence of malignancy using a rapid, cost-effective methodology. Future work to increase sample size and profile actionable genomic alterations that may inform therapeutic strategies is warranted.

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## S35 REDUCING REPEAT PLEURAL BIOPSIES IN SUSPECTED MESOTHELIOMA (REPLICA)

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10.1136/thorax-2025-BTSabstracts.41

**Background** Pleural mesothelioma (PM) can be challenging to diagnose. Some patients require multiple invasive procedures with associated delays before diagnosis. The TARGET study showed that PET-CT-guided biopsy did not improve diagnostic yield compared with CT in patients with initial non-diagnostic biopsies.[1] Newer immunohistochemical (IHC) tests such as BAP1 and MTAP were not available at the time of TARGET. REPLICA aimed to use these newer IHC tests to determine whether performing BAP1 and MTAP IHC on historical samples could have increased the diagnostic yield.

**Methods** Participants with suspected pleural malignancy and an initial non-diagnostic biopsy were recruited to the TARGET trial between 10/11/2015–19/09/2018. For REPLICA, BAP1 and MTAP IHC were performed on the original non-diagnostic and repeat biopsies for all PM diagnoses within TARGET. Histological analysis including MTAP1 and MTAP staining was performed independently by two thoracic pathologists. Diagnostic yields and time to diagnosis were compared with and without BAP1 and MTAP IHC.

**Results** We present the results of the first 21 patients, with a total of 50 biopsies. Participant characteristics were as expected (19; 90.5% male; median age 75 (70–83 IQR), 14; 67% epithelioid). 10/21 (47.6%) patients would have achieved a diagnosis after one biopsy, and a further 10/21 on second biopsy. This would have resulted in fewer biopsies overall (31 *vs* 50,p=0.0005). The addition of BAP1 and MTAP IHC would have reduced time to diagnosis (median 22 *vs* 71 days, p=0.0005), and required fewer diagnostic MDT discussions (31 *vs* 52,p=0.0005) with a potential biopsy-related cost saving of £741.90 per patient.

**Conclusions** This study demonstrates the potential benefit of BAP1 and MTAP IHC in diagnosing PM. These additional tests have the potential to reduce multiple biopsies and shorten time to diagnosis. Access to BAP1 and MTAP IHC should be available within every cancer network.

**Abstract S34 Table 1** Underlying diagnosis, PF cytology and method of final diagnosis

| Diagnostic or Confirmed | Cytology | Underlying diagnosis | Method diagnosis made |
|---|---|---|---|
| Confirmed | NSCLC | NSCLC - adenocarcinoma | Pleural fluid cytology |
| Confirmed | Negative | NSCLC - NOS | Pleural biopsy |
| Diagnostic | Negative | Sarcamatoid carcinoma | Thoracoscopy |
| Diagnostic | Negative | Diffuse B cell lymphoma | EBUS |
| Diagnostic | Metastatic malignant epithelioid tumour | Unknown origin - CK7 positive sarcoma/carcinoma - SMARCA4 | Pleural fluid cytology |

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## S36 THE STREAMLINE FEASIBILITY RANDOMISED CLINICAL TRIAL (EARLY PLEURAL BIOPSY + INDWELLING PLEURAL CATHETER IN SUSPECTED MALIGNANT PLEURAL EFFUSION)

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10.1136/thorax-2025-BTSabstracts.42

**Introduction** Malignant pleural effusion (MPE) affects 50 000 patients per year in the UK. Patients with MPE require multiple pleural procedures with prolonged time to diagnosis and fluid control. Thoracocentesis remains the guideline based first intervention in MPE, but has poor sensitivity and molecular completion and does not prevent effusion recurrence. This paradigm has not been challenged in over 40 years – we therefore co-created an accelerated pathway with and for patients with suspected MPE, with the aim of reducing symptom duration and number of procedures to diagnosis and definitive management.

**Aims** To assess feasibility of recruitment to a randomised trial comparing standard care (1st procedure thoracocentesis) vs accelerated pathway (1st procedure pleural biopsy + IPC) in suspected MPE.

**Methods** STREAMLINE was a UK NIHR funded multi-centre trial, opened to recruitment in January 2024. Patients were randomised 1:1 between standard care and accelerated pathways. Key eligibility criteria include: pleural effusion requiring therapeutic drainage, with suspicion of malignant cause based on imaging or history of cancer. The primary outcome was feasibility of recruitment, with key secondary outcomes including breathlessness (100 mm VAS) over 6 weeks from first procedure, quality of life (QLQ-C30), time to treatable diagnosis and adverse events.

**Results** The STREAMLINE protocol was co-created with MPE patients and expert pleural clinicians to ensure emphasis on patient and healthcare priorities. Target recruitment was completed (n=40) in June 2025. Full results including the effect on diagnostic time and symptoms will be available and presented at the Winter BTS meeting if accepted.

**Conclusion** The STREAMLINE feasibility trial will determine whether a novel direct to pleural biopsy + IPC approach is deliverable within the NHS and assess whether the accelerated pathway improves breathlessness and time to diagnosis, including molecular completion. 6 week follow up will be completed in August 2025 and results presented at the BTS contingent on selection. The results could lead to a paradigm shift in pleural management within UK healthcare systems.

# Bronchiectasis: clinical trials and real world impact

## S37 MOLECULAR DETECTION AND QUANTIFICATION OF PSEUDOMONAS AERUGINOSA IN BRONCHIECTASIS: DATA FROM THE GREAT-2 RANDOMIZED CONTROLLED TRIAL

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10.1136/thorax-2025-BTSabstracts.43

**Introduction and Objective** Molecular detection and quantification of bacteria may enhance sensitivity and responsiveness of bacteriological endpoints in clinical trials compared to culture. Here we test a *Pseudomonas aeruginosa* (PA) ribosomal RNA targeting RT-qPCR assay (RNA-qPCR) as a potential outcome measure for the GREAT-2 clinical trial.

**Methods** GRemubamab ErAdication Trial (GREAT-2) was a multi-centre, randomized, double-blind, placebo-controlled trial involving patients with CT-confirmed bronchiectasis and sputum cultures positive for PA. 37 patients were enrolled into GREAT-2 and randomised to receive either 1500 mg (n=12) or 500 mg (n=13) intravenous gremubamab—a monoclonal antibody targeting PcrV and Psl, PA virulence factors—or placebo (n=12). The primary outcome was the change in quantitative cultures of sputum samples from baseline to day 84 (end of treatment (EoT)). Quantitative culture was performed, and samples were stored in an RNA preservation buffer prior to RNA-qPCR assessment. Secondary outcomes included time to first exacerbation. Treatment arms were compared to the placebo arm through regression analysis; statistical significance was pre-specified at the one-sided $p<0.1$.

**Results** Measured by quantitative culture, the 500 mg gremubamab treatment dose led to a significant reduction in bacterial load at EoT compared with placebo ([estimate]; -1.28 log-CFU; $p=0.065$), while a non-significant reduction was observed for the 1500 mg dose (-0.67; $p=0.2$). In comparison when measured by RNA-qPCR, the 1500 mg dose led to a significant reduction in bacterial load (-1.08 log-copies/reaction; $p=0.030$); and a non-significant reduction was observed for the 500 mg dose (-0.55; $p=0.15$). RNA-qPCR findings were corroborated by another molecular method (DNA based). RNA-qPCR quantification was positively correlated with quantitative culture ([Pearson]; $r=0.42$; $p<0.001$) and with PA DNA quantification ($r=0.73$; $p<0.001$). Time to first exacerbation was significantly delayed with gremubamab at both 1500 mg ([Hazard ratio-HR]; 0.203; $p=0.023$) and 500 mg (HR 0.379; $p=0.058$) compared with placebo.

**Conclusions** Gremubamab reduced bacterial load and improved clinical outcomes in patients with bronchiectasis at both doses. RNA-qPCR identified a greater reduction in bacterial load at the 1500 mg dose which corresponded with clinical benefit suggesting the potential utility of molecular methods as an outcome measure in clinical trials.

## S38 EFFECTS OF BRENSOCATIB ON NEUTROPHIL SERINE PROTEASE LEVELS IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: A PHARMACODYNAMIC ANALYSIS OF THE ASPEN TRIAL

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10.1136/thorax-2025-BTSabstracts.44

**Background** Neutrophilic inflammation plays a key role in the pathophysiology of non-cystic fibrosis bronchiectasis (hereafter bronchiectasis). Neutrophil serine proteases (NSPs), such as neutrophil elastase (NE), cathepsin G (CatG), and proteinase 3 (PR3), are associated with disease progression and poorer clinical outcomes. Brensocatib, an oral, selective, competitive, and reversible inhibitor of dipeptidyl peptidase 1, prevents activation of NSPs. In the phase 3 ASPEN trial (NCT04594369), brensocatib 10 mg and 25 mg significantly reduced pulmonary exacerbation (PEx) burden vs placebo (annualized rate, time to first, increased odds of remaining PEx-free); Brensocatib 25 mg significantly reduced lung function decline and nominally significantly improved patient-reported symptoms. We report pharmacodynamic substudy data on the effect of brensocatib vs placebo on sputum NSP concentrations over treatment and follow-up periods in adults from ASPEN.

**Methods** In ASPEN, adults with ≥2 PEx in the 12 months before screening and were randomised 1:1:1 to once-daily brensocatib (10 mg or 25 mg) or placebo for 52 weeks. Sputum samples were collected at screening and pre-dosing at baseline and weeks 4, 16, 28, 40, 52, and 56 (4 weeks post-treatment). The pharmacodynamic analysis set includes adults who consented to the substudy, received ≥1 dose of study drug, and ≥1 pre-dose and post-dose biomarker measurement.

**Abstract S38 Table 1** Percent change from baseline in sputum concentrations of active neutrophil serine proteases in adult patients at week 52 (end of treatment)

| | | Median |
|---|---|---|
| NE | Brensocatib 10mg | –33.7% |
| | Brensocatib 25mg | –41.2% |
| | Placebo | –4.6% |
| CatG | Brensocatib 10mg | –39.7% |
| | Brensocatib 25mg | –48.1% |
| | Placebo | 0.0% |
| PR3 | Brensocatib 10mg | –27.7% |
| | Brensocatib 25mg | –58.4% |
| | Placebo | 0.0% |

CatG, cathepsin G; NE, neutrophil elastase; PR3, protease 3

**Results** ASPEN enrolled 1680 adults. The pharmacodynamic analysis set included brensocatib 10 mg n=105; brensocatib 25 mg n=108; placebo n=115. Baseline characteristics were consistent across treatment groups and with the overall population. All treatment groups displayed high inter-patient variability in baseline levels of active sputum NE, CatG, and PR3. Percent of baseline activity in NE, CatG, and PR3 decreased by week 4 in brensocatib 10 mg and 25 mg, with no appreciable change in placebo. These trends were maintained throughout treatment for all NSPs (table 1), with values returning toward baseline by 4 weeks post-treatment.

**Conclusions** Sputum pharmacodynamic analyses showed that, consistent with the phase 2 WILLOW trial, brensocatib treatment resulted in a dose-dependent reduction of NSP activity within 4 weeks. NSP activity was stably suppressed throughout the treatment period and increased toward baseline after 4 weeks off-treatment. Furthermore, brensocatib 25 mg showed a more pronounced reduction of sputum NSP activity than the 10 mg dose.

## S39 POLYPHARMACY IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: THE BRONCHUK OBSERVATIONAL STUDY

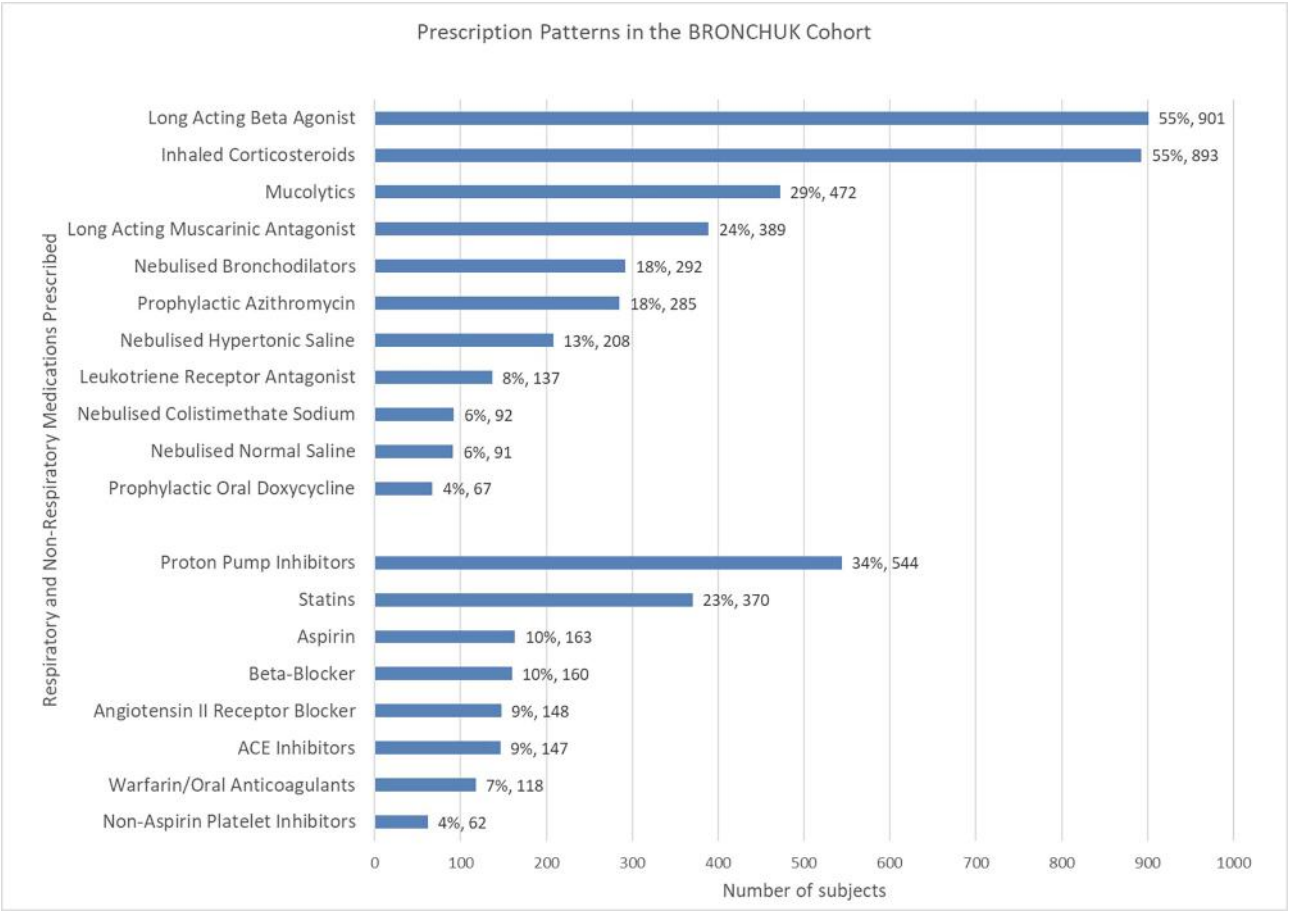
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**Introduction** BRONCHUK is a multicentre, prospective, observational cohort study enrolling adults with radiologically confirmed bronchiectasis across secondary centres in the UK. The objectives of the study are to develop a multicentre bronchiectasis registry incorporating baseline data collection with annual follow-up data for at least 5 years and to describe treatment patterns across the UK.

Polypharmacy is defined as the concurrent use of ≥5 medications and has been rarely documented in literature for bronchiectasis patients. Polypharmacy has been associated with increased risk of adverse drug reactions and increased treatment burden. The current BTS guidelines have not detailed the impact of polypharmacy in bronchiectasis patients.

**Methods** Adult patients with a clinical diagnosis of bronchiectasis supported by CT Thorax were recruited in the study across multiple centres in the UK. Patient data collected at baseline included demographics, aetiology testing,




**Abstract S39 Figure 1**

comorbidities, lung function, radiology, treatments, microbiology and quality of life. Both respiratory and non-respiratory medications were included in the database but are not exhaustive.

**Results** 1626 patients were included in the analysis [1000 females (61.5%); 626 males (38.5%)] with a mean age of 64.4±13.4 years. 611 (37.6%) patients had documented asthma, 255 (15.7%) COPD and 58 (3.6%) ABPA.

The mean BSI score was 7.3±3.5.

376 patients (23.1%) had ≥5 respiratory-related medications prescribed. 643 patients (39.5%) had documented polypharmacy (including non-respiratory medications).

321 (36%) patients without co-existing asthma/COPD were prescribed ICS.

A weak positive relationship was established between age and number of medications prescribed ($r=0.126; p<0.001$). Higher rates of polypharmacy were seen in male cohort (45.5% as compared to 37.1% in female cohort) ($\chi^2 = 11.360; p<0.001$).

A logistic regression analysis showed that with each 1-point increase in BSI score the odds of polypharmacy increased by 20% (OR = 1.197, p <.001).

**Conclusion** Polypharmacy is a growing concern due to risks of cumulative harm. Since this database didn't collect all medications prescribed to patients, the current polypharmacy rates are underreported. Polypharmacy was associated with gender and disease severity. LABA and ICS were the most frequently prescribed medications. As new therapies and precision medicine emerge in bronchiectasis minimising polypharmacy should be considered.

## S40 BRONCHIECTASIS & MULTIMORBIDITY: A DATA ANALYSIS OF THE BRONCHUK REGISTRY TO DETERMINE THE ASSOCIATIONS BETWEEN MULTIMORBIDITY AND PATIENT OUTCOMES

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10.1136/thorax-2025-BTSabstracts.46

Bronchiectasis is a chronic lung condition in which the presence of certain comorbidities is associated with poor patient outcomes. The presence of multimorbidity, 2 or more chronic conditions, in the general population also has an established association with poorer health outcomes. This study aimed to establish the prevalence of comorbidities and multimorbidity in the BronchUK registry and determine the associations between multimorbidity in bronchiectasis and health related quality of life (HRQoL), exacerbation severity/frequency and mortality.

**Methods** 3 comorbidity counts (CC) were derived, representing a basic, intermediate and extended selection of the comorbidities in the BronchUK Registry. The association between multimorbidity as per these CCs and HRQoL (measured by the total St George's Respiratory Questionnaire (SGRQ) score) at baseline was investigated using linear regression analysis. The association of multimorbidity with more severe (measured by hospitalisation)/frequent (measured by an incidence of 3 or more annually) exacerbations and mortality over 5 years were investigated using logistic regression analysis. This approach was also to investigate the association between comorbidities and mortality.

**Results** Multimorbidity was a common finding in 1548 patients analysed, present in 54.1% to 96.2% of patients in the basic and extended CCs respectively. Having 2 comorbidities was associated with a clinically significant increase in SGRQ score and a poorer HRQoL (minimum score increase: 4.68-points (95% CI: 2.88–6.48). An increased odds of more frequent or severe exacerbations, OR=2.02 (95% CI: 1.05–3.87), was associated with having 5 comorbidities and an increased odds of mortality, OR=3.04 (95% CI: 1.42–6.49), As expected Pseudomonas, prior hospitalisations were linked with death. Individual comorbidities linked with death on univariate analysis included CCF, Chronic Renal Failure, AF, CTD, Osteoporosis, COPD, Tuberculosis, Depression, Diabetes and GORD.

**Conclusions** We found that multimorbidity is common in bronchiectasis and is significantly associated with a poorer HRQoL, more frequent/severe exacerbations and mortality. In line with other work, several individual comorbidities showed a stronger association to mortality than others. It is therefore important to consider multimorbidity, as measured by CCs, when considering the management of bronchiectasis patients.

## S41 MORBIDITY IN PATIENTS WITH NCFBE AND EXACERBATIONS IN THE THIN UK DATABASE

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**Background** Patients with non-cystic fibrosis bronchiectasis (NCFBE) often have pulmonary exacerbations (PEx), which are linked to high disease burden. This study explored the association of PEx with morbidity in the UK using primary care data.

**Methods** Patients with NCFBE aged ≥12 years were from THIN® UK, a longitudinal database of electronic health record extracts recorded by primary care physicians. PEx were defined as GP visits with codes for PEx, haemoptysis, lower respiratory tract infection (LRTI), or antibiotic prescription with either bronchiectasis or ≥1 LRTI symptom. Baseline (previous 12 months, ie, 2018) characteristics and association of PEx with morbidity at follow-up (2019–2022) were assessed.

**Abstract S41 Table 1** Baseline characteristics, comorbidities, and treatment patterns for patients with NCFBE

| Baseline characteristics | Patients with ≥2 PEx (n=1,881) | Patients with <2 PEx (n=10,225) | *P*-value |
|---|---|---|---|
| Age, mean (SD) years | 70.5 (12.4) | 69.4 (13.9) | <0.001 |
| Female, n (%) | 1,149 (61.1) | 5,818 (56.9) | 0.001 |
| Comorbidities, n (%) | | | |
| Chronic obstructive pulmonary disease (COPD) | 1,121 (59.6) | 3,739 (36.6) | <0.001 |
| Asthma | 1,118 (59.4) | 4,288 (41.9) | <0.001 |
| Symptoms, n (%) | | | |
| Chronic bronchitis | 1,058 (56.2) | 3,518 (34.4) | <0.001 |
| Lower respiratory tract infection | 1,193 (63.4) | 1,245 (12.2) | <0.001 |
| Treatment, n (%) | | | |
| Any antibiotics | 1,866 (99.2) | 6,742 (65.9) | <0.001 |
| Long-term antibiotics (≥28 days) | 448 (23.8) | 1,452 (14.2) | <0.001 |
| Inhaled corticosteroids | 1,260 (67.0) | 3,940 (38.5) | <0.001 |

PEx, pulmonary exacerbations; COPD, chronic obstructive pulmonary disease; NCFBE, non-cystic fibrosis bronchiectasis

**Results** Of 12,106 patients included, 15.5% had ≥2 PEx and 84.5% <2 PEx during baseline. Patients with ≥2 baseline PEx (vs with <2 PEx) were more likely to have asthma and COPD and to have received antibiotics and inhaled corticosteroids during baseline (table 1). Around 70% of patients with ≥2 PEx during baseline had another PEx in year 1 of follow-up vs around 30% with <2 PEx. After adjusting for covariates, having ≥2 PEx during baseline was the strongest predictor of PEx during follow-up (hazard ratio 2.6, 95% CI 2.4–2.8).

**Conclusion** In UK patients with NCFBE, PEx were associated with higher rates of comorbid asthma and COPD, treatment burden and risk of further PEx, demonstrating the importance of exacerbation reduction and management.

## S42 THE RESOURCE IMPACT OF BRONCHIECTASIS AND ASSOCIATED EXACERBATIONS: BASED ON A 5-YEAR PROSPECTIVE OBSERVATIONAL COHORT STUDY (BRONCHUK)

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**Background** Bronchiectasis is a chronic respiratory disease that is increasingly recognised globally. However, research on its economic impact is scarce. The primary objective of this study is to document major healthcare resource use and associated costs in a U.K. patient population with a focus on exacerbations

**Methods** We used baseline and follow up data from the Bronchiectasis Observational Cohort and Biobank UK (BronchUK). The study includes a baseline cohort of 1,119 patients diagnosed with primary bronchiectasis, who are followed for a period of up to five years. We extracted data from the database and compared to centrally held NHS resources use data.

**Results** The average age of the cohort was 64 years, and 62% were female. The most common bronchiectasis aetiology was idiopathic or post-infectious. Over the five-year period, the average annual distribution of exacerbations was as follows: 32% of patients had no exacerbations, 21% had one, 17.5% had two, and 29% had more than two. Longitudinal data show that while a significant proportion of patients (32%) experienced no exacerbations, those who did were

predominantly managed in primary care settings. Based on exacerbation frequency and type, and applying 2024/25 NHS reference costs, the estimated total healthcare costs for the study population ranged from £2 million to £3.2 million. Disaggregated estimates for the highest cost scenario include GP-only exacerbations (n=2,824; £479,945), outpatient-managed exacerbations (n=311; £260,126), and inpatient-managed exacerbations (n=680; £2,428,291). The average estimated cost per patient over the full period was £2,824, with the majority of costs attributable to inpatient-managed exacerbations.

**Conclusion** Bronchiectasis and associated exacerbations are a significant cost to the NHS. This study, based on the BronchUK cohort, is one of the first to provide a detailed analysis of healthcare utilisation and costs associated with bronchiectasis in the UK, particularly among patients at risk of exacerbation. The findings highlight the economic burden of the disease and the importance of targeted management strategies to reduce exacerbation rates and associated healthcare costs.

**Abstract S43 Table 1**

| | Asthma mortality | Asthma ICU admission | Asthma population total |
|---|---|---|---|
| **Characteristic** | Total n=113 | Total n= 1955 | Total n= 1 363 839 |
| **Age at event (years), mean (SD)** | 13.93 (4.33) | 14.06 (5.14) | Not applicable |
| **Age categories at event (years), n (%)** | | | |
| **5-11** | 43 (38%) | 756 (38.7%) | |
| **12-18** | 50 (44.3%) | 744 (38%) | |
| **19-21** | 20 (17.7%) | 455 (23.3%) | |
| **Sex, n (%)** | | | |
| **Male** | 57 (50.4%) | 924 (47.3%) | 689 410 (50.6%) |
| **Female** | 56 (49.6%) | 1031 (52.7%) | 674 341 (49.4%) |
| **Ethnicity, n (%)** | | | |
| **White** | 77 (68.1%) | 1215 (62.1%) | 907 937 (66.6%) |
| **Black** | 14 (12.4%) | 276 (14.1%) | 52 898 (3.9%) |
| **South Asian** | 9 (8%) | 222 (11.4%) | 79 271 (5.8%) |
| **Mixed** | ≤5 (≤1.0%)* | 139 (7.1%) | 36 157 (2.6%) |
| **Missing or unknown** | 12 (10.6%) | 35 (1.8%) | 257 753 (18.9%) |
| **Other** | ≤5 (≤1.0%)* | 68 (3.5%) | 29 823 (2.2%) |
| **IMD quintile, n (%)** | 112 (data for one child missing) | 1947 (missing data for 8 CYP) | *Total for IMD 1 360 792 (missing data for 3 047)* |
| **1 Least deprived** | 13 (11.6%) | 207 (10.6%) | 232 532 (17.1%) |
| **2** | 14 (12.5%) | 243 (12.5%) | 248 551 (18.3%) |
| **3** | 15 (13.4%) | 336 (17.3%) | 255 033 (18.7%) |
| **4** | 26 (23.2%) | 493 (25.3%) | 297 060 (21.8%) |
| **5 Most deprived** | 44 (39.3%) | 668 (34.3%) | 327 616 (24.1%) |
| **Region, n (%)** | | | |
| **North East** | 7 (6.2%) | 30 (1.5%) | 42 143 (3.1%) |
| **North West** | 23 (20.3%) | 222 (11.4%) | 255 948 (18.8%) |
| **Yorkshire** | ≤5(≤5%)* | 65 (3.3%) | 57 455 (4.2%) |
| **East Midlands** | ≤5 (≤5%)* | 31 (1.6%) | 47 975 (3.5%) |
| **West Midlands** | 19 (16.8%) | 414 (21.2%) | 212 776 (15.6%) |
| **East of England** | ≤5 (≤5%)* | 54 (2.8%) | 51 440 (3.8%) |
| **London** | 28 (24.8%) | 552 (28.2%) | 244 759 (18%) |
| **South East** | 16 (14.2%) | 300 (15.3%) | 273 780 (20%) |
| **South West** | 12 (10.6%) | 287 (14.7%) | 177 563 (13%) |

* Exact data in cells with counts ≤5 have been suppressed for confidentiality reasons

# Paediatric asthma: are we getting it right?

## S43 LONGITUDINAL TRENDS IN ASTHMA MORTALITY AND LIFE-THREATENING ASTHMA IN CHILDREN IN ENGLAND

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**Introduction** Despite available treatments, there remains an unacceptably high number of asthma deaths in children and young people(CYP) in the UK, many of which are preventable (NRAD 2014). A 2024 report from the National Child Mortality Database reported 54 asthma deaths between 2019–2023. Temporal trends in fatal and near-fatal asthma, and how these differ between different ethno-socioeconomic groups, are unknown.

**Aim** To describe longitudinal trends in fatal and life-threatening asthma (defined as an ICU-admission) over the past 20 years in CYP in England, stratified by socioeconomic and ethnic groups.

**Methods** We conducted a retrospective cohort study using routinely collected linked primary and secondary care, and administrative data from the Clinical Practice Research Datalink (CPRD), Hospital Episode Statistics (HES), Office for National Statistics (ONS) mortality data, and the Index of Multiple Deprivation (IMD). We included CYP aged 5–21 in any year since 2002, with an asthma ICD-10 code as the primary reason for an ICU-admission, or as the underlying cause of death. We estimated the incidence rate of asthma deaths and life-threatening attacks for each year, followed by a meta-regression to explore temporal trends, using aggregated annual incidence and year as a covariate.

**Results** Cohort characteristics were stratified by sex, ethnicity, region, and IMD (table 1). Approximately 12% of the cohort were non-white, and 24% from the most-deprived areas.

Between 2002–2022, there were 113 CYP asthma deaths; among them, 21% were non-white, and 39% were from the most-deprived areas. Mean incidence was 0.20 deaths per 100,000 per year. There was no significant change in annual asthma mortality rate in CYP over the past 20 years(Coefficient 0(SE=0.003,95%CI-0.006- 0.00,p=1.0).

A total of 1,955 CYP were admitted to ICU with life-threatening asthma between 2008–2022; 33% were non-white, and 34% were from the most-deprived areas. The mean ICU admission incidence rate was 4.3 per 100,000 per year. There was no significant change in life-threatening asthma ICU admission trends over time(Coefficient-$1.16x10^{-16}$ (SE=0.025, 95%CI -0.05–0.05, p=1.00).

**Conclusions** Our data show that trends in asthma mortality and ICU admissions have not improved over time. Moreover, children from non-white ethnicity, and the most-deprived areas appear to be disproportionately affected.

## S44 ARE CHILDREN ADMITTED WITH NEAR-FATAL ASTHMA RECEIVING THE CORE ELEMENTS OF CARE AT DISCHARGE? RESULTS FROM NRAP, THE NATIONAL RESPIRATORY AUDIT PROGRAMME

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**Background** NRAP collects data on acute asthma admissions from hospitals in England and Wales. This allows for monitoring quality of care, outcomes for children and young people (CYP) with asthma, and identifying areas for improvement. Multiple metrics are measured, including whether the Personalised Asthma Action Plan (PAAP) was reviewed or issued, the inhaler technique was checked, and the patient was referred for a follow-up with an asthma-trained clinician. These key performance indicators are aligned with the recommendations of the National Bundle of Care for CYP with Asthma.

**Aim** To establish whether children with life-threatening asthma attacks receive the nationally recommended core elements of care at discharge.

**Methods** We analysed the care delivered to children in 2023–2024, in NRAP audit participating hospitals, who: 1) were admitted to Intensive Care Unit (ICU) or High-Dependency Unit (HDU), or 2) received intravenous (IV) treatment for acute asthma. We audited the core elements of discharge: 1) receiving a PAAP, 2) having the inhaler technique checked, and 3) being referred for a 2-day or 4-week follow-up at discharge.

**Results** In 2023–24 a total of 14,311 CYP were admitted with asthma, 2,871(20% of all admissions) were given IV-medication, of which 1,218(42%) were then admitted to ICU, HDU, or both. 54% of the CYP with asthma who were on IV-treatment received PAAP, 69% had their inhaler technique checked, and 59% were referred for a 2-day or 4-week follow-up at discharge. In the cohort of 1,404(10% of all admissions) CYP admitted to ICU or HDU, 57% received PAAP on discharge, 71% had their inhaler technique checked, and 54% were reported to have been referred for either a 2-day or 4-week follow-up appointment.

**Conclusions** NRAP data show that in the group of the sickest children, admitted with near-fatal asthma, the standards of care delivered are suboptimal. Targeted quality improvement programs should be implemented at the Trust level to improve the care for CYP and reduce the risks of future asthma attacks.

## S45 MART VS CONVENTIONAL THERAPY: IMPACT ON BIOMARKERS, LUNG FUNCTION AND SYMPTOM CONTROL IN CHILDREN AND YOUNG PEOPLE WITH ASTHMA

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**Introduction** Maintenance and Reliever Therapy (MART) with a combination inhaled corticosteroid (ICS)-formoterol inhaler is an alternative to conventional therapy (maintenance ICS plus a short-acting-beta-agonist (SABA) reliever) for children and young people (CYP) with asthma. While MART is effective in reducing asthma attacks in adults and adolescents, little is known about the impact on airway inflammation or real-life asthma control in CYP.

**Abstract S45 Table 1** Participant Demographics and Clinical Characteristics. This table presents the baseline demographic and clinical characteristics of the entire study cohort, participants receiving MART, those on conventional asthma therapy and the p-value between the MART and conventional cohort (where applicable). Continuous variables are reported as the mean ± standard deviation (SD), when normally distributed or the median with the interquartile range, when non-parametric. Categorical variables are presented as number of participants (n) and percentage (%) of their respective cohorts. The sample size (n) for Blood Eosinophil Count and FeNO is specified where it differs from the total cohort or subgroup size

| | **All Subjects** *(n=119)* | **MART** *(n=32)* | **Conventional Therapy** *(n=87)* | **P Value** |
|---|---|---|---|---|
| **Age** *(years, median; IQR)* | 13.00; 11.00 - 15.00 | 14.84; 12.98 - 14.84 | 13.00; 10.44 - 15.00 | N/A |
| **Gender** | Male - 76 (63.9%) | Male - 21 (65.6%) | Male - 56 (64.4%) | N/A |
| | Female - 43 (36.1%) | Female - 11 (34.3%) | Female - 31 (35.6%) | |
| **Ethnicity** *(GLI categories)* | | | | |
| Caucasian | 56 (47.1%) | 17 (53.1%) | 39 (44.8%) | |
| Black | 12 (10.1%) | 4 (12.5%) | 8 (9.2%) | N/A |
| Northeast Asian | 0 | 0 | 0 | |
| Southeast Asian | 0 | 0 | 0 | |
| Other | 51 (42.9%) | 11 (34.4%) | 40 (46.0%) | |
| **Height** *(cm, mean ± SD)* | 153.31 ± 15.7 | 152.81 ± 15.8 | 150.42 ± 14.7 | N/A |
| **Weight** *(kg, median; IQR)* | 47.1; 36.2 - 56.8 | 52.1; 46.1 - 52.1 | 46.0; 34.5 - 55.8 | N/A |
| **Body Mass Index** *(median; IQR)* | 19.25; 17.23 - 22.14 | 19.82; 16.93 - 24.17 | 19.23; 17.32 - 22.06 | N/A |
| **Medication** | | | | |
| ICS | 13 (10.9%) | 0 | 13 (14.9%) | |
| ICS+LABA | 106 (89.1%) | 32 (100.0%) | 74 (85.1%) | N/A |
| Montelukast | 31 (26.1%) | 6 (18.8%) | 25 (28.7%) | |
| Antihistamine | 55 (46.2%) | 15 (46.9%) | 40 (46.0%) | |

| | | | | |
|---|---|---|---|---|
| **Asthma attacks in the 12 months prior to test visit** | 0 - 50 (42.0%) | 0 - 13 (40.6%) | 0 - 36 (41.4%) | |
| | 1 - 28 (23.5%) | 1 - 7 (21.9%) | 1 - 22 (25.3%) | P = 0.63 |
| | 2 - 18 (15.1%) | 2 - 5 (15.6%) | 2 - 13 (14.9%) | |
| | 3+ - 23 (19.3%) | 3+ - 7 (21.9%) | 3+ - 16 (18.4%) | |
| **Blood Eosinophil Count** *($x10^9$/L, median; IQR)* | *n=109* | *n=31* | *n=78* | |
| | 0.3; 0.2 - 0.6 | 0.2; 0.1 - 0.5 | 0.4; 0.2 - 0.7 | P < 0.05 |
| **FeNO** | *n=110* | *n=29* 40; 17 - 64 | *n=81* 25; 11 - 51 | P = 0.21 |
| *(ppb, median; IQR)* | 29; 11 - 57 | | | |
| **Spirometry** | | | | |
| FEV1%Predicted | 88.57 ± 15.57 | 91.3 ± 13.0 | 84.3 ± 16.4 | P < 0.05 |
| (*mean ± SD*) | 0.82; 0.76 - 0.87 | 0.82; 0.77 - 0.87 | 0.81; 0.75 - 0.87 | |
| FEV1/FVC (*median; IQR*) | | | | |
| **Symptom Score** | <20 – 65 (54.6%) | < 20 - 11 (34.4%) | <20 - 54 (62.1%) | |
| *(ACT/cACT, ≥ 20 = good control)* | ≥ 20 - 54 (45.4%) | ≥ 20 - 21 (65.6%) | ≥ 20 - 33 (37.9%) | P < 0.05 |

**Abbreviations:** MART - Maintenance and Reliever Therapy, n – number of participants, IQR – interquartile range, SD – standard deviation, GLI - Global Lung Function Initiative, SABA – Short-acting beta 2-agonists, ICS – inhaled corticosteroids, LABA – Long-Acting Beta-Agonists, FeNO – fractional exhaled nitric oxide, FEV1 – forced expiratory volume in 1 second, FEV1/FVC – forced expiratory volume in 1 second/forced vital capacity, ACT – Asthma Control Test, cACT – childhood Asthma Control Test, N/A – not applicable

**Hypothesis** In CYP with asthma, at GINA steps 3–5, MART is associated with improved inflammatory biomarkers (peripheral blood eosinophil count and fractional exhaled nitric oxide [FeNO]), lung function and symptom control, when compared to CYP on conventional therapy regimens.

**Methods** Retrospective study of CYP (6–16 years) with confirmed asthma, attending a tertiary respiratory clinic. Participants on MART were prescribed treatment for a minimum of 12 months prior to testing. Blood eosinophil count, FeNO, spirometry, and Asthma Control Test (ACT)/childhood ACT (cACT) were assessed. Group comparisons were made using unpaired t-tests for normally distributed data (mean ± standard deviation), Mann-Whitney tests for non-normally distributed data (median and interquartile range) and chi-squared tests for categorical data.

**Results** Data from 119 children were analysed (76 males; median age: 13.00 years, IQR – 11.00–15.00), 32 were prescribed MART and 87 were on conventional therapy (table 1). Those prescribed MART had significantly lower blood eosinophil count ($0.2\times10^9$/L, IQR: 0.1–0.5 vs. $0.4\times10^9$/L, IQR: 0.2–0.7, $p<0.05$). There was no significant difference in FeNO levels between the groups; MART 40ppb (IQR: 17–64) vs. 25ppb (IQR: 11–51, p=0.21). CYP using MART had significantly higher mean percent predicted FEV1; 91.3 ± 13.0% vs. 84.3 ± 16.4%, $p<0.05$). A higher proportion of CYP prescribed MART had good symptom control (65.6% (21/32), ACT/cACT score ≥ 20) compared to those prescribed conventional therapy (37.9% (33/87), $p<0.05$).

**Discussion** CYP prescribed MART had significantly lower blood eosinophil count, better lung function and better asthma control compared to those prescribed conventional therapy. However, there was no difference in FeNO for unclear reasons. This supports the benefit of MART on asthma pathophysiology in addition to the known impact on asthma attacks, in a real-life setting.

## S46 EVALUATING RECENT CHANGES TO PAEDIATRIC ASTHMA DIAGNOSTIC GUIDANCE FROM GINA AND NICE

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**Introduction** In 2024 both the Global Initiative for Asthma (GINA) and National Institute for Health and Care Excellence (NICE) published updated asthma diagnostic guidance for children. Both removed the need to identify airway obstruction ($FEV_1$:FVC < lower limit of normal), NICE also added testing for atopy via house dust mite (HDM) sensitisation or raised blood IgE and eosinophil levels into the diagnostic algorithm.

**Aim** To review the effect of guidance updates on the sensitivity and specificity of asthma diagnosis.

**Methods** Children with a clinical suspicion of asthma and not yet treated with inhaled corticosteroids (ICS) were recruited to the RADicA study. An expert panel classified the children as 'asthma' or 'not asthma' based on clinical history & examination, spirometry, bronchodilator reversibility, fractional exhaled nitric oxide, peak expiratory flow variability, bronchial challenge testing, allergy testing, blood eosinophil count and treatment response to 8-weeks of ICS. Children were then reclassified using GINA and NICE guidance before and after updates. Children with a missing result were classed as having a negative test. For NICE 2024 either a positive HDM sensitisation or raised IgE and eosinophil was taken as a positive result. The sensitivity and specificity of the varying guidance was calculated using expert panel diagnosis as the reference standard.

**Results** 134 children aged 5–16 years (mean age 9 years [SD 3 years], male n=66 [49%]) were classified as asthma

**Abstract S46 Table 1** Performance of paediatric asthma diagnostic guidance before and after published changes

| | Asthma N (%) | Sensitivity % (CI) | Specificity % (CI) | PPV % (CI) | NPV % (CI) |
|---|---|---|---|---|---|
| GINA 2023 | 22 (16%) | 22 (14–31) | 100 (89–100) | 100 (85–100) | 29 (27–32) |
| GINA 2024 | 66 (48%) | 61 (51–71) | 88 (72–97) | 94 (86–98) | 43 (36–50) |
| NICE 2017 | 25 (19%) | 25 (17–34) | 100 (89–100) | 100 (86–100) | 30 (28–33) |
| NICE 2024 | 96 (70%) | 83 (74–90) | 64 (45–80) | 88 (82–92) | 55 (43–85) |

**Abbreviations:** PPV, positive predictive value; NPV, negative predictive value; CI, confidence interval

(n=101) or not asthma (n=33) by expert panel. Table 1 shows how these children were classified by the various diagnostic guidance. Both GINA and NICE demonstrated an increase in children diagnosed as asthma using updated guidance. Analysis of NICE diagnostic pathways using HDM alone resulted in 88 children diagnosed as asthma (sensitivity 75% [CI 66–83], specificity 62% [CI 45–80]) compared to IgE and eosinophils diagnosing 75 children (sensitivity 69% [CI 59–78], specificity 85% [CI 68–96])

**Conclusion** Both updated guidelines result in a significantly higher number of positive asthma diagnosis being made in children. After removing the need for obstruction, GINA maintains a moderate specificity. However, the specificity of NICE pathways has been reduced by the use of HDM or raised blood IgE and eosinophils.

## S47 THE UTILITY OF BLOOD EOSINOPHILS, IGE AND SKIN PRICK TESTING IN PAEDIATRIC ASTHMA DIAGNOSIS

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**Background** The new UK (NICE/BTS/SIGN) asthma diagnostic guidelines for children include the use of House Dust Mite skin prick test (SPT) or Total IgE along with eosinophil count (eos), where exhaled nitric oxide (FeNO), spirometry/reversibility (BDR) or peak flow (PEFv) testing is either unavailable, negative or the child is unable to complete the test. A positive mite SPT (>3 mm wheal diameter) or elevated Total IgE (>120 KU/L) and eosinophil count (>$0.5x10^9$cells/L) with symptoms suggestive of asthma are sufficient to make the diagnosis in children aged 5–16 yrs. Within an observational asthma diagnostic cohort study we investigated the sensitivity and specificity of these tests in children with suspected asthma.

**Method** Children aged 5–16 yrs, referred from primary care with suspected asthma underwent clinical history, examination, FeNO, spirometry, BDR, PEFv, bronchial challenge, SPT to common inhaled allergens, Total IgE, blood eosinophils, and completed 8 weeks inhaled corticosteroid treatment. A panel of at least 3 asthma specialists using all available data confirmed or rejected the diagnosis of asthma.

**Results** 95 children underwent testing and had a definitive diagnostic outcome (51.6% male; mean (SD) age 10.0 (2.8) years; 73 Asthma, 22 Not Asthma). Positive house dust mite SPT had the lowest sensitivity and specificity of all the tests. Elevated Eos had the highest specificity which was not improved by the addition of Total IgE. However, the standard lung function tests had higher specificity than the newly proposed tests, but not all children could complete these tests. Sensitivity of all the tests was poor. See table 1.

**Abstract S47 Table 1**

| | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) |
|---|---|---|---|---|
| Eosinophil count (n=95) | 46.6 | 81.8 | 89.5 | 31.6 |
| Total IgE (n=95) | 75.3 | 68.2 | 88.7 | 45.5 |
| Mite SPT (n=94) | 61.1 | 59.0 | 83.0 | 31.7 |
| Total IgE and Eos (n=95) | 45.2 | 81.8 | 89.2 | 31.0 |
| FeNO (n=80) | 45.8 | 95.0 | 96.4 | 38.5 |
| BDR (n=87) | 32.3 | 100 | 100 | 33.3 |
| PEFv (n=71) | 30.3 | 100 | 100 | 27.8 |

**Conclusion** In children who are unable to complete standard lung function tests for asthma, an eosinophil count above $0.5x10^9$ cells/L may be an alternative test to consider. In our cohort, the addition of Total IgE did not improve diagnostic accuracy. If children are diagnosed only on the basis of mite SPT, the poor specificity needs to be noted (2 in 5 cases will be wrong) and their response to treatment should be carefully reviewed.

## S48 MEASUREMENT OF CHILDREN AND YOUNG PEOPLE'S ASTHMA MEDICATION ADHERENCE BY PATIENT/FAMILY REPORT, ELECTRONIC MONITORING DEVICES AND PRESCRIPTION REFILL RATE

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10.1136/thorax-2025-BTSabstracts.54

**Background** Poor adherence to maintenance asthma medication is associated with reduced quality of life, higher risk of death, and increased healthcare costs. Adherence assessment is challenging. Electronic monitoring devices(EMDs) provide actuation, although not inhalation, data, but are not widely available. The Medication Adherence Report Scale(MARS) is a validated adherence self-report measure. Prescription refill rate (PRR) provides data on prescriptions issued, but not whether dispensed/taken.

**Objective** To compare adherence assessment measures for children and young people(CYP) prescribed maintenance inhaled corticosteroids(ICS) or combination ICS/long-acting beta agonist(LABA) inhalers in our paediatric difficult asthma(DA) service.

**Methods** Retrospective analysis of data from CYP attending a tertiary paediatric asthma service. We compared EMD inhaler data, MARS score(5-point scale) or MARS-Asthma(MARS-A) (10-point scale) and PRR(ICS/ICS-LABA% uptake). We used descriptive statistics to characterise adherence assessed by each measure and Pearson's correlation to explore relationships between measures. Mann-Whitney was used to compare Fractional Exhaled Nitric Oxide(FENO), bronchodilator reversibility(BDR) and asthma control test(ACT) between those with 'good'(≥80%) and 'poor'(<80%) adherence.

**Results** 248 CYP, 117(47%) female, median age 11 y(range3y-16y3m). Median(range) EMD adherence was 77(1–100)% and PRR 66(6–150)%. Self-reported adherence using MARS and

MARS-A was high with mean scores of 4.8 and 4.7 respectively.

There was a weak correlation between EMD and PRR (R=0.26, p<0.006). There was no correlation between EMD and MARS(R=0.18,p=0.71) or MARS-A(R=0.033,p=0.81) or between any of the other measures.

When comparing EMD data from those with good and poor adherence there was no significant difference in FENO or BDR. 'Poor adherers' had better ACT scores than 'good adherers'(15.7vs12.2, *z*-score=2.69 p=0.007).

**Conclusions** Median adherence was suboptimal; only 32% had good adherence based on EMD data. However, self-reported adherence was high. There was a significant but weak relationship between EMD and PRR.

The higher ACT score in those with poor adherence suggests that when CYP feel their asthma control is good, they are less likely to use their ICS/ICS-LABA. However, many of these have high FENO and evidence of BDR and therefore risk an attack.

This study highlights the importance of objective measures of adherence. EMDs providing inhalation as well as actuation data are needed to provide a gold-standard adherence measure.

# Tracking the science: smarter asthma strategies

## S49 GINA STRATEGY REPORT (2024) UPDATES IMPROVE THE SENSITIVITY OF ASTHMA DIAGNOSIS BY REMOVING AIRFLOW LIMITATION CRITERION IN ADULTS

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10.1136/thorax-2025-BTSabstracts.55

**Introduction** In the GINA 2024 strategy report the requirement for documented airflow limitation was removed for establishing a diagnosis of asthma, and criteria amended to a history of typical variable respiratory symptoms with confirmed variable expiratory airflow.

**Aim** To determine the effect of removing airflow limitation as a criterion for asthma diagnosis on the sensitivity and specificity of the GINA guidelines.

**Methods** Adults presenting with respiratory symptoms but not receiving inhaled corticosteroids were referred from primary care for comprehensive diagnostic evaluation as part of the RADicA study. Asthma diagnosis (reference standard) was established by a panel of asthma specialists based on clinical history, physical examination, spirometry, bronchodilator reversibility (BDR), fractional exhaled nitric oxide (FeNO), peak expiratory flow variability, bronchial challenge testing, allergy testing, blood eosinophil counts, and treatment response to 4–12 weeks ICS. The sensitivity and specificity of the GINA 2023 criteria for diagnosing asthma was calculated and compared to the GINA 2024 criteria.

**Results** 118 adults [75 female, mean (SD) age 36 (12) y] with a definitive diagnostic outcome from the asthma specialists: 70 (59%) had asthma, were included in the study. 32 (27%) patients met the GINA 2023 criteria for asthma, compared to 74 (63%) with the updated GINA 2024 guidance. Consequently, the sensitivity improved from 46% (95% CI: 34–58%) to 90% (80–96%), whilst specificity decreased from 100% (93–100%) to 77% (63–88%) (table 1). The overall accuracy improved with the updated guidance from 68% (59–76%) to 85% (77–91%) (table 1).

**Abstract S49 Table 1** Performance of GINA 2023 and GINA 2024 strategy report criteria for the diagnosis of asthma compared to expert panel objective evidence review

| | Sensitivity | Specificity | PPV | NPV | Accuracy |
|---|---|---|---|---|---|
| GINA 2023 (%, 95% CI) | 46 (34–58) | 100 (93–100) | 100 (89–100) | 56 (50–61) | 68 (59–76) |
| GINA 2024 (%, 95% CI) | 90 (80–96) | 77 (63–88) | 85 (77–91) | 84 (92–92) | 85 (77–91) |

**Abbreviations:** GINA, Global Initiative for Asthma; PPV, positive predictive value; NPV, negative predictive value.

**Conclusion** The removal of documented airflow limitation in the GINA 2024 strategy report results in significantly fewer missed cases of asthma and improves the overall diagnostic accuracy.

## S50 ASTHMA UNTANGLED: STANDARDISING EXPERT CLINICIAN JUDGEMENT WITH A STRUCTURED MULTI-MODAL APPROACH

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10.1136/thorax-2025-BTSabstracts.56

**Background** Diagnosis of asthma remains challenging due to its heterogeneity, symptom variability, and the absence of a universally accepted gold standard. Clinical diagnosis often depends on weighing evidence for asthma against evidence of plausible alternative conditions, guided by the principle of inference to the best explanation.[1]

**Aim** To develop and evaluate a structured, multi-modal diagnostic template that systematically incorporates key clinical features supporting a diagnosis of asthma versus alternative diagnoses.

**Methods** Within the SMART study (NCT05357274), participants with a prior clinical diagnosis of asthma and ongoing symptoms despite inhaled corticosteroid (ICS) therapy were assessed using a diagnostic template (figure 1a and 1b).[2] This multi-dimensional approach synthesised elements from existing clinical guidelines and trial criteria, including markers of allergy, Type 2 inflammation, lung function, treatment response, and structured exclusion of alternative conditions. Diagnosis was not based solely on clinician judgment but on systematic template-based assessment.

**Results** Application of the multi-modal template confirmed asthma in 63 of 113 participants (56%), highlighting significant diagnostic uncertainty within the previously diagnosed population, outlined in figure 1c.

**Conclusion** A structured, template-based approach improves diagnostic rigour by replicating and standardising expert clinical judgment, integrating all relevant domains of evidence. This method enhances diagnostic consistency compared with

A **Asthma Diagnosis Template**

| Variable | Points |
|---|---|
| Physiology | |
| Obstructive spirometry | +1 if FEV1/FVC ratio < 0.75<br>+2 if FEV1/FVC ratio < 0.75 on more than one occasion |
| Bronchodilator responsiveness | +1 if > 10% improvement in predicted $FEV_1$ |
| $FEV_1$ variability over time | +1 if > 10% change in predicted FEV1 between visits |
| Small airways dysfunction (IOS) | +1 if < 2SD from mean value R5-20 |
| Bronchial hyper-responsiveness (Methacholine challenge test) | +2 if PD20 <0.5 umol or 100 ug<br>+1 if PD20 0.5-2.0 umol or 100-400ug |
| Inflammation | |
| FeNO | +1 if > 25ppb<br>+2 if > 50ppb |
| Blood Eosinophil Count (BEC) | +1 if > 0.3 x $10^9$<br>+2 if > 0.5 x $10^9$ |
| IgE | +1 if total or specific IgE positive |
| Clinical progress during digital period of monitoring | |
| Symptom alignment with objective asthma features | +1 if ACT improves (or worsens) as inflammation (BEC, FeNO) or physiology improves (or worsens)<br>***OR***<br>+1 if ACT does not improve but no other definite cause for symptoms |
| Objective change in relation to ICS | +1 if physiology **or** T2 only improves with ICS (or worsens off ICS)<br>+2 if physiology **and** T2 improves (or worsens off ICS)<br>***OR***<br>+1 if physiology or T2 do not improve but no other definite cause for symptoms |
| Exacerbations requiring oral corticosteroid treatment | +1 |
| Clinical progress at final follow up | |
| Clinical progress favours asthma | +1 |
| No better diagnosis emerges with additional investigations | +1 |
| Exacerbations requiring oral corticosteroid treatment | +1 |

B **Alternate Diagnosis Template**

| Variable | Points |
|---|---|
| Symptoms | |
| Improvement in symptoms in response to treating other condition | +1 |
| Inflammation and Physiology | |
| Blood Eosinophil Count | +1 if never > 0.3 |
| FeNO | +1 if never > 25 |
| Obstruction (Spirometry) | +1 if never obstructed |
| AHR (Challenge Testing) | +1 if negative MCT |
| Clinical Progress | |
| No exacerbations despite cessation or reduction in dosage of ICS | +1 |
| Follow Up | |
| Remains asymptomatic at follow up | +1 |
| Additional investigations reveal clear alternative cause for symptoms | +1 |
| Patient agreement of alternative cause | +1 |

C **Multi-Modal Diagnosis Template**

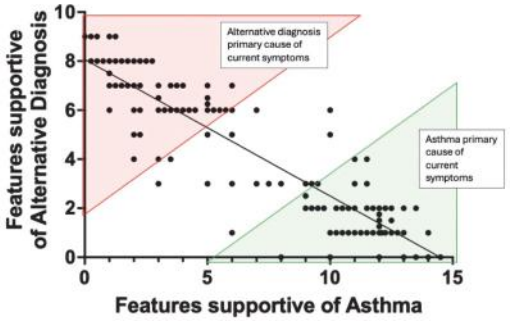



**Figure 1A.** (Far left) *Multi-modal diagnosis template highlighting features supportive of asthma as the cause for current symptoms*

***Figure 1B.*** *(Upper right) Multi-modal diagnosis template highlighting features supportive of an alternative cause for current symptoms*

***Figure 1C.*** *(Lower right) Scatter plot highlighting degree of certainty for asthma versus alternative condition as cause for current symptoms*

**Abstract S50 Figure 1**

guideline-based clinical assessment alone. However, it is time-consuming and resource-intensive, underlining the need for future development of more efficient, scalable diagnostic tools.

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## S51 MULTIDIMENSIONAL ASSESSMENT IN SEVERE ASTHMA: EARLY FINDINGS FROM THE BRISTOL EVALUATION OF NOVEL AIRWAYS DIAGNOSTICS, THERAPIES AND HEALTHCARE OUTCOMES (BREATHE) STUDY

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10.1136/thorax-2025-BTSabstracts.57

**Introduction** A treatable traits approach enables personalised management of asthma phenotypes, demonstrating improved patient outcomes.[1] The BREATHE study aims to characterise patients with suspected or established complex airways disease using multidimensional assessment to improve diagnosis and management based on treatable traits, including pulmonary, extra pulmonary and and risk factors domains.[1]

**Methods** BREATHE is a prospective, longitudinal cohort study recruiting adult patients referred to a UK tertiary asthma service with suspected or confirmed airways disease(REC: 23/LO/0279). Baseline assessment includes protocolised clinical history, examination, pulmonary function tests, imaging, multidisciplinary input and additional specialist investigations including continuous laryngoscopy examination. Here we provide a descriptive analysis of the first 100 unselected participants.

**Results** Recruitment started in November 2023. Of the first 100 patients, 69% were female; median age 54 yrs[42–65]. Most referrals came from primary (51%) and secondary care (40%). 6% were on an asthma biologic at referral. 74% of the 86 patients referred with severe asthma had this diagnosis confirmed following assessment. This group had high Th2 inflammation (raised eosinophils(61%) and FeNO(55%)) and ≥3 exacerbations/year(64%) despite 80% being on high dose inhaled corticosteroid (ICS) at referral. Treatable traits were common in confirmed asthma, 36% had symptoms of breathing pattern disorder (BPD) and 30% had symptoms of inducible laryngeal obstruction (ILO) and were referred for assessment. 26% (n=22) of severe asthma referrals did not have a confirmed asthma diagnosis following assessment. This group still had a high burden of OCS (55% ≥3) but lower biomarkers of Th2 inflammation when compared to the asthma confirmed group (eosinophils p≤0.001). Comorbid reflux and anxiety/depression featured highly in non-confirmed asthma, with 50% and 59% of patients referred for ILO or BPD assessment respectively.

**Conclusions** 26% of patients referred with severe asthma were found not to have an asthma diagnosis following multidimensional assessment. Treatable traits and comorbidities were common especially in patients with non-confirmed asthma who had significantly higher burden of mental health (54.5%) and suspected ILO (>59%). Together this highlights the value of

**Abstract S51 Table 1** Domain-based assessment of all asthma referrals comparing characteristics of those with confirmed asthma and those that need further investigation/diagnosis in doubt

| | | Asthma Confirmed n=64 | Asthma not confirmed n=22 | p-value |
|---|---|---|---|---|
| **CHARACTERISTICS** | | | | |
| **Gender** | Female n (%) | 44 (68.8) | 19 (86.4) | 0.183 |
| **Age** | Mean (SD) | 51.2 (17.1) | 51(13.6) | 0.96 |
| **IMD (quintile)** | Most deprived | 5(7.8) | 3(13.9) | 0.468 |
| | 2 | 17(26.6) | 2(9.1) | |
| | 3 | 11(17.2) | 4(18.2) | |
| | 4 | 15 (23.4) | 7(31.8) | |
| | Least deprived | 15(23.4) | 4(18.2) | |
| **Ethnicity** | White British n(%) | 56 (87.5) | 22 (100) | 0.188 |
| **Annualised OCS use** | 0 n (%) | 10 (15.6) | 5 (22.7) | |
| | 1 to 2 n (%) | 11 (17.1) | 5 (22.7) | |
| | ≥3 n (%) | 41 (64) | 12 (54.5) | |
| | Mean(SD) | 3.4(2.5) | 3.0(2.9) | 0.602 |
| **Evidence of PEFR variability or reversibility** | n (%) | 18(28.1) | 1 (4.5) | 0.045 |
| **CT features of airways disease** | ≥1 n (%) | 15 (23.4) | 2 (9.1) | 0.215 |
| **AIRWAYS ASSESSMENT** | | | | |
| **Eosinophils last 12m** | ≥0.15 n (%) | 55 (85.9) | 10 (45.4) | |
| | ≥0.3 n (%) | 39 (60.9) | 3 (13.6) | 0.001 |
| **Eosinophils ever** | ≥0.15 n (%) | 62 (96.8) | 17 (77.7) | |
| | ≥0.3 n (%) | 50 (78.1) | 8 (38.8) | 0.001 |
| **FeNO/ppb** | ≥25 n (%) | 35 (54.7) | 6 (27.3) | 0.083 |
| **COMORBIDITY ASSESSMENT** | | | | |
| **Cardiac** | n (%) | 23 (35.9) | 8 (36.3) | 1 |
| **Atopy** | n (%) | 43 (67.1) | 8 (36.3) | 0.018 |
| **ENT incl. rhinitis** | n (%) | 25 (39.1) | 4 (18.2) | 0.127 |
| **Chronic infection** | n (%) | 14 (21.8) | 0 (0) | 0.039 |
| **Reflux** | n (%) | 32 (50) | 12 (54.5) | 0.904 |
| **Referred for ILO assessment** | n (%) | 19 (29.6) | 13 (59) | 0.027 |
| **Referred for BPD assessment** | n (%) | 23 (35.9) | 11 (50) | 0.362 |
| **Depression/Anxiety** | n (%) | 20 (31.2) | 12 (54.5) | 0.09 |
| **RISK FACTORS** | | | | |
| **Poor adherence** | n (%) | 9 (14) | 2 (9) | 0.816 |
| **High BMI (≥30 kg/m$^2$)** | n (%) | 13 (20.3) | 7 (31.8) | 0.418 |
| **Smoking status** | Current smoker n(%) | 4 (6.2) | 0 (0) | 0.5 |
| | Ex-smoker n (%) | 23 (35.9) | 6 (27.2) | 0.568 |

BMI, Body mass index; BPD, Breathing pattern disorder; CT, computed tomography, ENT, ears nose and throat; FeNO, Fraction exhaled nitric oxide; ILO, Inducible laryngeal obstruction; IMD, index multiple deprivation; OCS, oral corticosteroids; PEFR, Peak expiratory flow rate; ppb, parts per billion.

multidimensional assessment. Longitudinal follow-up will be vital to assess the impact on longer-term outcomes.

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## S52 AGREEMENT BETWEEN FREQUENT HOME-BASED OR CLINIC-BASED MONITORING OF FENO IN ASTHMA: A SECONDARY ANALYSIS OF THE INFORM ASTHMA TRIAL

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10.1136/thorax-2025-BTSabstracts.58

**Introduction** The clinical utility of home fractional exhaled nitric oxide (FeNO) monitoring is unknown. The American Thoracic Society cut-point for an elevated FeNO in adults is >50 ppb and values above this threshold are associated with an increased risk of asthma exacerbations. A possible clinical application of home FeNO monitoring is it may facilitate many more FeNO measurements, without the constraints of attending a clinic appointment, and it is unknown how many more patients with a high FeNO may be identified with this approach compared to the usual practice of periodic clinic-based monitoring.

**Methods** The INFORM ASTHMA trial was a 26-week, open-label randomised controlled trial that compared budesonide-formoterol versus terbutaline for symptom relief in 181 adults with mild-moderate asthma using maintenance budesonide. The primary outcome for the study was a clinic-based FeNO measurement 26 weeks after randomisation. As part of the study, participants were given a home FeNO measurement device, the 'Vivatmo *me*' and recorded a daily measurement for the first two weeks of the study followed by weekly measurements for 10 weeks. Clinic-based FeNO were measured at weeks 0 (randomisation), 13 and 26 using a 'Vivatmo *pro*' device. Agreement between participants with any home measurement >50 ppb and any clinic measurement were assessed by a two-by-two contingency table and associated McNemar's test of agreement.

**Results** The mean (SD) age of participants was 33.9 (15.5) years, with age at asthma diagnosis of 11.2 (11.1) years, the mean (SD) baseline ACQ was 1.5 (0.68). The median (IQR) baseline FeNO was 62.5 (36 to 100.5) ppb. The agreement is shown in the table 1. There were 154/181 (69%) participants with a FeNO >50ppb for any of the clinic-based measurements and 119 (66%) with a FeNO >50ppb with the home-based measurements; McNemar's test for agreement was not statistically significant: Chi-square 1.1 (1df), P=0.30.

**Discussion** In this study of home-based versus clinic-based measurements of FeNO, there was no evidence of disagreement in the proportions of participants who had at least one FeNO >50ppb. This is consistent with frequent home-based measurement not offering greater detection of high FeNO compared to more routine clinic-based measurement.

**Abstract S52 Table 1** Agreement for FeNO >50ppb for home-based versus clinic-based measurement

| | Any clinic measurement greater than 50 ppb N/181 (%) | |
|---|---|---|
| **Any home measurement greater than 50 ppb** | **No** | **Yes** |
| **No** | 48 (27) | 14 (8) |
| **Yes** | 9 (5) | 110 (67) |

## S53 HIDING IN PLAIN SIGHT: USING BTS GUIDANCE TO IDENTIFY AND OPTIMISE MANAGEMENT OF 'ASTHMA-ECTASIS' AND ABPA THROUGH CLINICAL PHENOTYPING OF A BRONCHIECTASIS COHORT

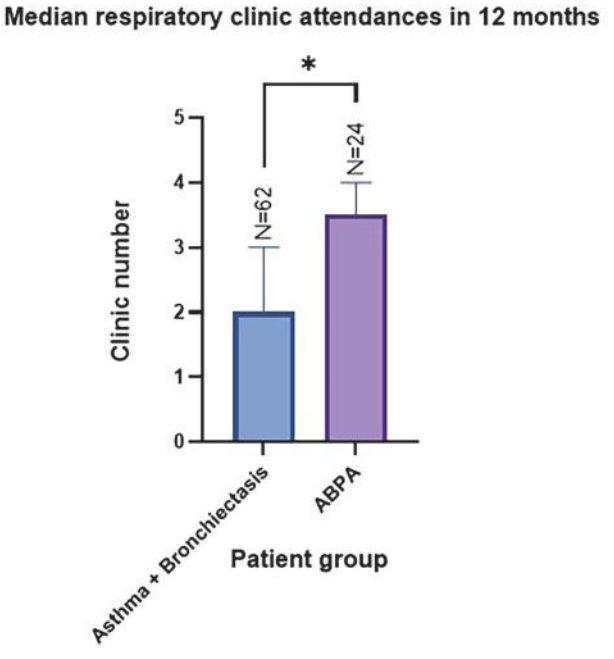
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10.1136/thorax-2025-BTSabstracts.59

**Introduction** Combining BTS/SIGN/NICE Asthma guidance with the recent BTS guidance on pulmonary aspergillosis (2025) a clear diagnostic pathway for clinical phenotyping of ABPA and Asthma within Bronchiectasis cohorts is now possible. We utlised this guidance to clinically phenotype those with asthma or ABPA within a specialist bronchiectasis service to optimise management and identify those in need of escalation or de-escalation of therapy.

**Methods** The database of a specialist bronchiectasis service was interrogated. Patients with radiological evidence of bronchiectasis and a co-diagnosis of asthma were identified. The 2024 joint BTS/NICE/SIGN asthma criteria was retrospectively applied. In those identified as having asthma, the new diagnostic criteria for ABPA were evaluated. From primary and secondary care electronic records, healthcare utilization burden, inhaled therapy and oral corticosteroids usage was compared between ABPA and non-ABPA cohorts.

**Results** Of the 113 patients with bronchiectasis and historical asthma diagnosis, 86 patients met current diagnostic criteria for asthma. Of these, 24 also met the diagnostic criteria for ABPA. In the 27 bronchiectasis patients with no current evidence of asthma, 25 were prescribed inhaled corticosteroids with an annual cost of £10,144 and 2,736 kg of CO2.




**Abstract S53 Figure 1** Comparison of number of respiratory clinic attendances over a 12-month period between patients with ABPA and patients with asthma + bronchiectasis. Bars represent median values ± 95% confidence interval for each group. Analysis was done using the Mann-Whitney U test due to non-normal distribution of the data. Asterisks indicate the statistically significant $p = 0.0114$ (<0.05)

Respiratory clinic attendance was more frequent in the preceding 12 months for patients with ABPA (median 3.5) than with 'asthma-ectasis' (2, p = 0.0114) (figure 1). Of the Asthma-ABPA cohort,16 patients have been highlighted for consideration of escalation of asthma therapy, including biologics, based on corticosteroid use or exacerbation frequency.

**Conclusion** Historical asthma diagnoses are common in bronchiectasis patients, but updated guidance suggests many may be suitable for de-labelling and de-escalation of therapy.[1] Inappropriate corticosteroid treatment carries significant morbidity risk, with financial and environmental cost. Concurrently others with 'asthma-ectasis' may require escalation of asthma therapy including biologics but this need may be masked by concurrent bronchiectasis. New guidance allows effective clinical phenotyping to formally identify specific cohorts with overlap between Asthma, Bronchiectasis and ABPA to improve access to expertise and treatment options to reduce healthcare burden and morbidity.

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## S54 PREDICTORS OF RESPONSE TO BIOLOGIC THERAPY IN A UK SEVERE ASTHMA POPULATION

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10.1136/thorax-2025-BTSabstracts.60

**Background** Biologic therapies have revolutionized severe asthma management, reducing systemic corticosteroid use and exacerbations. Individual treatment response is variable and difficult to predict.

**Objective** To characterise the clinical response to biologic therapy in patients with severe asthma and identify predictors of response and non-response.

**Methods** We conducted a retrospective analysis of severe asthma patients treated with biologics for 12 months (March 2014– 2025). Clinical response was defined as ≥50% reduction in annualised asthma exacerbation rate (AER) and/or oral corticosteroid (OCS) dose. A stratified, weighted penalised logistic regression (PLR) model was trained (AUC-optimised) and validated using stepwise logistic regression (SLR).

**Results** 321/374 (85.8%) achieved a clinical response at 12 months. At baseline, responders had better asthma control and health-related quality of life compared to non-responders; ACQ-6 score (2.50 [1.50 - 3.34] vs 3.33 [2.32 - 4.15]; p = 0.001) respectively; EQ-5D-5L score (10.80 [7.00 - 17.00] vs 38.77 [10.14 - 103.50]; p < 0.001) and AQLQ score 3.76 [2.63 – 4.88] vs 2.34 [1.13 – 3.91]; p < 0.001). Responders also had shorter disease duration (20.0 [5.0 - 33.9] years vs 27.0 [14.0 - 39.5] years; p = 0.008), lower blood neutrophil counts (7.01 [4.93 - 9.83] vs 8.40 [6.45 - 12.17]; p = 0.002), higher serum IgA levels (2.29 [1.67 - 2.78] vs 2.01

[1.31 - 2.35]; p = 0.006) and lower levels of Aspergillus-specific IgE (0.07 [0.02 - 0.78] vs 0.22 [0.03 - 4.94]; p = 0.004). PLR identified several predictors of response with strongest associations included higher serum IgA (OR = 1.08), male sex (OR = 1.16), and lower ACQ6 scores (OR = 0.96). In the SLR model lower ACQ6 scores (OR = 0.64, p = 0.0007) and diminished Aspergillus sensitisation (OR = 0.96, p = 0.0044) were significantly associated with treatment response and reduced blood neutrophil counts (OR = 0.93, p = 0.061), showing a trend towards significance.

**Conclusion** Lower baseline ACQ6 scores, diminished Aspergillus sensitisation, higher serum IgA, and lower neutrophil counts were independently associated with good clinical response to biologics. Prospective studies are needed to validate these biomarkers' performance in guiding personalised treatment.

## S55 BACTERIAL IMMUNOTHERAPY FOR THE PREVENTION OF EXACERBATIONS IN PATIENTS WITH CHRONIC RESPIRATORY DISEASES

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10.1136/thorax-2025-BTSabstracts.61

**Introduction** Bacterial autovaccines are inactivated bacterial suspensions made from one or more bacterial strains obtained from the culture of a sample taken from the patient. They are then administered to develop an acquired immune response. Bacterial immunotherapy (BI) is indicated in recurrent bacterial infections. Good results have been described in otorhinolaryngological and urinary infections. In bronchopulmonary infections, experience is more limited. We studied retrosprectively the effect of BI in chronic respiratory patients.

**Methods** We studied 53 patients treated with BI over a 12-month period. The samples were taken in the Pneumology office, by swab and at oropharyngeal level. The number of infections before and after BI was reviewed, as well as the number of hospital admissions for lower respiratory tract infections.

**Abstract S55 Table 1**

| | BEFORE BI | AFTER BI | DIFFERENCES |
|---|---|---|---|
| **EXACERBATIONS** | | | |
| **0** | 7 (13.2%) | 36 (67.9%) | |
| **1** | 13 (24.5%) | 12 (22.6%) | |
| **2** | 20 (37.7%) | 5 (9.4%) | **p <0.06** |
| **3** | 12 (22.6%) | 0 | |
| **4** | 1 (1.9%) | 0 | |
| **EXACERBATIONS** | | | |
| **≤1** | 20 (37.7%) | 48 (90.6%) | |
| | | | **↓52.9%** |
| **≥2** | 33 (62.3%) | 5 (9.4%) | |
| **HOSPITAL ADMISSIONS** | 17 (32.1%) | 4 (7.5%) | **↓24.6%** |

**Results** 53 patients (32 men) with a mean age of 72.8±10.3 and BMI of 29.8±6.36. Smokers 16 (30.2%), former smokers 25 (47.2%). 38 patients had COPD: mild grade 6 (15.4%), moderate (38.5%), severe (46.2%). 61 patients (30.8%) were asthmatic and 13 (24.5%) had bronchiectasis.Bacteria detected: S. pneumonia 48 (90.6%), S. aureus 15 (28.3%), Klebsiella 12 (22.6%), P aeruginosa 10 (18.9%), Enterobacter 6 (11.3%), Acinetobacter iwoffi 3 (5.7%) and Serratia 1 (1.9%). 42 patients (79.2%) combined two of these germs in their vaccines. Hospital admissions for infectious causes before (17 patients; 32.1%) and after BI (4 patients; 7.5%). Exacerbations (≤1 vs ≥2): ≤1 before BI 20 patients (37.7%)//after 48 (90.6%). ≥2 before BI 33 patients (62.3%), after 5 (9.4%). Before BI with zero exacerbations 7 patients (13.2%), after BI 36 (67.9%), indicating a 54.7% increase of patients without exacerbations.The results are shown in table 1.

**Conclusions** Due to the low number of patients in our study, statistical significance has not been reached (p<0.06) although there is an evident tendency, since the number of exacerbations and admissions have clearly decreased. It is noteworthy that the percentage of patients without new infections after autovaccines increased by 54%. It seems to us that bacterial lysates, through stimulation of the immune system can be an effective therapeutic tool for prevention respiratory infections.

# Impact of infection on disease mechanisms

## S56 MACHINE LEARNING AND ENRICHMENT ANALYSIS REVEAL PERSISTENT PROTEIN SIGNATURES IN LONGITUDINAL POST-COVID SYMPTOM TRAJECTORIES

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10.1136/thorax-2025-BTSabstracts.62

Post-COVID syndrome (PCS) is characterised by persistent symptoms affecting multiple organ systems. Despite evidence of immune dysregulation, the protein signatures driving PCS remain unclear. This study applies stratified machine learning and enrichment analysis to identify protein markers distinguishing PCS groups from recovered individuals longitudinally. We analysed samples from 365 individuals who had been hospitalised for COVID-19, classifying them into four symptom-based groups (Fatigue, Affective, Cardiopulmonary, and Gastrointestinal) and a recovered group. Up to 384 inflammatory plasma proteins were measured at two research visits (5 and 12 months post-hospital discharge) using Olink Explore. Stratified, cross-validated penalised logistic regression models were trained and evaluated using AUC, accuracy, and F1-score. Enrichment analysis was performed to identify functionally related pathways. Overall, several biomarkers (CCL7, CTRC, FCAR, IL10RA, LAMP3, MVK, TPSAB1, LGALS9) were consistently upregulated across most groups, suggesting a shared systemic immune dysregulation mechanism. Per-group analysis

revealed additional insights: Fatigue (HSD11B1, CKMT1A, MATN2), Gastrointestinal (CLEC4C, CKMT1A, CNTNAP2), and Affective (MATN2, ISM1). Additionally, enrichment analysis revealed that shared pathways across PCS symptom subgroups predominantly involve immune system regulation, including leukocyte activation, cytokine-mediated signalling, and response to cytokines. These findings suggest a common underlying axis of persistent immune dysregulation contributing to symptom manifestation across domains. Our findings suggest that all PCS groups share immune and vascular dysregulation as a core mechanism, with some symptom-specific proteins identified for each group.

## S57 EP395, A NOVEL NON-ANTIBIOTIC MACROLIDE, ENHANCES ANTIVIRAL RESPONSES IN BRONCHIAL EPITHELIAL CELLS FROM ASTHMA PATIENTS

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10.1136/thorax-2025-BTSabstracts.63

Macrolide antibiotics such as azithromycin have been shown to reduce asthma exacerbations and improve anti-viral properties of bronchial epithelial cells (BECs). However, its long-term use has been associated with increased antimicrobial resistance. EP395 is a novel macrolide with negligible antimicrobial activity being developed as a potential treatment to reduce exacerbations of chronic respiratory diseases (Hohlfeld et al., 2025. Pulm. Pharmacol. Ther. 90). We have now studied its effect on rhinoviral-induced anti-viral and pro-inflammatory responses in BECs.

Primary BECs from asthma patients (n=6–9) were pre-treated with EP395 (10 μM) or azithromycin (10 μM) for 24 hours before infection with rhinovirus (RV) 1B for 24 or 48 hours. Gene expression of IFN-β, MDA5, CCL20 and TNF-α as well as viral RNA was measured using RT-qPCR. Lactate dehydrogenase (LDH) activity was measured in cell supernatants as a marker of cell toxicity.

EP395 treatment of RV1B infected BECs did not alter the LDH activity in cell supernatants, indicating a lack of cytotoxicity. Furthermore, EP395 significantly increased RV-induced mRNA expression of IFN-β (6.6-fold compared to RV alone, p=0.01) and, concomitantly, significantly reduced viral load (mRNA) (0.7-fold compared to RV alone, p=0.003), in BECs at 24 hours post infection. There was also a trend towards an increased RV-induced expression of MDA5 (3-fold compared to RV alone, p=0.1). EP395 treatment did not alter RV-induced mRNA expression of TNF-α nor CCL20. Compared to azithromycin, the effects of EP395 on the BEC anti-viral and pro-inflammatory response were similar.

EP395 improves the IFN-β response and anti-viral activity in BECs from asthma patients, without affecting the pro-inflammatory response. This suggests EP395 could, by modulating epithelial antiviral immunity, be a beneficial treatment for reducing viral-induced exacerbations in asthma without the risk of contributing to antimicrobial resistance.

## S58 VIRAL INFECTION PERTURBS THE RESPIRATORY MYCOBIOME IN COPD

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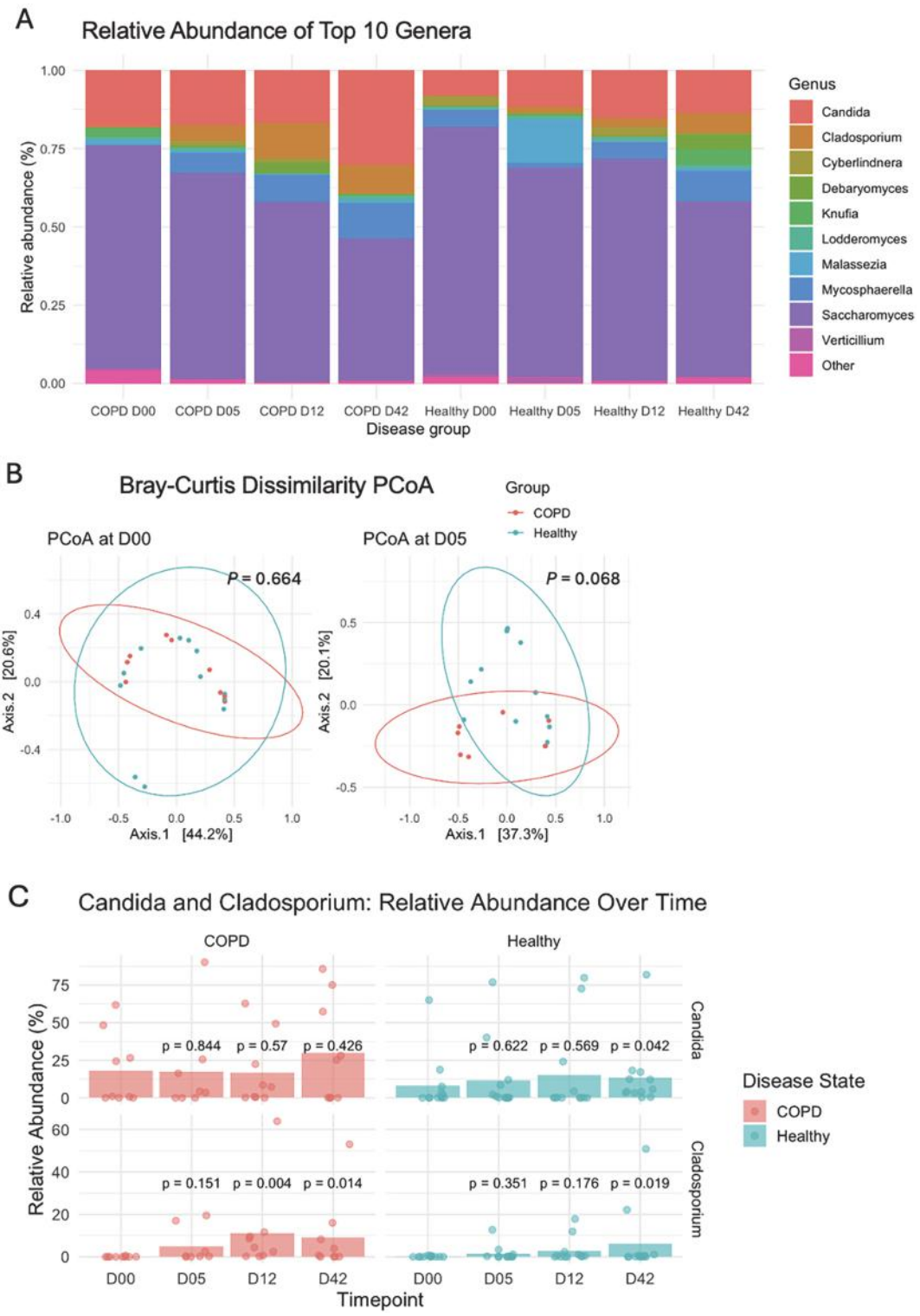
**Introduction** Molecular studies have recently revealed that the lower respiratory tract harbours a microbiome consisting of bacteria, fungi, and viruses. Emerging evidence indicates that commensal bacteria ('bacteriome') can modulate host immunity but very little is known about commensal fungi ('mycobiome'). Mycobiome perturbations have been reported in stable COPD but the role they play in driving susceptibility to infectious exacerbations are unknown. An emerging paradigm of exacerbation pathogenesis argues that consideration of the bacteriome alone may be an over-simplification in understanding drivers of these heterogenous episodes. We therefore sought to understand how the mycobiome changes and relates to inflammatory responses during virus-exacerbated COPD.

**Methods** We analysed samples collected during carefully controlled studies of rhinovirus challenge in COPD (n=9) and healthy controls (n=12). Sputum was analysed at baseline (pre-infection) and at days 5, 12 and 42 following rhinovirus inoculation. ITS2 sequencing was used to profile the mycobiome and correlations with cytokine measurements by MSD were assessed.

**Results** Changes in fungal genera occurring during the infection course in COPD and healthy subjects are shown in figure 1A. Rhinovirus infection led to a trend towards increased alpha diversity in COPD subjects ($P=0.129$) at day 12 post-infection, an effect not observed in healthy controls. Bray-Curtis dissimilarity showed separation between the COPD and healthy groups at day 5 post-infection ($P=0.068$) (figure 1B). Mycobiome changes during infection broadly showed inter-subject heterogeneity (figure 1C). Significant expansion of *Cladosporium* was observed in COPD subjects from baseline to day 12 post-infection ($P=0.004$) (figure 1C).

In COPD patients, early expansion of *Candida* (day 5 post-infection) correlated with sputum concentrations of pro-inflammatory (MIP-1beta, $P<0.01$) and type 2 cytokines (IL-13, $P<0.05$). The most prominent correlations in the healthy participants occurred at day 12, where *Candida* and *Cladosporium* abundance correlated with a range of pro-inflammatory and type 2 cytokines.

**Conclusion** The respiratory mycobiome is perturbed during respiratory viral infection with heterogenous changes seen in COPD and healthy individuals. Correlations between fungal genera and inflammation raise the possibility that these commensals directly influence immune responses during exacerbations but functional studies would be required for further confirmation.




**Abstract S58 Figure 1** **(A)** Relative abundance of the top 10 genera at baseline, days 5, 12 and 42 following RV infection in COPD and healthy subjects. **(B)** PCoA by Bray-Curtis dissimilarity at day 0 and day 5. **(C)** Relative abundance of Candida and Cladosporium at each timepoint in COPD and healthy subjects

## S59 EFFECTS OF BACTERIA AND HEME ON NEUTROPHIL-ENDOTHELIAL INTERACTIONS IN COPD

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Neutrophil recruitment to the lungs is elevated in COPD patients. Lung endothelial cells are fundamental in neutrophil recruitment, attachment and diapedesis. Red blood cell-derived iron (heme) is increased in COPD alveoli; heme can cause endothelial dysfunction, specifically increased inflammatory responses and junctional permeability. Neutrophilic inflammation in COPD is associated with bacterial colonisation including H. *influenzae* and M. *catarrhalis*. We evaluated the influence of H. *influenzae* and M. *catarrhalis* on endothelial cell- neutrophil adhesion, and assessed if heme altered the effects of bacterial exposure on endothelial cells.

Lung endothelial cells (HULEC-5a) were exposed to either H. *influenzae* or M. *catarrhalis*, at MOI 5:1 for 24h following pe-treated with/without heme (50 μM or 100 μM) for 24h. Neutrophil-endothelial adhesion was assessed using fluorescently labelled neutrophils, quantitative measurement of fluorescence by microplate reader and confirmed observationally by microscopy (n=12). Endothelial cell expression of neutrophil adhesion genes (ICAM-1, VCAM-1 and E-Selectin) was measured using RT-qPCR (n=8).

Endothelial cells exposed to H. *influenzae* and M. *catarrhalis* significantly upregulated expression of adhesion genes. M. *catarrhalis* induced upregulation of ICAM-1, E-Selectin and VCAM-1 (91.1, 130.3 and 3.6 fold respectively, $p<0.05$ for all). H. *influenzae* increased ICAM-1 and E-Selectin (91.1 and 139.8 fold respectively, $p<0.01$ for all). M. *catarrhalis*, but not H. *influenzae*, significantly elevated neutrophil adhesion ($p<0.0001$). Pre-treatment with heme had no significant effect on bacteria induced adhesion gene expression or neutrophil adhesion.

Endothelial cell expression of genes involved in neutrophil recruitment and adhesion are modulated by exposure to pathogenic bacteria H. *influenzae* and M. *catarrhalis*, with the latter also functionally increasing neutrophil adhesion. Heme did not directly affect neutrophil adhesion to the endothelium. Bacterial modulation of neutrophil migration through the endothelium may be a feature of endothelial dysfunction, amplifying neutrophilic inflammation and increasing exacerbation risk in COPD patients.

## S60 RESOLVIN D1 AMELIORATES THE INFLAMMATORY RESPONSE TO HAEMOPHILUS INFLUENZAE INFECTION IN COPD MONOCYTE-DERIVED MACROPHAGES

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10.1136/thorax-2025-BTSabstracts.66

**Background** Chronic Obstructive Pulmonary Disease (COPD) is associated with persistent airway inflammation, which may be driven by an imbalance between pro-inflammatory macrophages and dysfunctional anti-inflammatory macrophages. Resolvin D1 (RvD1), is a specialised pro-resolving mediator derived from the breakdown of docosahexaenoic acid by arachidonate 15-lipoxygenase (ALOX15). RvD1's role on macrophage function is poorly understood.

**Objective 1** To investigate the effect of RvD1 on the phagocytic activity and inflammatory cytokine response of COPD monocyte-derived macrophages (MDMs) to *Haemophilus Influenzae* (*HI*).

**Objective 2** To establish the relationship between RvD1 signalling and anti-inflammatory IL-10 production via STAT3.

**Methods** Peripheral blood monocytes were derived from COPD patients (n=10) and then cultured in GM-CSF/M-CSF respectively for 12 days to generate pro/anti-inflammatory G-MDMs/M-MDMs. MDMs were treated with vehicle (EtOH) or exogenous 1nM/10nM RvD1 30 minutes prior to and 1-hour post-exposure to fluorescently labelled *HI*. Phagocytosis was measured using FLUOstar OPTIMA at 4h and 24h, and values corrected using MTT assay for cell viability. Supernatants and cell-lysates were collected at 0, 4h and 24h for ELISA, qPCR and western blot.

**Results** There was no improvement in the phagocytic activity of RvD1 treated MDMs compared to vehicle in both G-MDMs and M-MDMs. In M-MDMs, 10nM RvD1 treatment compared to vehicle resulted in a significant reduction in both IL-6 (452.5 vs 171.1 pg/mL, p= 0.0197) and CXCL8 (923.8 vs 853.9 pg/mL, p= 0.0186) at 24 hours. RvD1 also increased production of IL-10 (150.8 vs 418.9, p=0.0523). Interestingly, this corresponded to a 2-fold increase in STAT3. Further, *HI* infected cells showed 1.5-fold increase in RvD1 receptor (FPR2) expression at 24 hours, compared to vehicle. There was also increased expression of ALOX15 in the vehicle group at 4h compared to the RvD1 treated group (1.1 vs 0.8 fold change, p=0.044).

**Conclusion** RvD1 ameliorated the inflammatory cytokine response to *HI* in COPD M-MDMs with greatest effects seen at 24h. ALOX15 is involved in synthesis of RvD1 during the early stages of bacterial infection in COPD MDMs, which may explain why patients with viral AECOPD experience worse symptoms and delayed recovery. Work is ongoing to determine the effects of RvD1 on virus-infected MDMs.

## S61 PROTECTIVE EARLY INNATE IMMUNE ACTIVATION BY MITOCHONDRIAL ANTIVIRAL SIGNALLING PROTEIN (MAVS) IN BACTERIAL PNEUMONIA

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10.1136/thorax-2025-BTSabstracts.67

**Introduction** Mitochondrial antiviral signalling protein (MAVS), a 540-amino acid protein primarily located on the mitochondria outer membrane plays a pivotal role in antiviral immunity as an intracellular pathogen recognition receptor for viruses, inducing type I/III IFN and activating NFκB and IRF3. Recent findings implicate MAVS signalling also in antifungal immune responses. However, the role of MAVS in antibacterial host defence remains unknown. The Gram-negative bacterium non-typeable *Haemophilus influenzae* (NTHi) is the predominant pathogen in airways diseases such as asthma and chronic obstructive pulmonary disease. We aimed to determine the role of MAVS in immune defence against NTHi *in vivo*.

**Methods** We infected C57BL/6 (wt) and MAVS KO mice with NTHi and analysed bacterial loads, weight loss, pathological change of lung tissues, infection progression, inflammatory

and immune responses by transcriptomics and spectral flow cytometry.
**Results** NTHi infection leads to the degradation of MAVS. MAVS deficiency results in compromised bacterial control during NTHi infection. *Mavs*$^{-/-}$ mice demonstrated impaired early production of many proinflammatory cytokines including a weaker induction of IFN-β and reduced accumulation and activation of NK cells. Depletion of macrophages eliminated the disparity in bacterial loads and weight loss between the two mouse strains. Moreover, *in vitro* experiments using freshly isolated alveolar macrophages revealed that MAVS played an important role in macrophage function against bacterial infections by detecting bacterial RNA.
**Conclusions** We have discovered an important new role for MAVS in early innate immunity against bacterial lung infection, both *in vitro* and *in vivo*.

# Biology and clinical insights in cancer

## S62 HIGH DIMENSIONAL IMMUNE ANALYSIS REVEALS IMMUNE SUBSETS ASSOCIATED WITH LUNG CANCER

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10.1136/thorax-2025-BTSabstracts.68

Lung cancer causes more deaths than any other cancer because it is usually diagnosed after spreading beyond the margins of curative treatment. Clinical trials have demonstrated a reduction in deaths from lung cancer by screening with low-dose CT scans, but applying these at scale is expensive, and the national program is therefore only available to people at the highest risk. This means that 60% people diagnosed with lung cancer would not have been eligible for a screening scan, missing out on the chance for an early diagnosis. New tests (biomarkers) are needed for early detection screening across the population. Immune cells are intimately involved in cancer evolution from the earliest stages. Given that multiple immune cells can interact with each cancer cell, signals of cancer exposure should be detectable when cancer burden is low in early-stage disease. In this study, we tested the hypothesis that lung cancer is associated with detectable differences in circulating peripheral immune cells. We recruited 17 patients with lung cancer and 32 current/former smokers without lung cancer and performed mass cytometry (30 marker panel) on whole blood, processed fresh on the day of sample donation. Lung cancer was associated with a significant increase in intermediate monocytes ($p<0.0001$) and a decrease in sub-populations of CD8 T cells ($p<0.05$). Intermediate monocyte levels could separate lung cancer from controls with a sensitivity of 94% and a specificity of 71%, with area under the curve (AUC) of 0.8989. Current work involves confirming these findings in a larger cohort of patients and identifying minimal panels of antibodies that distinguish these subset changes for prototype clinical tests.

## S63 METABOLIC PROFILING OF PERIPHERAL BLOOD AND TISSUE SPECIFIC IMMUNE ALTERATIONS IN DIFFUSE IDIOPATHIC PULMONARY NEUROENDOCRINE CELL HYPERPLASIA (DIPNECH)

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10.1136/thorax-2025-BTSabstracts.69

**Introduction and objectives** Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH) is a rare lung disorder with limited treatment options. Abnormal proliferation of pulmonary neuroendocrine cells (PNEC) results in the formation of tumourlets and lymphocytic infiltrates. The pathogenesis and role of immune cells in DIPNECH remains poorly understood. We hypothesise that immune cells with dysregulated metabolism are recruited to the DIPNECH lung where they drive PNEC dysfunction.
**Methods** Peripheral blood was collected from 11 DIPNECH and 7 control patients (sex/age matched). Circulatng granulocytes, lymphocytes and myeloid cells were identified using flow cytometry. Single Cell ENergetIc metabolism by profiling Translation inHibition (SCENITH) was used to investigate metabolic in T cell subsets. To explore the lung/tumour microenvironment immunohistochemistry and histological analysis was performed using 12 DIPNECH and 13 control biopsy samples.
**Results** In the peripheral blood, while populations of granulocytes and myeloid cells remained unchanged, reduced frequencies of T/B lymphocytes were detected in DIPNECH samples compared to controls. Interestingly, CD71 surface expression was significantly elevated on CD4+ T lymphocytes from DIPNECH patients. CD71 is a key mediator of cellular iron uptake, suggesting that iron transport in the periphery may be dysregulated in DIPNECH. The amount of CD69, a marker of early T cell activation, was higher on CD8+ T cells from DIPNECH patients compared to controls. Mitochondrial dependence, glycolytic capacity and fatty acid/amino acid oxidation were measured in both control and DIPNECH patient T cells. We detected tumourlets (Chromogranin A+) near small bronchioles and lymphocytic recruitment in DIPNECH patient lung biopsies. This was accompanied by alterations in proteins that regulate cell size, proliferation, metabolism and mTOR signalling (RPS6 and 4EBP1). Altered metabolism in recruited immune cells may promote lung PNEC cell survival/proliferation *via* immune-epithelial crosstalk. Taken together, our data suggest both systemic and tissue-specific immune alterations in DIPNECH accompanied by alteration in cellular metabolism.
**Conclusions** Altered metabolism and metabolic plasticity in immune cells may promote adjacent lung PNEC proliferation and dysfunction. Disrupting this interaction may be a novel treatment for DIPNECH.

## S64 INTEGRATIVE IMMUNE AND GENOMIC PROFILING OF THYMIC EPITHELIAL TUMOURS: A TRANSLATIONAL STUDY OF BIOMARKERS AND THERAPEUTIC POTENTIAL

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10.1136/thorax-2025-BTSabstracts.70

**Background** Thymic epithelial tumours (TETs) are rare, histologically diverse malignancies with limited treatment options and poorly characterised molecular drivers. Given the scarcity of biomarkers to guide immunotherapy in TETs, we conducted an integrative analysis to characterise immune phenotypes and identify actionable genomic and copy number alterations across different TET subtypes.

**Methods** Three datasets were analysed:

1. Immune-checkpoint expression (B7-H3, PD-L1, PD-1) via immunohistochemistry in 125 archival TET specimens.
2. Retrospective analysis of Next Generation Sequencing (NGS) data, of FFPE-derived DNA from [n=5] TETs (TruSight Oncology 500 panel).
3. N GS performed on FFPE-derived TET sample DNA from the KHP BioBank [n=6] using the OncoDEEP™ Kit panel.

**Results**

1. Immune profiling identified four checkpoint phenotypes across TET subtypes: immune ignorance (B7-H3$^+$, PD-L1$^-$, PD-1$^-$) was prevalent in type A and AB thymomas; tolerance (B7-H3$^+$, PD-1$^+$) was observed in B2 and B3 thymomas; evasion (B7-H3$^+$, PD-L1$^+$, PD-1$^+$) associated with thymic carcinomas; and exclusion (B7-H3$^+$, PD-L1$^+$, PD-1$^-$) was enriched in AB and B2 thymomas. B7-H3 was overexpressed across all subtypes, with highest expression in high-grade tumours, supporting its candidacy as a therapeutic target.
2. Retrospective variant analysis identified TP53 DNA-binding domain variants (p.C176F and p.E286K), in two thymic carcinomas, with the latter co-occurring with a CDKN2A truncation (p.W110*). A separate metastatic thymoma harboured an NRAS p.Q61R mutation.
3. OncoDEEP profiling confirmed low tumour mutational burden (1.1–4.4 Mut/Mb), microsatellite stability, and absence of homologous recombination deficiency in all cases. Detected variants included NRAS p.Gly13Arg, a canonical MAPK-activating hotspot in a type A thymoma.. Two truncating KMT2C variants were detected in one AB thymoma, implicating epigenetic dysregulation as a mechanism of immune exclusion. Copy number analysis of a type A thymoma revealed gains in 1q and 6p, along with losses in 2q, 3p, 6q, 12p, and 22q, regions harbouring known tumour suppressor genes. These alterations may indicate early genomic instability despite histological classification.

**Conclusion** By mapping immune checkpoint profiles onto genomic alterations and histological subtypes, we reinforce the value of integrated immune-genomic subtyping in this rare malignancy. While limited by cohort size, these findings provide a rationale for biomarker-driven classification and therapeutic stratification in TETs.

## S65 EVALUATION OF PROTEIN X-TARGETING BISPECIFIC T CELL ENGAGER THERAPY IN PATIENT-DERIVED MESOTHELIOMA CELL CULTURES

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10.1136/thorax-2025-BTSabstracts.71

**Background** Pleural mesothelioma (PM) is a highly aggressive malignancy with limited therapeutic options and poor prognosis.[1] Emerging immunotherapeutic approaches, including bispecific T-cell engagers (BiTEs), offer promise by redirecting cytotoxic T cells to target tumour-associated antigens. Protein X, overexpressed in various malignancies including PM, represents a promising immunotherapeutic target.

**Methods** We investigated the efficacy of a novel protein X targeting BiTE construct in five patient-derived PM cell lines (three epithelioid, two biphasic) versus a control BiTE, each in triplicates. The BiTE X construct crosslinks CD3 on T cells with Protein X-expressing tumour cells. Surface expression of Protein X was confirmed via flow cytometry. Allogeneic T cells isolated from healthy donors were co-cultured with PM cells for 72 hours. T cell activation was assessed using antibodies against CD69, CD25, and CD137. Tumour cell cytotoxicity was measured via XTT viability assay, and a dose-response curve established optimal BiTE concentration. Brightfield microscopy visually confirmed mesothelioma cell killing.

**Results** High surface expression of protein X was confirmed across all cell lines. BiTE X induced robust, dose-dependent T cell activation and proliferation. After 72 hours, we observed cell clustering with BiTE X, but not control BiTE, indicating T cell activation. Flow cytometry at 72 hours showed high levels of endogenous T cell activation, ranging between 9%–91% CD25 (T cell activation marker) positivity in a dose-dependent manner. Both CD4+ and CD8+ T cells exhibited BiTE X- induced expression of CD25 and CD137 in all cell lines. Co-culture led to significant tumour cell lysis, with >70% cytotoxicity observed in some lines. No target cell killing or T cell activation occured in control or untreated samples.

**Conclusion** These findings support further evaluation of Protein X-directed BiTEs in unpurified pleural effusion cells, which reflect the tumour microenvironment and heterogeneity of real-world PM patients. Evaluating BiTE responses in this setting may identify subtypes or molecular features linked to therapeutic sensitivity or resistance, informing biomarker development. Ultimately, this work supports the development of personalised BiTE-based immunotherapy which may have the potential to improve clinical outcomes.

### REFERENCE

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## S66 A BINARY PROGNOSTIC SCORE, INCORPORATING FIBULIN-3, PREDICTS 1-YEAR SURVIVAL IN PLEURAL MESOTHELIOMA

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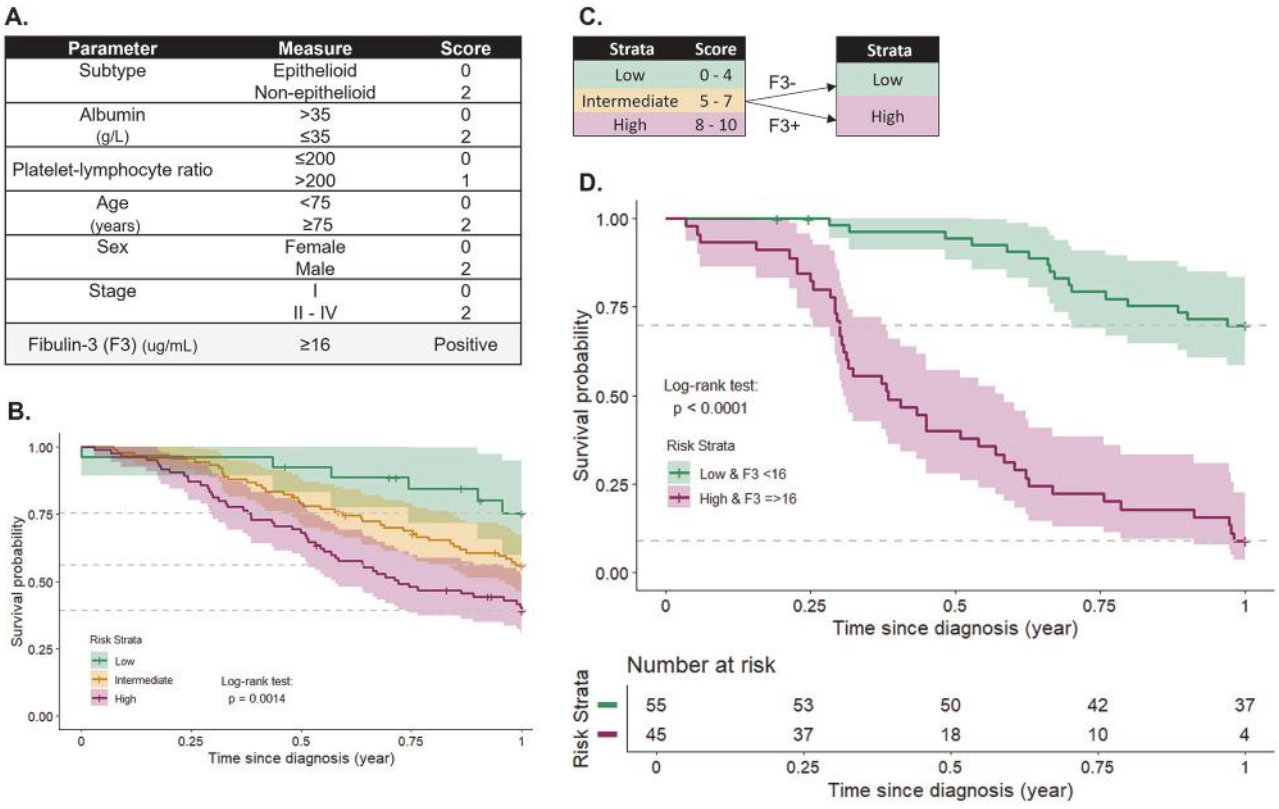
10.1136/thorax-2025-BTSabstracts.72

**Introduction** The Brims model classifies patients with pleural mesothelioma (PM) into 4 prognostic groups. However, at diagnosis, clinicians and patients commonly face a binary choice between immediate treatment or 'watch-and-wait'. The latter is high-risk if patients rapidly progress but reasonable if clinical progression is slow, since immediate treatment may result in unacceptable toxicity. We applied plasma Fibulin-3 data, which in isolation has yet to find a useful clinical role, to design and validate a binary prognostic model that could be used to inform clinical decision-making in this context. The study aspired to create a classifier with prognostic performance that was at least equal to the Brims model using data from the multicentre DIAPHRAGM study (Tsim, JTO 2021) and Scottish National MDT.

**Methods** We first performed a retrospective analysis of prospectively recorded DIAPHRAGM data (n=749, 23 UK centres). Using 100 recruits with a final diagnosis of PM and baseline plasma Fibulin-3 data, we defined a 3-group 1-yr survival model based on hazard ratios generated by multivariable Cox regression. This defined clear high/low-risk groups, plus an intermediate group for stratification using Fibulin-3. Next, we externally validated the performance of the 3-group model in an independent set of 203 Scottish National MDT cases. Finally, to create the intended binary model, we stratified intermediate-risk cases in the validated 3-group model by baseline Fibulin-3, generating the Glasgow Mesothelioma Score (GMS).

**Results** Six independent features contributed to the initial 3-group DIAPHRAGM model (figure 1a), which delivered superior performance to Brims (C=0.72 v 0.66). The 3-group model was successfully validated in the Scottish MDT dataset, with significant prognostic performance retained (C=0.60, 1-yr survival 38%, 56%, 75% in high/intermediate/low-risk; figure 1b). In the final, binary GMS model, stratification of intermediate-risk cases by Fibulin-3 (dichotomised using an optimal threshold of 16µg/mL), improved performance (C=0.73, 1-yr survival 70% v 9% in high/low risk; figure 1c-d).

**Conclusion** The GMS combines Fibulin-3 with an externally validated 3-group model to create a powerful binary classifier. This model may be useful in identifying patients unsuitable for 'watch-and-wait'. Ongoing work includes further external validation of the final binary GMS using international cohorts with available Fibulin-3 data.




**Abstract S66 Figure 1**

S67

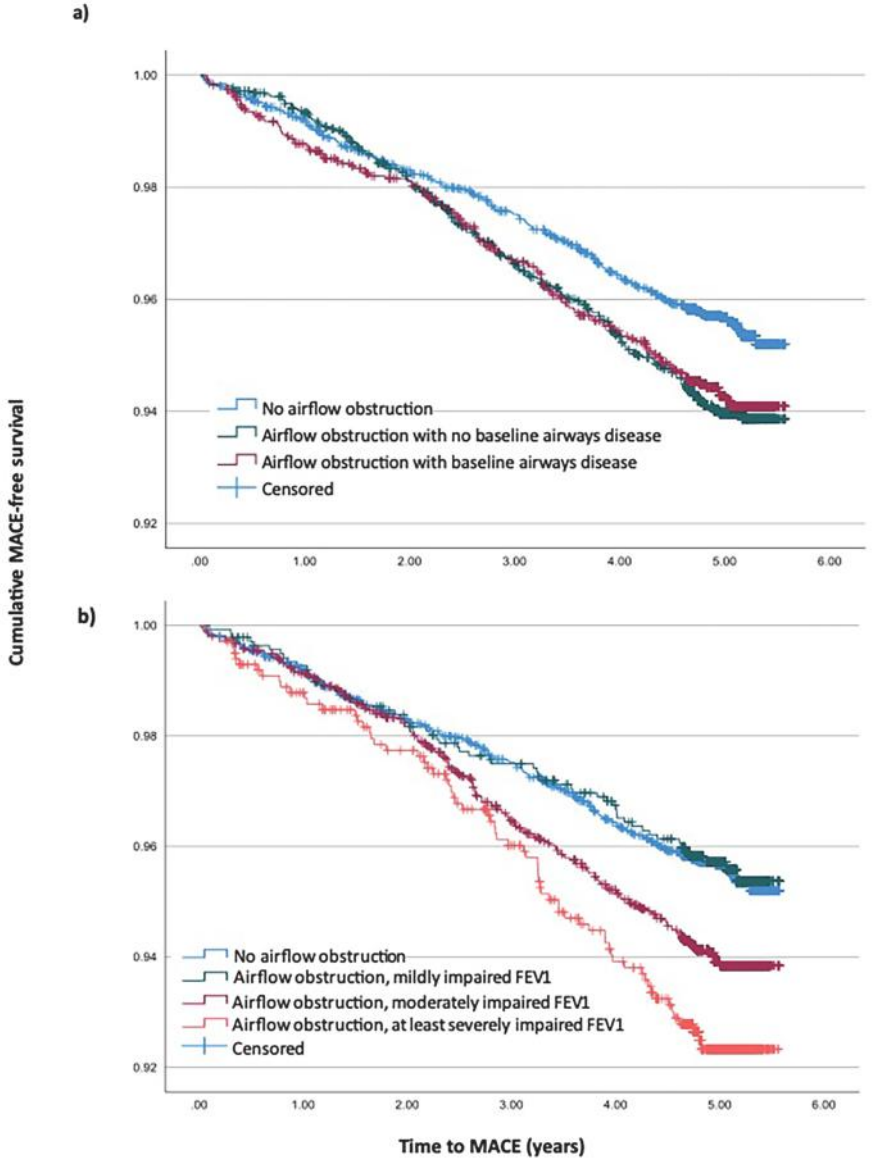
## ASSOCIATION OF AIRFLOW OBSTRUCTION IDENTIFIED AT LUNG CANCER SCREENING WITH MAJOR ADVERSE CARDIOVASCULAR EVENTS

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**Introduction** Lung cancer and cardiovascular disease are leading causes of death in individuals with COPD. Lung cancer screening (LCS) reduces lung cancer related mortality and offers an opportunity to identify those with undiagnosed airflow obstruction. However, there is limited evidence linking airflow obstruction with cardiovascular morbidity and mortality in LCS cohorts.

**Methods** Quality-assured handheld pre-bronchodilator spirometry was performed during baseline SUMMIT study (NCT03934866) LCS appointments. The best of three forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) values were recorded, with airflow obstruction defined as an FEV1/FVC ratio of <0.7. GOLD criteria were used to categorise airflow obstruction severity. Baseline airways disease was either self-reported or obtained from primary care records. Hospital Episode Statistics and National Cancer Registration and Analysis Service data identified MACE (acute myocardial infarction, stroke, cardiovascular death, unstable angina and heart failure). Kaplan-Meier and Cox regression analyses were used to quantify the association between airflow




**Abstract S67 Figure 1** Kaplan-Meier cumulative major adverse cardia event (MACE)-free survival **a)** by airflow obstruction in those with and without baseline airways disease and **b)** by severity of airflow obstruction

obstruction and MACE. Covariates included age, sex, smoking status, smoking history, BMI, coronary artery calcification and baseline cardiovascular disease.

**Results** 11,025 participants underwent spirometry. Median age was 65 (IQR 60–70 years). 58% (n=6337/11025) were men, 49% (n=5346/11025) current smokers and 31% (n=3414/11025) had baseline airways disease. 49% (n=5449/11025) had obstructive spirometry at LCS, 58% of whom (n=3148/5449) had no baseline airways disease.

5% (n=552/11,025) of participants had at least one MACE over a median follow-up of 5.0 years (IQR 4.8–5.2). Kaplan-Meier analyses demonstrated that regardless of baseline airways disease, airflow obstruction was associated with worse MACE-free survival compared to no airflow obstruction (figure 1a) and increasing severity of airflow obstruction was associated with worse cardiovascular outcomes (figure 1b). In multivariable Cox regression analysis, airflow obstruction was independently associated with an increased risk of MACE (aHR 1.22 (95%CI 1.02–1.45)) compared to no airflow obstruction.

**Discussion** Airflow obstruction is common in LCS populations and associated with an increased risk of MACE. LCS provides an enriched population at high risk of poor cardiovascular outcomes due to overlapping risk factors. Identifying airflow obstruction in this population may offer an opportunity to more comprehensively assess cardiovascular risk, enabling implementation of targeted prevention strategies.

# Cystic fibrosis: impact of CFTR modulators

## S68 LONG-TERM EFFECTS OF ELEXACAFTOR/TEZACAFTOR/IVACAFTOR ON CLINICAL OUTCOMES AND QUALITY OF LIFE AMONG ADOLESCENTS AND ADULTS AGED 12+ WITH CYSTIC FIBROSIS: INTERIM RESULTS FROM THE TRAJECTORY STUDY

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10.1136/thorax-2025-BTSabstracts.74

**Introduction** TRAJECTORY is an ongoing, observational, medical record (MR) abstraction study in the United Kingdom, Canada, Germany, Italy, Portugal and Spain among people with CF (PwCF) who initiated elexacaftor/tezacaftor/ivacaftor (ELX/TEZ/IVA) August 2019 – June 2023. We assessed long-

**Abstract S68 Table 1** Summary of Mean CFQ-R Domains at Baseline and Change from Baseline

| CFQ-R Domain[1] | Patients Aged ≥ 12 with at least 12 months post-ELX/TEZ/IVA initiation (N = 508) | | | | Patients Aged ≥ 12 with at least 24 months post-ELX/TEZ/IVA initiation (N = 403) | | | | Patients Aged ≥ 12 with at least 36 months post-ELX/TEZ/IVA initiation (N = 189) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | Baseline[2] Mean (SD) | n | Average absolute change[3] from baseline to 0-12 months Mean (SD) | n | Baseline[2] Mean (SD) | n | Average absolute change[3] from baseline through 24 months Mean (SD) | n | Baseline[3] Mean (SD) | n | Average absolute change[3] from baseline through 36 months Mean (SD) |
| Respiratory Symptoms | 290 | 63.0 (21.7) | 258 | +23.4 (20.1) | 210 | 61.9 (21.7) | 202 | +23.5 (19.2) | 74 | 58.4 (22.7) | 71 | +26.3 (19.3) |
| Body Image | 290 | 70.0 (24.9) | 260 | +7.9 (17.1) | 210 | 70.7 (24.8) | 204 | +8.3 (18.4) | 74 | 69.1 (24.8) | 71 | +8.5 (21.2) |
| Digestive Symptoms | 290 | 77.8 (19.9) | 257 | +4.4 (17.2) | 210 | 77.9 (19.8) | 203 | +3.7 (16.1) | 74 | 76.7 (21.3) | 71 | +4.1 (17.3) |
| Eating Problems | 290 | 86.7 (20.2) | 260 | +5.6 (16.9) | 210 | 88.6 (19.5) | 204 | +4.3 (17.6) | 74 | 87.7 (20.9) | 71 | +5.1 (20.6) |
| Emotional Functioning | 290 | 73.6 (20.5) | 260 | +6.6 (15.7) | 210 | 73.9 (20.4) | 204 | +5.8 (16.3) | 74 | 71.3 (19.8) | 71 | +7.7 (15.5) |
| Health Perceptions | 267 | 57.4 (24.6) | 244 | +15.4 (20.3) | 200 | 56.1 (24.7) | 195 | +16.2 (19.5) | 73 | 50.4 (24.9) | 70 | +15.3 (18.3) |
| Physical Functioning | 289 | 68.9 (25.7) | 259 | +13.8 (20.8) | 210 | 67.6 (25.9) | 204 | +13.7 (19.1) | 74 | 64.3 (24.3) | 71 | +12.2 (19.6) |
| School Functioning | 265 | 77.9 (20.8) | 240 | +8.3 (15.8) | 198 | 77.1 (20.6) | 192 | +8.0 (15.4) | 71 | 73.0 (20.9) | 69 | +8.2 (16.5) |
| Social Functioning | 290 | 64.3 (19.8) | 260 | +8.9 (15.7) | 210 | 63.1 (19.2) | 204 | +10.4 (15.6) | 74 | 58.2 (16.9) | 71 | +12.0 (16.2) |
| Treatment Burden | 289 | 59.6 (22.5) | 259 | +8.3 (16.1) | 210 | 59.8 (22.8) | 204 | +8.4 (17.6) | 74 | 59.6 (22.1) | 71 | +9.1 (14.4) |
| Vitality | 266 | 57.7 (21.9) | 243 | +11.5 (19.6) | 200 | 56.9 (22.7) | 195 | +9.8 (18.6) | 73 | 53.3 (22.0) | 70 | +9.7 (17.6) |
| Weight | 266 | 70.9 (36.0) | 241 | +11.7 (30.5) | 200 | 72.0 (35.9) | 193 | +9.7 (32.3) | 73 | 79.9 (29.8) | 70 | +6.5 (29.3) |

CFQ-R: Cystic Fibrosis Questionnaire – Revised; ELX/TEZ/IVA: elexacaftor/tezacaftor/ivacaftor; SD, standard deviation. Please note that this is a medical chart abstraction study where CFQ-R is collected as part of standard of care. Not all sites collected the CFQ-R as part of the standard of care. The counts vary by domain and study duration. Each domain yields a standardized score ranging from 0–100, with higher scores indicating better health-related quality of life. [1]For the analyses of CFQ-R domains Respiratory Symptoms, Body, Digestion, Eating Problems, Emotional Functioning, Physical Functioning, Social Functioning and Treatment Burden, CFQ-R data from patients aged 12 – 13 and data from patients aged ≥ 14 were pooled. [2]For the analyses of CFQ-R domains Respiratory Symptoms, Body, Digestion, Eating Problems, Emotional Functioning, Physical Functioning, Social Functioning and Treatment Burden, CFQ-R data from patients aged 12 – 13 and data from patients aged ≥ 14 was pooled. [2]Start of ELX/TEZ/IVA therapy is defined as the study baseline. [3]Average absolute change through the post-baseline is calculated as the average of all measurements during the post-baseline period minus baseline. Measurements within four weeks following ELX/TEZ/IVA initiation are excluded.

term effects of ELX/TEZ/IVA on clinical outcomes and quality of life (QoL) in PwCF aged ≥12 years with a minimum of 12 months and up to 36 months of ELX/TEZ/IVA treatment.

**Methods** MR data from 42 sites were collected for 12 months before ELX/TEZ/IVA initiation (baseline) and up to 5 years after (follow-up). Outcomes included body mass index (BMI), percent predicted forced expiratory volume in one second ($ppFEV_1$), annualized event rates of PEx and hospitalizations, and QoL (self-report using the Cystic Fibrosis Questionnaire-Revised [CFQ-R; Child 12–13, Teen-Adult 14+]). Descriptive analyses were stratified by prior CF transmembrane conductance regulator modulator (CFTRm) use and baseline $ppFEV_1$. Outcomes were evaluated at 12 months, 24 months, and 36 months post-ELX/TEZ/IVA initiation.

**Results** Of 508 PwCF aged ≥12 years with ≥12 months of follow-up, ~50.0% were female (49.6%, 252/508), homozygous for *F508del-CFTR* (50.0%, 254/508), and had prior CFTRm use (48.4%, 246/508). At ELX/TEZ/IVA initiation, mean (SD) age, $ppFEV_1$, and BMI were 30.4 (12.8) years, 63.2 (24.0) and 21.5 (3.6) $kg/m^2$. At initiation, 58.5% had $ppFEV_1$ between 40 and 90 ($ppFEV_1$ ≥40 to <70: 36.8%, 187/508; $ppFEV_1$ ≥70 to ≤90: 21.7%, 110/508). After 36 months, ELX/TEZ/IVA treatment led to mean (SD) increases in BMI (+1.9 $kg/m^2$ [2.2]) and $ppFEV_1$ (+9.3 percentage points [10.1]), and decreases in annualized event rates of PEx (0.4 to 0.3) and hospitalizations (1.0 to 0.5).

Regardless of prior CFTRm use, all CFQ-R domain mean scores showed improvement at 12 months, 24 months, and 36 months. The largest domain increases in mean (SD) absolute change from baseline at 36 months were Respiratory Symptoms (26.3 [19.3]), Health Perceptions (15.3 [18.3]), Physical Functioning (12.2 [19.6]), School Functioning (+12.0 [16.2]), Vitality (9.7 [17.6]) and Treatment Burden (9.1 [14.4]) (table 1).

**Conclusions** ELX/TEZ/IVA treatment showed substantial benefits in clinical outcomes and QoL in the real-world setting with sustained improvement up to at least 36 months for PwCF aged ≥12 years.

## S69 REDUCTION IN SERUM MARKERS OF INFLAMMATION IN PEOPLE WITH CYSTIC FIBROSIS WHO ARE CHRONICALLY INFECTED WITH PSEUDOMONAS AERUGINOSA IS SEEN AFTER TREATMENT WITH ELEXACAFTOR/TEZACAFTOR/IVACAFTOR

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**Objectives** To evaluate changes in systemic inflammation in people with (pw) CF chronically infected with Pseudomonas aeruginosa (Pa) after ≥6 months of the CFTR modulator, Elexacaftor/Tezacaftor/Ivacaftor (ETI).

**Methods** Retrospective review of pwCF at Royal Brompton Hospital with Leeds-defined chronic Pa infection ≥1 of the 2 years pre-ETI. Serum inflammatory markers (immunoglobulin G (IgG), immunoglobulin A (IgA), immunoglobulin M (IgM), C-reactive protein (CRP), white cell (WCC), neutrophil, lymphocyte, and eosinophil counts) taken at annual assessment were assessed at baseline (closest prior to starting ETI) and paired to values after ETI start (6–24 m). Subanalysis by sex, genotype (homozygous F508del vs. others), persistence of airway sample Pa positivity, and percent predicted forced expiratory value in one second ($ppFEV_1$) analysis performed using either paired t-test or Mann-Whitney test.

**Abstract S69 Table 1**

| Inflammatory Marker | Absolute reduction | P value | Percentage reduction | P value |
|---|---|---|---|---|
| IgG (6.4–16.0 g/L) | - 2.1 g/L | <0.001 | **16.4%** | **<0.001** |
| IgA (0.7–3.2 g/L) | -0.3 g/L | <0.001 | **15.3%** | **<0.001** |
| IgM (0.5–2.8 g/L) | - 0.1 g/L | <0.001 | **9.0%** | **<0.001** |
| CRP (0–5 mg/L) | - 2.0 mg/L | <0.01 | 50.0% | ns |
| WCC (5.1 – 11.4 $x10^9$/L) | - 1.7 $x10^9$/L | <0.001 | **20.5%** | **<0.001** |
| Neutrophils (2.6–7.9 $x10^9$/L) | -1.5 $x10^9$/L | <0.001 | **26.3%** | **<0.01** |
| Lymphocytes (1.3–3.7 $x10^9$/L) | -0.1 $x10^9$/L | ns | 4.8% | ns |
| Eosinophils (0.1–0.5 $x10^9$/L) | 00 $x10^9$/L | ns | 0.0% | <0.01 |

**Results** In 168 patients included, there was reduction in absolute IgG/A/M, CRP, WCC and neutrophil count after ETI initiation. Analysis as a proportion of baseline showed reduction in IgG/A/M, WCC and neutrophils with no difference for CRP, lymphocytes and eosinophils (table 1). Subanalysis showed no difference in observed change by genotype or sex.

120 patients had complete paired data for annual Pa culture frequency and inflammatory markers 1 year before and 2 years after starting ETI. At their year 2 annual assessment, 23% of patients were no longer growing Pa in the sample provided; a subanalysis showed no difference in observed inflammatory marker change based on this Pa status.

$ppFEV_1$ showed a median(IQR) increase of 9.62 (1.71,23.88)% ($p<0.001$) post-ETI. Patients with $ppFEV_1$ increase >10%, compared to those without increase, showed greater neutrophil reduction (-2.9 vs -0.7 x $10^9$/L, $p=0.04$), with no difference noted for other inflammatory markers.

**Conclusion** In those with chronic Pa infection, taking ETI for at least 6 months was associated with a reduction from baseline in systemic inflammation. When we sub-analysed by sex, genotype, and clearance of Pa in the sample provided, we did not see inflammatory marker differences. Those with a >10% improvement in $ppFEV_1$ experienced greater reduction in neutrophil count, suggesting an association between change in systemic inflammation and airway health on ETI. Post-ETI inflammatory change and its impact warrants further exploration.

## S70 OXYGEN ENHANCED (OE-)MRI AND MULTIPLE BREATH WASHOUT WITH SHORT EXTENSION (MBWSHX) REVEAL LUNG DISEASE PROGRESSION IN PEOPLE WITH CYSTIC FIBROSIS DESPITE TRIPLE CFTR MODULATOR THERAPY

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**Introduction** The post modulator era is raising several new challenges, particularly with a need for sensitive pulmonary outcome measures (OM). Current tools are suboptimal; to

**Abstract S70 Table 1** Pulmonary function parameters including values from oxygen enhanced (OE-)MRI at baseline, 6 months, 12 months and 18 months as well as deltas generated between follow up visits and the baseline visit

| Parameter | Baseline (N=36) Mean (SD) | 6 months (N=32) Mean (SD) | Δ (95%CI) P value | 12 months (N=32) Mean (SD) | Δ (95%CI) P value | 18 months (N=28) Mean (SD) | Δ (95%CI) P value |
|---|---|---|---|---|---|---|---|
| $ppFEV_1$ | 89.5% (±19.4%) | 88.9% (±19.2%) | -0.5% (-3.8 to 2.7) P>0.05 | 88.7 (±19.7%) | -0.8% (-4.0 to 2.4%) P>0.05 | 88.1% (±19.2%) | -1.3% (-4.6 to 2.0%) P>0.05 |
| $LCI_{2.5}$ | 9.1 (±4.2) | 9.1 (±4.0) | 0.02 (-0.62 to 0.58) P>0.05 | 9.1 (±3.9) | 0.02 (-0.61 to 0.57) P>0.05 | 9.4 (±3.9) | 0.25 (-0.36 to 0.87) P>0.05 |
| $LCI_{ShX}$ | 12.7 (±9.0) | 13.3 (±9.8) | 0.58 (-0.5 to 1.7) P>0.05 | 13.6 (±9.7) | 0.91 (-0.2 to 2.0) P>0.05 | 14.2 (±8.5) | 1.54 (0.5 to 2.53) **P<0.01** |
| UVLU | 3.6 (±5.2) | 4.2 (±6.3) | 0.62 (-0.3 to1.6) P>0.05 | 4.5 (±6.0) | 0.90 (0.03 to 1.74) **P<0.05** | 4.7 (±4.6) | 1.1 (0.2 to 1.98) **P<0.01** |
| $\Delta R_2^*{}_{MS-1}$ | 0.076 (±0.025) | 0.072 (±0.025) | -0.004 (-0.010 to 0.002) P>0.05 | 0.069 (±0.025) | -0.006 (-0.013 to 0.00) **P<0.05** | 0.065 (±0.025) | -0.011 (-0.017 to 0.00) **P<0.001** |
| VDP% | 8.2% (±7.7%) | 9.9% (±8.9%) | 1.6% (0.3 to 3.6%) P>0.05 | 10.6% (±8.4%) | 2.5% (0.6 to 4.4%) **P<0.01** | 12.7% (±10.6%) | 4.5% (2.5 to 6.6%) **P<0.0001** |

address this our group developed $MBW_{ShX}$ to assess previously overlooked under ventilated lung units (UVLU), but it cannot provide spatial information. Functional lung MRI has the potential to be a sensitive OM to track CF lung disease. We hypothesised that in the context of improved clinical status, any lung disease progression would be observed more clearly using OE-MRI and $MBW_{ShX}$ than conventional measures.

**Methods** PwCF were recruited as part of a large observational study to determine the clinimetric properties of OE-MRI; all were on ETI therapy (>6 months at baseline). Participants performed OE-MRI, $MBW_{ShX}$, and spirometry whilst clinically stable on the same day at baseline and at 6-monthly intervals over 18 months. *OE-MRI parameters*: ventilation defect percentage (VDP) and $\Delta R_2^*$ (ventilation signal). *$MBW_{ShX}$ parameters*: UVLU and $LCI_{ShX}$ ($LCI_{2.5}$ + UVLU) a measure of global lung health. Data is presented as mean (SD) and Δbaseline (95%CI) and assessed using a mixed effects model with Dunnett's test for multiple comparisons.

**Results** Thirty-six pwCF aged 20.3 (±12.7) completed the baseline visit. $FEV_1$ and $LCI_{2.5}$ appeared stable, whereas significant deterioration in the novel $MBW_{ShX}$ and MRI parameters could be observed (table 1).

**Conclusions** OE-MRI and $MBW_{ShX}$ are novel, sensitive markers of abnormalities in lung structure and function not detected by conventional outcomes ($FEV_1$ and $LCI_{2.5}$) and demonstrate potential as clinical outcome measures. Reliance on conventional OMs may lead to false reassurance about disease stability over time; particularly in relation to assessing safe withdrawal of standard treatments.

Funded by CF Foundation.

## S71 PRE-CONCEPTION LUNG FUNCTION AND THE PREGNANCY OUTCOMES AMONG FEMALES WITH CYSTIC FIBROSIS (FWCF) IN THE CF-TRANSMEMBRANE CONDUCTANCE REGULATOR MODULATOR (CFTRM) ERA

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**Introduction** Since widespread availability of highly effective CFTRm elexacaftor/tezacaftor/ivacaftor, pregancy rates have doubled among fwCF in the UK.[1]

Historically, CF-pregnancy has been high risk, and guidelines from pre-CFTRm era considered pre-conception FEV1<50% and/or pulmonary hypertension absolute contraindications,[2] with lower lung function correlating with worse maternal and fetal outcomes including pre-term (<37 weeks) births.

With improved female CF subfertility and general health the association of pre-conception lung function and obstetric outcomes warrants renewed investigation.

**Method** Retrospective electronic health care records review of fwCF who completed pregnancies in a large, dedicated CF-Maternal Health Service September 2020-June 2025. Analysis of pregnancy outcomes according to pre-conception ppFEV1 impairment: Severe<50%, moderate 50–80%, mild >80%.

**Results** There were 106 pregnancies over the time period: 16 terminations of pregnancy (1 for fetal abnormalities, 15

**Abstract S71 Table 1**

| | ppFEV1 >80% (n=31) | ppFEV1 50-80% (n=31) | ppFEV1 <50% (n=9) | p-value |
|---|---|---|---|---|
| Mean ppFEV1 pre-conception mean ±SD) | 97% ± 15 | 65% ± 7 | 42% ± 4 | <0.001 |
| Mean age (yrs, mean ±SD) | 31 ± 4.7 | 31 ± 5.3 | 27.8 ± 5.8 | 0.17 |
| CFTRm (all or part of pregnancy) n(%) | 21 (68%) | 28 (90%) | 7 (78%) | 0.35 |
| Pre-term birth (<37 weeks) | 5 (16%) | 8 (26%) | 3 (33%) | 0.67 |
| Gestation at delivery (weeks +days, mean ±SD) | 37+5 ±10 days | 37+3 ± 15 days | 36+4 ± 11 days | 0.24 |
| Hospital admissions for IECF during pregnancy | 5 (16%) | 10 (32%) | 5 (56%) | 0.03 |
| PE during pregnancy | 0 | 1 | 0 | 1 |
| Massive Haemoptysis in pregnancy | 0 | 0 | 0 | 1 |
| Pneumothorax in pregnancy | 0 | 0 | 0 | 1 |
| c-section delivery | 13 (42%) | 15 (48%) | 7 (78%) | 0.25 |

maternal choice); 19 1st trimester miscarriages; and 71 completed pregnancies (53 fwCF) with 73 live births (2 sets of twins).

There were no maternal deaths or critical care admissions during pregnancy, and no infant deaths. Admissions for pulmonary exacerbations occurred in 20 pregnancies (28%). Most fwCF continued CFTRm throughout pregnancy in, of the 12 who paused CFTRm 9 (75%) restarted due to significant health decline.

The majority of pregnancies (40/71, 56%) were in fwCF with moderate-severe FEV1 impairment. Severe lung function impairment was associated with a greater risk of antenatal IECF requiring hospitalisation.

**Conclusion** CF-pregnancies are increasingly prevalent among fwCF previously cautioned against pregnancy. Overall reassuring outcomes among fwCF with moderate-severe spirometric impairment highlights the importance of continued health stabilisation conferred by CFTRm and dedicated specialist CF-pulmonary and obstetric support in this cohort, as they are still more likely to deliver pre-term and require admission to hospital during pregnancy. Research is ongoing to define optimal CF-obstetric management strategies and update the evidence base.

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## S72 USE OF CARDIOVASCULAR RISK SCORES IN ADULT POPULATIONS WITH CYSTIC FIBROSIS


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10.1136/thorax-2025-BTSabstracts.78

**Introduction** Advancements in Cystic Fibrosis (CF) transmembrane conductance regulator modulators have significantly

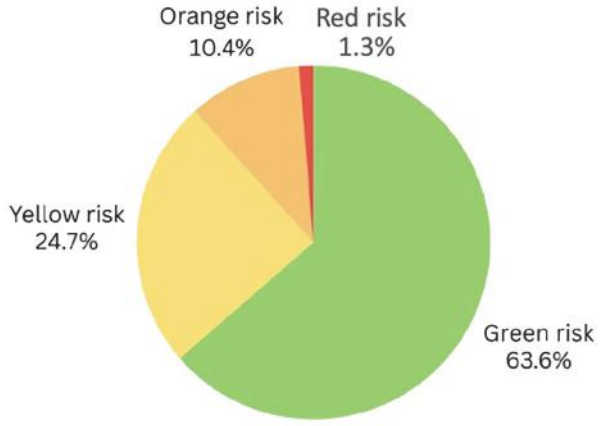



Figure 1. WHO CVD Risk score
*Green <5%, Yellow 5 to <10%, Orange 10 to <20%, Red 20 to <30%

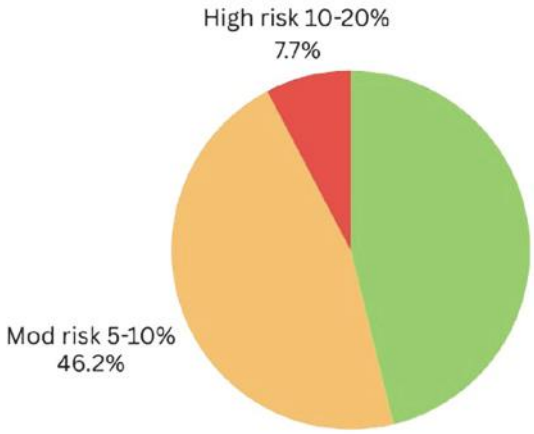



Figure 2. SCORE2-Diabetes

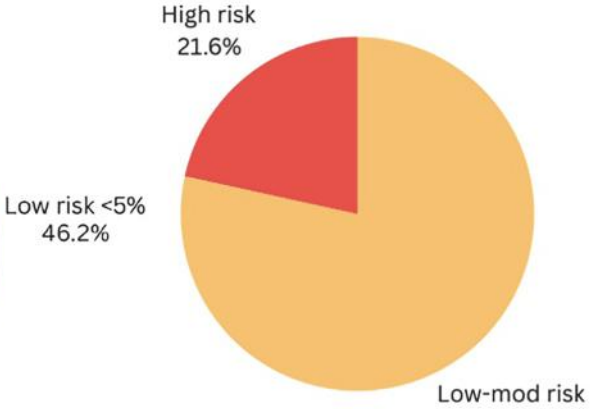



Figure 3. SCORE2
*Low-mod risk: <2.5% in <50y, <5% in 50-69y
High risk: 2.5 to <7.5% in <50y, 5 to <10% in 50-69y

**Abstract S72 Figure 1**

increased the life expectancy of people living with CF (PwCF). The increased longevity, changes in body composition and increased prevalence of diabetes and chronic kidney disease in this cohort has led to emerging concerns about their potential increased risk of cardiovascular disease (CVD).

**Aims** We sought to assess the CVD risk in PwCF aged ≥40, and to determine if they are appropriately receiving lipid-lowering therapy in line with current guidelines.

**Methods** We retrospectively calculated the 10-year CVD risk scores of PwCF aged ≥40 attending our National Referral Centre for Adults with CF between 2020 and 2023. This was calculated using the European Society of Cardiology (ESC) SCORE2 and SCORE2-Diabetes (for those with CF-related diabetes, CFRD) algorithms, and the World Health Organisation (WHO) CVD risk prediction charts. Clinical data were collected from patients' most recent Annual Review documents. Age of diabetes diagnosis was standardized as five years prior to the assessment year.

**Results** Of the 427 PwCF attending our CF unit, 100 were aged ≥40, and 77 had complete data for risk score calculation. Mean age was 47.6 years (SD ±6.1), 63.6% were male and 33.8% had CFRD. Using the WHO risk prediction chart, 63.6% of the 77 PwCF were classified as green-risk, 24.7% as yellow-risk, 10.4% as orange-risk, and 1.3% as red-risk. Of the 26 PwCF with CFRD, SCORE2-Diabetes classified 46.2% as low-risk, 46.2% as moderate-risk, and 7.6% as high-risk. Of the 51 PwCF without CFRD, SCORE2 classified 78.4% as low-to-moderate-risk and 21.6% as high-risk. Based on ESC guidelines, 19.5% of the included PwCF would be candidates for lipid-lowering therapy. However, only 9.1% were prescribed either a statin or ezetimibe.

**Conclusion** A significant proportion of our cohort met criteria for lipid-lowering therapy, but few were on appropriate treatment, suggesting potential under-recognition of CVD risk in this cohort. Challenges such as co-existing liver disease and polypharmacy may also influence prescribing practices. Given the evolving demographics and risk profiles of PwCF, further research is warranted to validate risk prediction tools and tailor cardiovascular risk management strategies for this unique cohort.

## S73 EPIDEMIOLOGY OF CYSTIC FIBROSIS-RELATED DEATHS IN THE UNITED STATES, 1999–2023: A CDC WONDER-BASED STUDY

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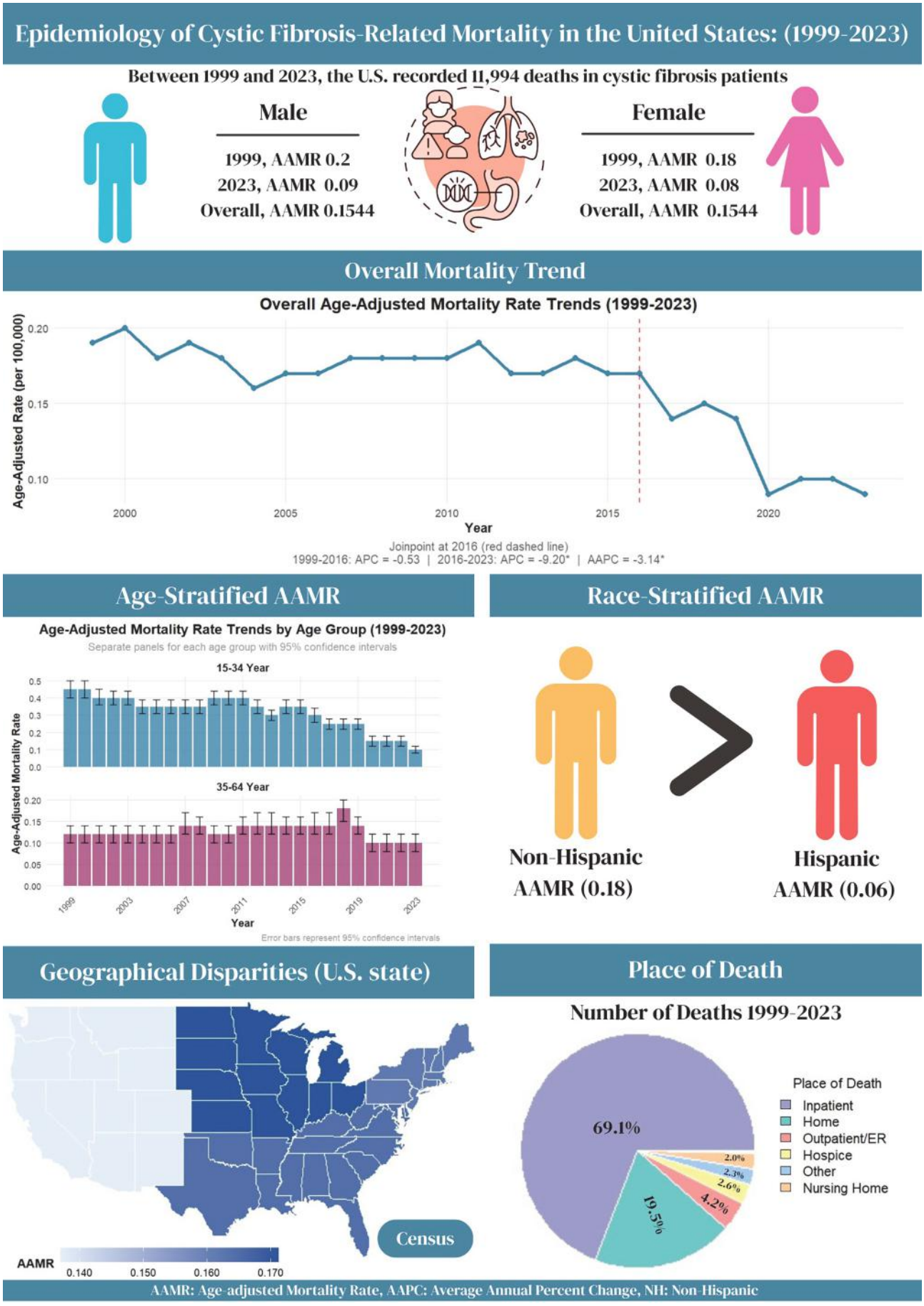
10.1136/thorax-2025-BTSabstracts.79

**Background** Cystic fibrosis (CF) is a hereditary multisystem disorder predominantly affecting the respiratory system, contributing significantly to morbidity and mortality in the developed nations, despite advancement in treatments. Characterizing mortality patterns across demographic and geographic populations is essential for developing targeted prevention and management strategies. Therefore, we analyzed temporal mortality trends in cystic fibrosis across diverse populations in the United States from 1999–2023.

**Methods** Data were extracted from the CDC WONDER database (1999–2023) to identify mortality rates among individuals aged ≥1 year with CF listed as the underlying cause of death (ICD-10: E84.0, E84.1, E84.8, E84.9). Age-adjusted mortality rates (AAMRs) per 100,000 population were calculated and stratified by sex, age group, race/ethnicity, geographic region, and place of death. Temporal trends were analyzed using Joinpoint regression to estimate average annual percent change (AAPC).

**Results** From 1999–2023, 11,997 deaths were reported among individuals with CF across all age groups (<1 to ≥65 years). The AAMR decreased significantly from 0.19 in 1999 to 0.09 in 2023 (AAPC: -3.1%; p<0.000001), with accelerated decline in recent years. Both sexes demonstrated comparable average AAMRs (0.15), though with different rates of decline: men (AAPC: -2.7%; 95% CI: -4.17 to -1.29; p=0.0002) and women (AAPC: -2.9%; 95% CI: -4.19 to -1.66; p=0.000008). By race/ethnicity, non-Hispanic populations exhibited higher overall AAMRs compared to Hispanics (0.18 vs. 0.06), with incongruent trends observed among non-white races and Hispanic populations. Regionally, the Midwest recorded the highest AAMR (0.17), followed by the South (0.16), Northeast (0.15), and West (0.13). Age-stratified analysis revealed peak mortality in the 15–34 years group (0.32/100,000), followed by the 35–64 years group (0.12/100,000). Most deaths occurred in inpatient medical facilities (8,260 deaths; 68.8%).

**Conclusion** CF mortality rates declined significantly over two decades, with comparable reductions in both sexes. However, substantial disparities persist, with young adults (15–34 years) experiencing high mortality rates and notable racial/ethnic differences. Regional disparities were evident across geographic areas. While these findings suggest improved CF management and care, they underscore the critical need for targeted interventions addressing persistent demographic and geographic disparities to ensure equitable outcomes across all populations.




**Abstract S73 Figure 1**

# Sleep apnoea and beyond

## S74 USEFULNESS OF OSLER FOR PATIENT AND CLINICIAN: 5-YEAR DATA FROM A TERTIARY SLEEP SERVICE

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10.1136/thorax-2025-BTSabstracts.80

**Introduction** Excessive daytime sleepiness (EDS) in obstructive sleep apnoea (OSA) has important public health implications. The Oxford Sleep Resistance Test (OSLER) is a tool in sleep medicine to assess fitness to drive, by evaluating a subject's daytime vigilance and ability to maintain wakefulness, and Sheffield Teaching Hospitals provides a tertiary sleep service, including OSLER, for patients with OSA.

**Aims and Objectives** This retrospective cohort study evaluates use of OSLER in assessing fitness to drive and its real-life implications.

**Methods** We analysed data over a 5-year period and recorded sleep diagnostics, clinical information, Epworth Sleepiness Score (ESS), OSLER data, and DVLA outcome. Follow-up phone calls were conducted to assess present-day symptoms and implications of OSLER on driving, lifestyle, and occupation.

**Results** We identified 26 patients with OSA who were referred for OSLER over a 5-year period.

Four patients failed OSLER, but one patient disputed their result and passed a repeat test with no errors. One patient had difficulties understanding OSLER principles and had multiple attempts. He subsequently failed, but was recorded as a lack of concentration rather than a microsleep. One patient who failed OSLER was lost to follow up, but later reported severe ongoing symptoms and was unable to work as a lorry driver, leading to financial difficulties and mental health problems.

In 13/26 patients, DVLA were notified of sleep investigations. In 6/26 patients, it is unclear whether DVLA were contacted, and one of these failed OSLER. Over half the patients continued to drive with no recorded accidents or near misses. Overall, two patients had their driving license revoked.

**Conclusion** Majority of patients passed OSLER, but interpretation was subjective and open to patient opinion. ESS, EDS whilst driving, and AHI were poorly predictive of OSLER result, but the majority who failed OSLER had severe OSA. Patient self-awareness of sleepiness while driving does not appear to correlate with OSLER outcomes. We need a better way of assessing driving risk (e.g. driver-specific questionnaire and modern driving simulator) and clearer guidance from DVLA, as outcomes have implications on patient occupation and well-being.

**Abstract S74 Table 1** Demographic and sleep diagnostics results in the 'passed OSLER' and 'failed OSLER' patient groups

| | | | Passed OSLER (n=22) | Failed OSLER (n=4) |
|---|---|---|---|---|
| Demographics | Mean age at initial assessment (years) | | 55.5 | 61.8 |
| | Mean BMI | | 34.4 | 33.7 |
| | Gender (n=males) | | 15 (68%) | 2 (50%) |
| | Number of occupational drivers | | 9 (40.1%) | 2 (50%) |
| Sleep diagnostics | Severity of OSA | Mild | 4 (18.0%) | 0 |
| | | Moderate | 6 (27.3%) | 1 (25.0%) |
| | | Severe | 12 (54.5%) | 3 (75.0%) |
| | Mean baseline ESS | | 12.3 | 12.0 |
| | Feels tired when driving? | | 3 (13.6%) | 1 (25.0%) |
| | History of road traffic accident/near miss | | 0 | 1 (25.0%) |
| | Mean Apnoea-Hypnoea Index (AHI) | | 28.5 | 29.5 |
| | Median Oxygen desaturation index (ODI) | | 37.6 | 53.5 |

## S75 AN EXPLORATORY STUDY OF A NOVEL, LOW-COST, SIMPLE SLEEP APNOEA SCREENING DEVICE, APNESCAN DC1, TO IDENTIFY APNOEAS AND HYPOPNEAS

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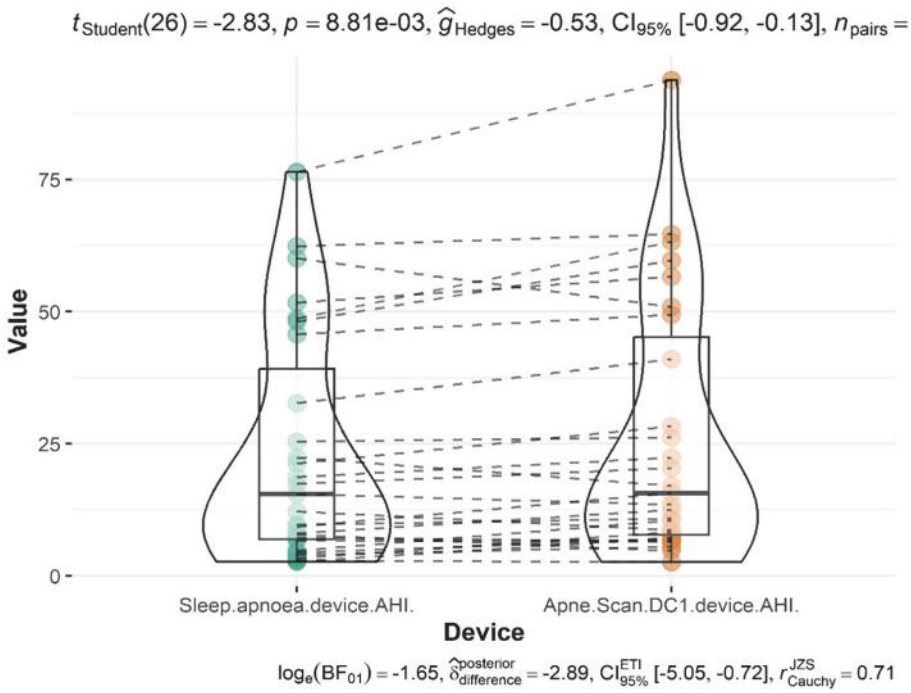
10.1136/thorax-2025-BTSabstracts.81

**Aim** Sleep apnoea (SA) is an under-diagnosed condition, which can lead to multimorbidity and societal costs. The current diagnostic devices are expensive, complicated for patients to use and complex to analyse. We conducted an exploratory study of a novel, low-cost, simple-to-use SA screening device to compare its capability to identify apnoea (A) and hypopnea with a current home sleep apnoea test (HSAT) (NOX T3).

**Method** 40 Participants were recruited from a SA clinic from a UK NHS hospital. They were trained in the use of the HSAT, which they were given to use concurrently with ApneScan (AS) overnight at home.

**Results** An expert scorer blinded to the HSAT output, manually scored the respiratory signal from AS for both A and indicative hypopneas (IH) (not validated against a drop in% spO2) to deliver a total Indicative AHI. This was then compared to the AHI delivered from the HSAT and results were manually validated by an expert. There was strong correlation between AHI score for the two devices (0.955) and excellent agreement for the proportion of severity of sleep apnoea categories (93%).

**Conclusions** The results of this exploratory study indicate that, with further refinement of the automated analysis of the respiratory signal, would help the diagnostic throughput in sleep clinics and reduce costs. Due to device design it could also assess patient tolerance to therapy.




**Abstract S75 Figure 1** Plot of AHI values for both devices to check the agreement between measurements (with test of significance result – Parametric method – paired T test)

## S76 THE CLINICAL IMPACT OF 3D PRINTED POSITIVE AIRWAY PRESSURE (PAP) THERAPY INTERFACES (3DPIPPIN): A RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2025-BTSabstracts.82

**Introduction and objectives** The recognised treatment for sleep disordered breathing is PAP therapy. Three-dimensional printing of customised interfaces could improve comfort and clinical outcomes.

The primary aim was to assess the impact of customised versus conventional interfaces on Apnoea Hypopnea Index (AHI) at 6 months. Secondary outcome measures were: interface leak (L/min), PAP therapy concordance (%), prevalence of pressure ulcers and Epworth Sleepiness Score (ESS).

**Methods** A Randomised Controlled Trial (RCT) with 160 adults naïve to PAP therapy and diagnosed with SDB (AHI ≥15 events/hr). Randomisation was stratified by age and ethnicity. Structured light facial scans (POP2, Revopoint, China) were post-processed to engineer a 3D printed mould (Fuse 30 +, Formlabs, USA). The mould was injected with silicone to produce a customised PAP oronasal interface cushion.

Demographics are reported using descriptive statistics. AHI was compared using quantile regression, adjusting for baseline age, ethnicity and AHI. Quantile regression was used to account for the skewed distribution of the AHI data, providing a more robust analysis than mean-based methods. Secondary outcomes were compared using logistic (pressure ulcer), quantile (interface leak, concordance) and linear regressions (ESS).

**Results** The intervention and control groups were similar at baseline (table 1.). 56 control and 42 intervention participants completed the trial (p=0.007). The intervention was associated with a higher number of events/hr (difference in medians = 1.5, 95% CI: 0.65 to 3.12, p=0.059) a higher interface leak

**Abstract S76 Table 1** Demographics and anthropometrics at baseline

| | Control n=78 | Intervention n=82 |
|---|---|---|
| **Age(years) Mean(SD)** | 54.2 (13.3) | 54.4 (11.7) |
| **Sex(M:F) n(%)** | | |
| Male | 58 (74) | 6 6(80) |
| Female | 20 (26) | 16 (20) |
| **Ethnicity n(%)** | | |
| Asian | 19 (15) | 15 (18) |
| Black | 9 (12) | 11 (14) |
| Caucasian | 54 (69) | 56 (68) |
| **Waterlow score Mean(SD)** | 4 (2) | 4 (2) |
| **Body Mass Index Kg/m$^2$ Mean(SD)** | 34.9 (6.7) | 35.2 (7.0) |
| **Smoking Status n(%)** | | |
| Current smoker | 8 (10) | 6 (7) |
| Ex-smoker | 33 (42) | 36 (44) |
| Never smoker | 37 (47) | 40 (49) |
| **Baseline sleep study Mean(SD)** | | |
| Apnoea Hypopnea Index (events/hr) | 38.1 (17.0) | 39.5 (20.1) |
| Overnight Desaturation Index (events/hr) | 31.3 (19.8) | 29.5 (18.7) |
| Mean $SpO_2$ | 91.8 (2.4) | 91.5 (2.7) |
| Total Sleep Time $SpO_2$ <90% (%) | 18.4 (18.3) | 20.5 (19.4) |
| **Baseline Epworth Sleepiness Score** | 8.5 [5.3 to 13.0] | 9.0 [5.0 to 14.0] |

**Median[IQR]**

(difference in medians 30.0L/min, 95% CI 7.36 to 40.14, p <0.0001), and lower compliance (%) (difference in compliance = 0.78, 95% CI 0.05 to 1.54, p =0.04) at six months. There was a non-significant 87% higher chance of developing a pressure ulcer compared to the control group (OR = 1.87, 95% CI: 0.39 to 10.1, p = 0.434). There was no difference in ESS between the control and intervention groups at six months (difference between control and intervention = 0.361, 95% CI -1.61 to 2.33, p = 0.72).

**Conclusions** Customised oronasal interfaces in their current format were not superior to conventional interfaces. Improvements in the design and manufacturing workflow are required before theoretical benefits of customised interfaces can be realised.

## S77 TRANSCUTANEOUS ELECTRICAL STIMULATION-INDUCED FATIGUE IN THE TIBIALIS ANTERIOR: A SURROGATE MODEL FOR UPPER AIRWAY MUSCLE FUNCTION

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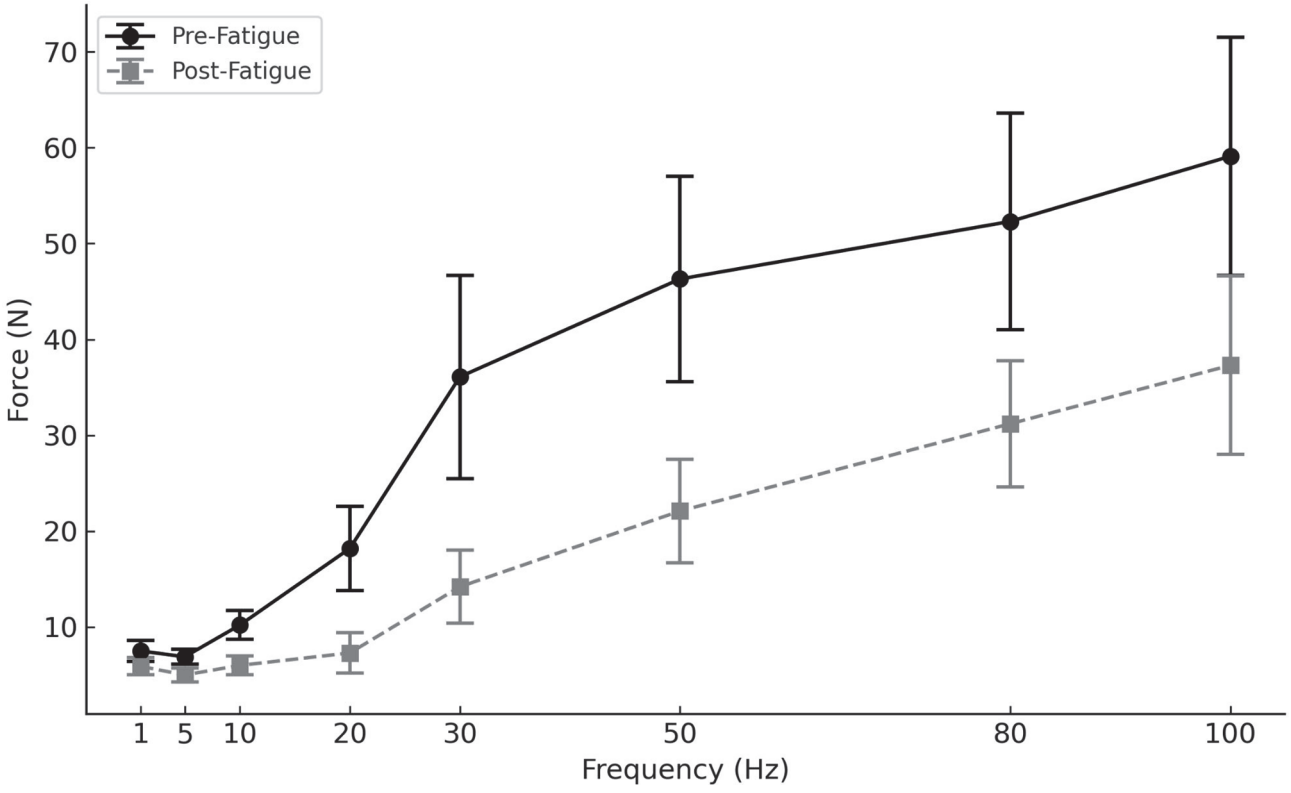
10.1136/thorax-2025-BTSabstracts.83

**Background** Transcutaneous electrical stimulation (TES) has been explored as a potential non-CPAP therapy for patients with obstructive sleep apnoea (OSA). However, limited data exist regarding its effects on upper airway muscle function and fatigue. This study used the tibialis anterior (TA) as a surrogate muscle to investigate the impact of TES on skeletal muscle fatigue.

**Methods** Ethical approval was obtained from King's College London Research Ethics Committee (Ref: HR/DP-24/25–46029). TA isometric maximum voluntary contraction (MVC) force was measured before and after fatigue using a forceplate attached to the foot. Fatigue was induced by 15 minutes of continuous TES from a constant-current stimulator at 30 Hz, 250 μs pulse width via 4×4 cm surface electrodes. Stimulation intensity (1–100 mA) was titrated to visible contraction. The TA force-frequency relationship was assessed pre- and post-fatigue via one-second trains of stimuli (1, 5, 10, 20, 30, 50, 80, 100 Hz). Protocol repeatability was evaluated using intraclass correlation coefficients (ICC) with a two-way random effects model.

**Results** Eleven healthy participants (7 males, 4 females; mean age 35 ± 10 years; BMI 22 ± 4 kg/m$^2$) were studied. MVC fell from 276 ± 166 N at baseline to 253 ± 160 N post-TES (p = 0.095). The force-frequency curve showed a characteristic sigmoidal shape (figure 1). At 100 Hz, mean (SD) evoked force decreased from 60 ± 46 to 40 ± 27 N (−34%), and at 30 Hz from 47 ± 36 to 22 ± 14 N (−54%). The mean (SD) tetanic force across all frequencies decreased from 38 ± 29 N pre-fatigue to 20 ± 13 N post-fatigue, corresponding to an absolute reduction of -17 ± 18 N and a relative reduction of -46 ± 31%. Stimulation threshold to elicit visible contraction was 7 ± 2 mA. Protocol repeatability was excellent (ICC = 0.86, 95% CI: 0.73–0.93; p < 0.001).

**Conclusion** Fifteen minutes of continuous electrical stimulation applied to the TA led to consistent, measurable reductions in evoked muscle force, supporting the feasibility of using this protocol to model muscle fatigue in a controlled setting with implications for clinical applicability.




**Abstract S77 Figure 1**

## S78 EXPLORING PHENOTYPE-SPECIFIC RESPONSE TO HOME NON-INVASIVE VENTILATION IN CHRONIC HYPERCAPNIC RESPIRATORY FAILURE

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10.1136/thorax-2025-BTSabstracts.84

**Introduction** Home non-invasive ventilation (NIV) is an established treatment for chronic hypercapnic respiratory failure, but physiological responses and adherence may differ between disease phenotypes. We compared three-month outcomes between chronic obstructive pulmonary disease (COPD) and obesity-related respiratory failure (ORRF) patients initiated on remotely monitored outpatient NIV.

**Methods** We analysed prospectively collected data from all patients initiated on remotely monitored outpatient NIV at a UK tertiary centre (February 2023-January 2025). Data included baseline and first follow-up data, including gas exchange, COPD Assessment Test (CAT), Epworth Sleepiness Scale (ESS) and device usage. The primary endpoint was change in (Δ) $PaCO_2$ at first follow-up. Secondary endpoints included $\Delta PaO_2$, $\Delta HCO_3^-$, ESS and CAT scores at first follow-up and responder rate (defined as $\Delta PaCO_2 \geq 0.5$ kPa). Acceptable adherence was pre-defined as ≥4 hours nightly use (30 day mean).

**Results** Fifty-one patients were analysed, 37 with COPD (72.5%) and 14 with ORRF (27.5%): mean age 63 ±13 years, 26 (51%) were female. Adherence data were available for 49 (96%) patients, with median follow-up of 3.7 months. Adherence was similar between groups, COPD patients 4.45h (IQR 1.51–7.80) versus ORRF patients 4.34h (IQR 2.30–5.47), p=0.56. Acceptable adherence was achieved by 20/37 (54%) of COPD patients and 8/14 (57%) of ORRF patients.

Mean $PaCO_2$ decreased in all groups with the largest reduction in adherent ORRF patients (–1.21 kPa [95% CI: -2.00, -0.42]) (table 1). Adherent COPD patients experienced an improved $PaO_2$ (+1.04 kPa [95% CI: 0.40, 1.49]) and reduced bicarbonate (–3.15 mmol $L^{-1}$ [95% CI: -3.92, -1.14]). Responder rate was 7/14 (50%) in ORRF and 15/37 (40.5%) of COPD patients. Median CAT scores improved in COPD patients (-8.5 [95% CI: -15.00, -1.00]).

**Conclusions** With similar usage of NIV, ORRF patients demonstrated a better response to normalise hypercapnic respiratory failure, while COPD patients had greater improvement in oxygenation and reduction in their bicarbonate levels. There was an effect of adherence on therapeutic efficacy. However, these findings underline the effectiveness of outpatient set-up of home NIV in improving respiratory failure and symptoms in patients with COPD and ORRF.

## S79 ADHERENCE TO A NON-INVASIVE VENTILATION (NIV) TRUST GUIDELINE TARGETING TIDAL VOLUMES AND ITS CORRELATION WITH CLINICAL OUTCOMES

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10.1136/thorax-2025-BTSabstracts.85

**Introduction** NIV is utilised to manage acute hypercapnic respiratory failure, improving patient outcomes with decreased mortality, length of stay (LOS) and re-admission. Early reversal of acidosis is associated with survival and a critical treatment goal.[1]

The British Thoracic Society (BTS) NIV guidelines provide general recommendations but do not specify target tidal volumes (TV). Trust NIV guidelines recommend TV's of 7–10 ml/kg of Ideal Body Weight (IBW) but little evidence supports this. We aimed to review adherence to trust guidelines and whether TV-IBW influenced clinical outcomes.

**Method** A retrospective, observational study was completed at a large UK NHS Hospital between October 2022-March 2024. Using electronic health records; patient demographics and NIV characteristics were collected. This included TV at 15-minute intervals (following initiation), inspiratory/expiratory positive airway pressures, treatment duration and time to reversal, defined as a pH level >7.35, categorised as: <1hour/1–3 hours/4–24 hours/>24 hours.

**Results** Data was available for N=95 patients (table 1). No significant association was seen between demographics and

**Abstract S78 Table 1 Changes in physiological parameters and symptom/quality of life scores from baseline to first follow-up**

| | COPD all (n=37) | COPD Adherence ≥ 4 h (n=20) | COPD Adherence < 4 h (n=15) | ORRF all (n=14) | ORRF Adherence ≥ 4 h (n=8) | ORRF Adherence < 4 h (n=6) |
|---|---|---|---|---|---|---|
| $\Delta PaCO_2$ (kPa) | –0.20 (± 1.41) *[6.92→6.72]* | –0.47 (± 1.30) *[6.89→6.43]* | –0.10 (± 1.00) *[6.88→6.77]* | **–0.78*** (± 0.91) *[6.71→5.93]* | **–1.21*** (± 0.95) *[6.89→5.68]* | –0.21 (± 0.43) *[6.48→6.27]* |
| $\Delta PaO_2$ (kPa) | +0.54 (± 2.65) *[8.26→8.80]* | **+1.04*** (± 0.94) *[8.05→9.09]* | –0.20 (± 3.98) *[8.68→8.48]* | +0.88 (± 2.65) *[8.72→9.60]* | +1.76 (± 4.67) *[8.77→10.53]* | +0.22 (± 0.36) *[8.64→8.86]* |
| $\Delta HCO_3^-$ (mmol $L^{-1}$) | **–1.59*** (± 8.56) *[31.13→29.54]* | **–3.15*** (± 2.30) *[31.03→27.88]* | +2.28 (± 8.09) *[30.84→33.12]* | –1.14 (±12.46) *[30.46→29.3]* | –1.57 (± -) *[30.20→28.63]* | –0.97 (± 0.10) *[30.85→29.88]* |
| ΔESS | –1.0 (-4 to 0) *[7.0→6.0]* | –3.0 (-5 to 0) *[9.0→6.0]* | –1.0 (-4 to 0) *[7.0→6.0]* | –3.0 (-7 to 0) *[8.5→5.5]* | –2.0 (-9 to 2) *[7.5→5.5]* | –2.5 (-5.5 to -0.5) *[8.5→6.0]* |
| ΔCAT | **–8.5* (-12 to 3)** *[22.5→14.0]* | **–9.0* (-12 to -4)** *[23.0→14.0]* | –4.0 (-15 to 3) *[18.0→14.0]* | — | — | — |
| $PaCO_2$ reduction ≥ 0.5 (kPa) N (%) | 15 (41%) | 9 (45%) | 5 (33%) | 7 (50%) | 6 (75%) | 1 (25%) |

Data are presented as mean change (SD) [*baseline mean to follow-up mean*] for physiological parameters ($\Delta PaCO_2$, $\Delta PaO_2$, $\Delta HCO_3^-$) and median change (IQR) [*baseline median to follow-up median*] for symptom/quality of life scores (ΔESS, ΔCAT). *p < 0.05.

**Abstract S79 Table 1** Associations between cohort characteristics and TV-IBW

| | Whole Cohort (N=95) | TV-IBW <7.0ml/kg$_{IBW}$ (N=8) | TV-IBW 7.0-10.0ml/kg$_{IBW}$ (N=32) | TV-IBW >10.0ml/kg$_{IBW}$ (N=55) | Spearman's Rho | p-Value |
|---|---|---|---|---|---|---|
| TV-IBW (ml/kg$_{IBW}$) | 10.7 (8.7-13.5) | 6.4 (5.7-6.8) | 8.7 (7.7-9.1) | 12.8 (11.5-15.5) | - | N/A |
| ***Demographics*** | | | | | | |
| Female Gender | 56 (59%) | 5 (63%) | 17 (53%) | 34 (62%) | -[c] | 0.368[c] |
| Pathology | | | | | -[c] | 0.216[c] |
| *COPD* | 75 (79%) | 5 (63%) | 25 (78%) | 45 (82%) | | |
| *OSA/OHS* | 16 (17%) | 1 (13%) | 6 (19%) | 9 (16%) | | |
| *Others* | 4 (4%) | 2 (25%) | 1 (3%) | 1 (2%) | | |
| Height (cm) | 160 (155-170) | 165 (159-172) | 160 (156-171) | 160 (155-168) | -0.123 | 0.236 |
| IBW (kg) | 55 (48-65) | 58 (51-67) | 56 (48-66) | 55 (48-63) | -0.125 | 0.228 |
| Weight (kg) | 70 (59-98) | 57 (49-68) | 66 (58-84) | 76 (61-105) | 0.250 | **0.014** |
| BMI (kg/m$^2$) | 27 (22-35) | 22 (20-24) | 26 (21-30) | 29 (25-39) | 0.304 | **0.003** |
| ***Ventilation*** | | | | | | |
| IPAP (cm$H_2O$) | 19 (16-23) | 15 (12-17) | 19 (17-21) | 20 (17-24) | 0.290 | **0.004** |
| EPAP (cm$H_2O$) | 6 (5-8) | 5 (5-6) | 6 (5-8) | 7 (5-8) | 0.221 | **0.031** |
| Median TV (ml) | 598 (458-763) | 373 (287-421) | 481 (422-563) | 721 (648-933) | 0.843 | **<0.001** |
| ***Outcomes*** | | | | | | |
| Time to Reversal | | | | | -0.225 | **0.028** |
| *<1 Hour* | 39 (41%) | 3 (38%) | 8 (25%) | 28 (51%) | | |
| *1-3 Hours* | 24 (25%) | 2 (25%) | 11 (34%) | 11 (20%) | | |
| *4-24 hours* | 19 (20%) | 3 (38%) | 4 (13%) | 12 (22%) | | |
| *>24 Hours*[a] | 13 (14%) | 0 (0%) | 9 (28%) | 4 (7%) | | |
| Died on NIV | 1 (1%) | 1 (13%) | 0 (0%) | 0 (0%) | -[c] | **0.021**[c] |
| NIV Duration (Days; N=94)[b] | 6 (4-9) | 10 (6-15) | 6 (4-9) | 6 (4-8) | -0.127 | 0.221 |
| Died in Hospital | 9 (9%) | 2 (25%) | 3 (9%) | 4 (7%) | -[c] | 0.178[c] |
| Hospital Stay (Days; N=86)[b] | 14 (9-21) | 14 (11-24) | 15 (9-24) | 14 (9-17) | -0.055 | 0.615 |

*Data are reported as N (column %), or as median (interquartile range), as appropriate. Associations with TV-IBW (as a continuous variable) were assessed using Spearman's rank correlation coefficients (rho), unless stated otherwise, and bold p-values are significant at p<0.05. [a] The >24 hour category includes N=3 patients where reversal was not achieved. [b] Excludes patients who died on NIV/in hospital. [c] For nominal factors, p-values are from Mann-Whitney U / Kruskal-Wallis tests, comparing the average TV-IBW (as a continuous variable) across the levels of the factor (e.g. between male vs. female).*

TV-IBW (P>0.2). Most patients had COPD (79%) or OSA/OHS (17%) with median NIV duration being 6 days. Median TV was 598 ml (IQR:458–763), correlating to 10.7 ml/kg-IBW (IQR:8.7–13.5), Only N=32 (34%) patients TV-IBW were trust guideline compliant, 58% (N=55) had values above this range.

Reversal was achieved in <1 hour in N=39 (41%). Increasing TV-IBW was associated with significantly shorter reversal times (rho:-0.225,p=0.028) with 51% of patients >10.0 ml/kg-IBW reversing in <1hour. Additionally, a significantly lower on-NIV mortality rate, with the single death in the patient with the lowest TV-IBW value in the cohort.

**Conclusion** This is the first study to review TV-IBW targets during NIV initiation and possible correlations to clinical outcomes. Only 34% of patients were compliant with trust guidelines, with the majority being above the TV-IBW target. Increasing TV-IBW was associated with progressively improved clinical outcomes, including shorter acidosis reversal times and lower on-NIV mortality. A review of local NIV guidelines would be warranted. Further research could compare current BTS guidelines vs efficacy of targeting >10 ml/kg-IBW.

## REFERENCE

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# Pulmonary hypertension and CTEPH – classification and treatment

## S80 EXPLORING MACHINE LEARNING TECHNIQUES FOR CLASSIFYING PULMONARY HYPERTENSION USING UK BIOBANK ELECTRONIC HEALTH RECORDS

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**Background** Pulmonary hypertension is a rare, progressive and life-threatening condition, which is subject to delayed diagnosis due to its non-specific symptoms. Machine learning (ML) when applied to electronic health records (EHR) may aid earlier detection of PH. This study evaluated whether ML models could accurately classify PH using OCD-10 codes and identify comorbidities with strong predictive contributions using the UK Biobank dataset.

**Methods** The UK Biobank dataset was pre-processed to extract participants with 2 or more of 20 predefined conditions associated with PH, creating an unmatched cohort (n=152,853). PH cases were identified by the ICD-10 codes (I27.0, I27.2, I27.9) and matched 1:1 with controls based on age and sex (n=5,142). Three supervised ML models: K-Nearest

Neighbour (KNN), Support Vector Machine (SVM) and Random Forest (RF), were trained on an 80% of the matched cohort. Shapley Additive exPlanations (SHAP) was applied for both feature selection and final model interpretation. A temporal analysis of comorbidity diagnosis timing was conducted for PH cases, prior to diagnosis.

**Results** The SVM model using 12 features achieved the best performance with (AUC 0.88, sensitivity 0.82 and accuracy 0.82). Key predictive comorbidities included left heart failure, restrictive airway disease, atrial fibrillation, valvular disease, pulmonary embolism and chronic obstructive pulmonary disease. Temporal analysis showed that many predictive comorbidities emerge up to 3 years prior to PH diagnosis, with increasing prevalence closer to diagnosis date.

**Conclusions** ML models can accurately classify PH using fewer, clinically relevant features. SHAP improves model transparency and identifies high-impact predictors. The findings support the feasibility of developing early screening tools for PH based on comorbidity profiles. Future work should include more diverse EHR features and externally validate models with alternate datasets.

## S81 EPIDEMIOLOGY, PROGNOSIS, AND HEALTHCARE RESOURCE USE OF PATIENTS WITH PULMONARY HYPERTENSION ASSOCIATED WITH LUNG DISEASE AND/OR HYPOXIA IN THE UK

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10.1136/thorax-2025-BTSabstracts.87

**Background** Pulmonary Hypertension associated with Lung Disease and/or Hypoxia (PH-LD), including PH with interstitial lung disease (PH-ILD), are severe conditions with a high mortality. However, data on epidemiology and healthcare management in the real-world setting are limited.

**Objective** To estimate the epidemiology and the healthcare resource use (HCRU) of patients with PH-LD/ILD.

**Methods** We conducted a retrospective observational study using the UK Clinical Practice Research Datalink (CPRD) and Hospital Episode Statistics (HES), including >18 million patients. An algorithm, developed with six PH medical experts, identified PH-LD and PH-ILD patients from January 2017 to December 2019 based on a combination of PH and LD ICD-10 codes. The index date was the first PH diagnosis, with a 24-month minimum lookback period and up to 60 months follow-up until end of study, loss to follow-up or death.

**Results** A total of 6,690 patients with PH-LD (mean age 71, 54% female) and 1,561 with PH-ILD (mean age 71, 62% female) were included. The estimated period prevalence (2017–2019) and annual incidence of PH-LD was 2.65 (95% CI 2.59–2.70) and 0.67 (0.651–0.68) per 10,000 people, and PH-ILD 0.66 (95%CI 0.64–0.69) and 0.16 (0.15–0.16), respectively. During the first-year of follow-up, incident PH-LD and PH-ILD patients had a mean (95%CI) 3.46 (3.40–3.51) and 2.93 (2.82–3.03) all-cause inpatient hospitalizations per person-year, including 1.87 (1.83–1.91) and 1.78 (1.70–1.87) due to PH, and 2.94 (2.89–2.99) and 2.51 (2.41–2.60) due to LD. Mean length of stay (SD) was 10 (15) and 10 (13) days, respectively. Hospitalization rates decreased over time. One, two, and three-year survival rates (95%CI) for PH-LD and PH-ILD patients were 58% (57%-59%), 46% (45%-48%), and 40% (39%-41%), and 53% (51%-56%), 43% (41%-46%), and 38% (35%-40%), respectively.

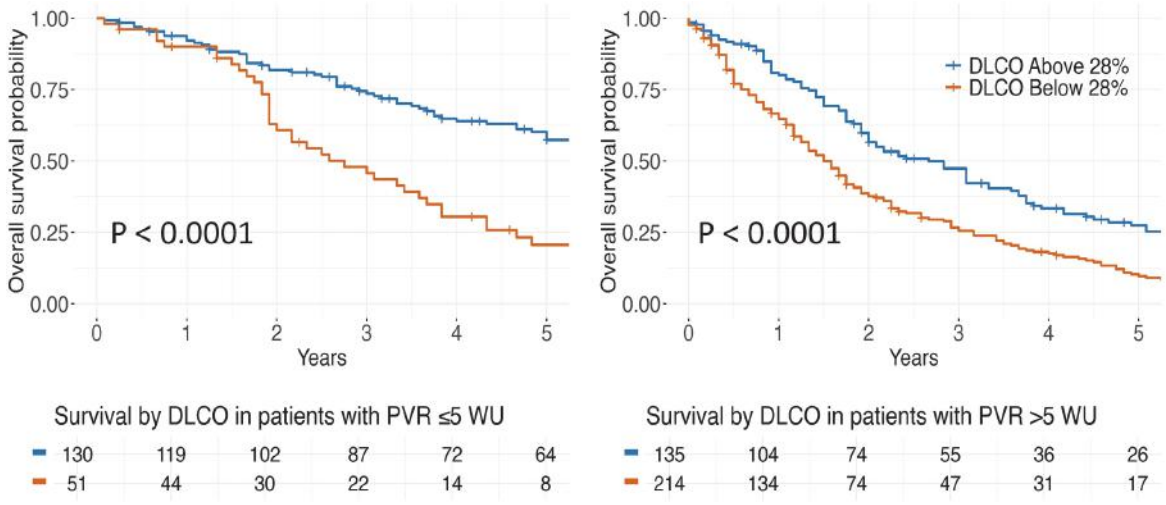
**Conclusion** With current therapies patients with PH-LD and PH-ILD have a significant unmet need with high levels of HCRU and a poor prognosis.

## S82 PREDICTORS OF OUTCOME IN PULMONARY HYPERTENSION ASSOCIATED WITH LUNG DISEASE

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**Background** Current ERS/ESC guidelines use a pulmonary vascular resistance (PVR) of 5WU to distinguish severe from non-severe PH associated with lung disease (Group 3).




**Abstract S82 Figure 1**

However, there are limited data on how best to risk stratify patients.

**Aims and Objectives** To assess the prognostic value of non-invasive and invasive testing in Group 3 PH.

**Methods** Consecutive patients from the ASPIRE registry with group 3 PH diagnosed between 2007 and 2023 were included. Patients underwent a comprehensive assessment including pulmonary function and exercise testing, CT imaging, cardiac MRI and right heart catheterisation. Prognostic value was assessed using Kaplan-Meier (KM) and multivariable Cox regression analyses.

**Results** 774 patients, 52% male, mean age 67(12) yrs were included. 48% had COPD/emphysema, 27% ILD, 14% CPFE, and 11% non-parenchymal restriction. There were 588 deaths with a median survival of 24 months (10–55). Predictive variables from univariate analysis were incorporated into a multivariable model and refined. Age, male sex, DLCO%predicted (HR: 0.98, 95% CI: 0.97–0.99, p<0.001), walk distance (HR 0.98 95% CI 0.97–0.99 p=0.006), RVEF (HR 0.98 95% CI 0.97–1 p=0.04) and PVR (HR 1.06 95% CI 1.02–1.11 p=0.002) were independently associated with survival. KM analysis stratifying patients by PVR > or ≤5WU and DLCO > or ≤ 28% (median) demonstrated that low DLCO further differentiated survival within both high and low PVR groups. DLCO also predicted survival in all Group 3 subgroups.

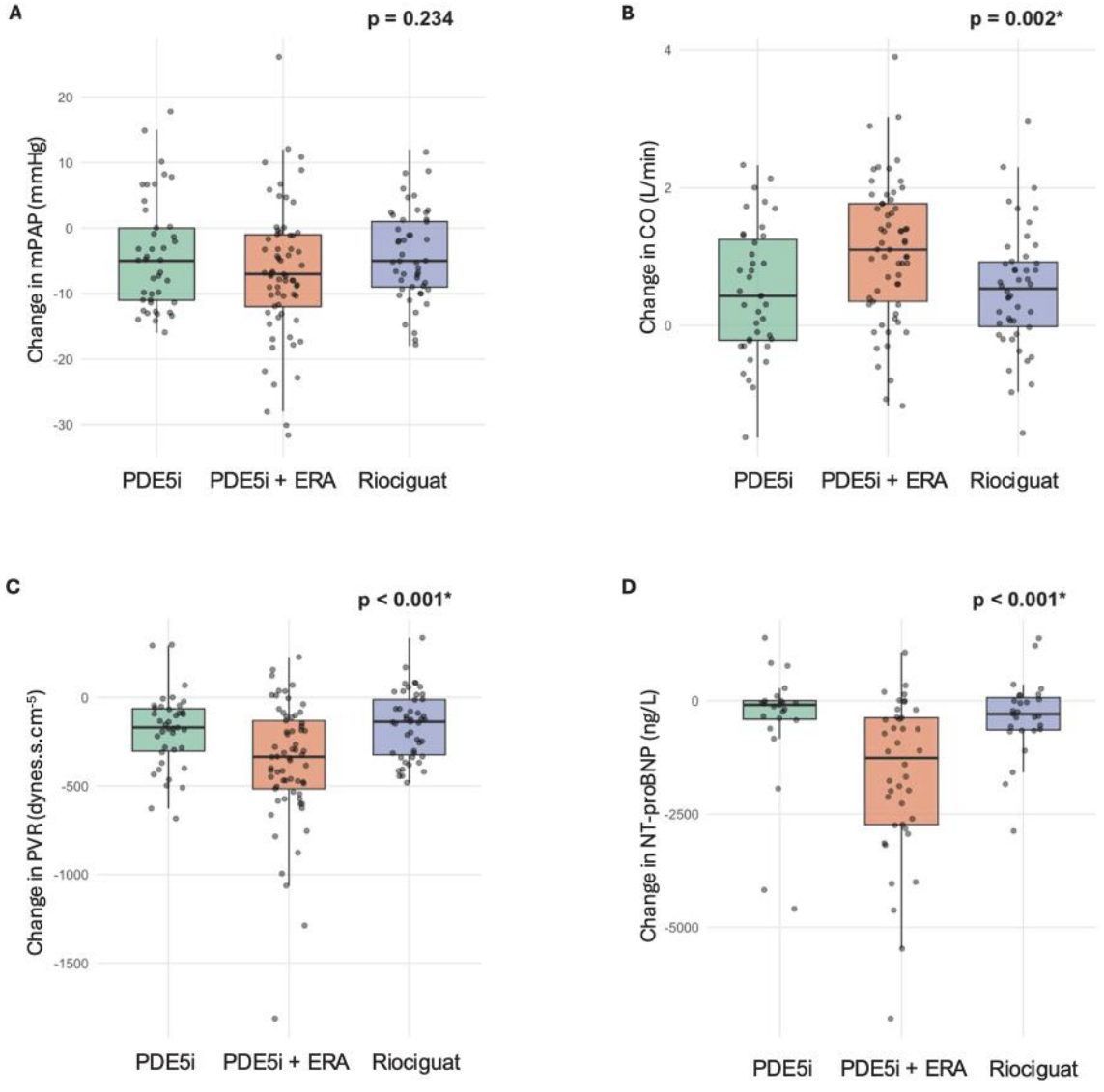
**Conclusion** DLCO is a strong, independent predictor of mortality in group 3 PH. When used in combination with PVR it improves mortality prediction in all subgroups.

## S83 COMPARING BRIDGING THERAPIES PRIOR TO BALLOON PULMONARY ANGIOPLASTY IN CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION

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**Introduction** Patients with chronic thromboembolic pulmonary hypertension (CTEPH) may undergo balloon pulmonary angioplasty (BPA) if pulmonary endarterectomy is unsuitable. Medical therapies including phosphodiesterase-5 inhibitors (PDE5i),




**Abstract S83 Figure 1** Absolute change in haemodynamic parameters and NT-proBNP from diagnosis (baseline) to immediately before BPA for (A) mPAP; (B) CO; (C) PVR; (D) NT-proBNP

endothelin receptor antagonists (ERA) and riociguat are commonly used as bridging therapies to BPA, but data comparing these therapies are limited. We compared the impact of bridging therapies on serially measured haemodynamic and physiological parameters.

**Methods** We conducted a secondary analysis of the prospective UK CURATE registry. Consecutive patients awaiting BPA at Royal Papworth Hospital between 1 October 2015 and 1 October 2024 were recruited. Baseline haemodynamic and physiological parameters at CTEPH diagnosis (first right heart catheter) and immediately prior to starting BPA therapy (second right heart catheter) were collected. Differences in parameters were assessed using Kruskal-Wallis to compare the impact of medical therapies used in the intervening period. Propensity score matching was performed to account for baseline differences.

**Results** 158 patients (median age 66 and 54% male) with CTEPH taking bridging therapy before their 1st BPA were included. 42 received PDE5i monotherapy, 67 received PDE5i and ERA dual therapy, and 49 received riociguat monotherapy. Patients with more severe baseline haemodynamic data were preferentially treated with dual therapy. All bridging therapy approaches were associated with improved haemodynamic parameters including mean pulmonary arterial pressure (mPAP), cardiac output (CO) and pulmonary vascular resistance (PVR), and NT-proBNP. Dual therapy was associated with a greater increase in CO ($p=0.002$), reduction in PVR ($p<0.001$) and reduction in NT-proBNP ($p<0.001$) versus monotherapies (figure 1). Comparison of percentage change, correcting for baseline differences, yielded similar results, with dual therapy associated with greater increase in CO ($p<0.001$), reduction in PVR ($p<0.001$) and reduction in NT-proBNP ($p=0.004$) versus monotherapies. Propensity score matching further supported these findings.

**Conclusion** Patients with CTEPH awaiting BPA had greater improvements in haemodynamic parameters and NT-proBNP with PDE5i and ERA dual bridging therapy versus monotherapies, with no differences between PDE5i and riociguat monotherapies. These differences persisted following correction for baseline differences. As improvements in haemodynamic parameters are associated with improved BPA safety profile, bridging with dual therapy may enhance BPA safety, although more work is needed to validate this hypothesis.

## S84 BALLOON PULMONARY ANGIOPLASTY VERSUS RIOCIGUAT IN INOPERABLE CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMISED CONTROLLED TRIALS

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10.1136/thorax-2025-BTSabstracts.90

**Background** Chronic thromboembolic pulmonary hypertension (CTEPH) is a life-threatening condition caused by persistent obstruction of the pulmonary arteries. While pulmonary endarterectomy is curative in eligible patients, many are inoperable due to distal thrombi or comorbidities. Balloon pulmonary angioplasty (BPA) and riociguat, a soluble guanylate cyclase stimulator, are treatment options in this group, yet comparative efficacy remains uncertain. We aimed to systematically review and meta-analyse randomised controlled trials (RCTs) comparing BPA and riociguat in inoperable CTEPH.

**Methods** A comprehensive literature search was conducted across PubMed, Embase, Cochrane CENTRAL, and ClinicalTrials.gov from January 2018 to April 2025, following PRISMA guidelines. Eligible studies were RCTs comparing BPA and riociguat in adult patients with confirmed inoperable CTEPH. Outcomes included mean pulmonary arterial pressure (mPAP), cardiac output (CO), pulmonary vascular resistance (PVR), mean right atrial pressure (mRAP), and NT-proBNP levels. Data extraction and quality assessment were conducted independently by two reviewers. A random-effects model was used for pooled estimates, with heterogeneity assessed by $I^2$ statistics and sensitivity analyses.

**Results** Three RCTs involving 262 patients (134 BPA, 128 riociguat) were analysed. BPA led to a significantly greater reduction in mPAP (MD: 12.23 mmHg; 95% CI: 9.15–15.32; $p < 0.00001$) and pulmonary vascular resistance (MD: 208.58 dyn·s/cm$^5$; 95% CI: 117.32–299.85; $p < 0.00001$), with both outcomes showing initial heterogeneity that resolved upon sensitivity analysis. BPA also reduced mRAP more than riociguat (MD: 2.18 mmHg; 95% CI: 1.23–3.13; $p < 0.00001$). NT-proBNP levels, a biomarker of cardiac stress, decreased more substantially with BPA (MD: 989.61 pg/mL; 95% CI: 522.55–1456.66; $p < 0.0001$). In contrast, riociguat was superior in improving CO (MD: 0.47 L/min; 95% CI: 0.37–0.58; $p < 0.00001$), suggesting a distinct mechanism of benefit. All outcomes showed consistency across studies except for one trial identified as a source of heterogeneity that resolved upon sensitivity analysis. No significant difference was found in overall adverse event rates, though BPA carried procedural risks including pulmonary injury.

**Conclusion** BPA offers superior hemodynamic and biomarker improvements over riociguat in inoperable CTEPH, whereas riociguat enhances cardiac output. A patient-specific approach considering feasibility and treatment goals may optimise outcomes.

## S85 PULMONARY HAEMODYNAMICS AFTER MECHANICAL THERAPY WITH PULMONARY ENDARTERECTOMY (PEA) AND BALLOON PULMONARY ANGIOPLASTY (BPA) PREDICT SURVIVAL IN PATIENTS WITH CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION (CTEPH)

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10.1136/thorax-2025-BTSabstracts.91

**Background** CTEPH can be treated by mechanical therapy with PEA and/or BPA. The haemodynamic treatment goal is ill defined. We explored post-procedure mean pulmonary arterial pressure (mPAP), pulmonary vascular resistance (PVR), and 5-year survival and QoL differences using 6MWD and CAMPHOR symptoms score for mPAP.

**Method** 1,752 PEA and 172 BPA patients from the UK national database were stratified into 5 groups by 3-month

follow-up mPAP and PVR to generate Kaplan-Meier curves with Kruskal-Wallis and pairwise log-rank tests. Cox proportional hazards models assessed survival differences, adjusted for age, sex and comorbidities. We similarly assessed 6MWD and CAMPHOR scores to examine QoL.

**Results** After PEA, 5-year survival was significantly higher in patients with mPAP ≤25 mmHg compared to higher groups (all pairwise comparisons $p < 0.002$); there was no additional survival advantage between ≤20 and 21–25 mmHg. A continuous PVR model showed a 0.16% increase in mortality risk per 1 dyn·s·cm$^{-5}$ increase ($p = 2.1e-10$; concordance = 0.699). Adjusting for hazards with a Cox model, results remained significant. QoL measurements showed a similar trend ($p = 2.2e-16$ for the 6MWD).

In the BPA cohort, 5-year survival was significantly higher when mPAP ≤30 mmHg was achieved. A continuous PVR model variable showed a 0.34% increase in mortality risk per unit increase ($p = 0.002$; concordance = 0.70). For QoL we saw no significant difference between mPAP groups.

**Conclusions** Post-procedure haemodynamics predict 5-year survival and QoL in PEA patients, with mPAP ≤30 mmHg and ≤25 mmHg conferring the best prognosis for BPA and PEA-treated patients, respectively. These data should encourage therapy escalation in those not achieving mPAP thresholds.

# Mechanisms of IPF pathogenesis

## S86 INTEGRATION OF IPF-ASSOCIATED SNPS WITH DIFFERENTIAL DNA METHYLATION AND GENE EXPRESSION SHOWS CELL-TYPE SPECIFIC MECHANISMS IN AIRWAY AND PARENCHYMAL FIBROBLASTS

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10.1136/thorax-2025-BTSabstracts.92

**Rationale** Idiopathic Pulmonary Fibrosis (IPF) is linked to over 35 genetic risk loci, but their contribution to disease mechanisms remains unclear. DNA methylation (DNAm) is a cell-type specific molecular mechanism that may mediate genetic risk by altering cell type specific gene expression (GE). We previously demonstrated widespread DNAm-associated GE alterations in IPF versus non-IPF fibroblasts derived from airways (AF) and parenchyma (PF). Here, we aimed to test the hypothesis that cell type specific GE is associated with IPF-associated SNPs, with potential modulation by DNAm.

**Methods** DNA and RNA were isolated from AF (8 non-IPF, 8 IPF) and PF (14 non-IPF, 8 IPF) at passage 4 (AllPrep DNA/RNA Mini Kit, Qiagen). DNA was profiled using the Illumina HumanMethylation EPIC array. RNA expression was assessed using Affymetrix Human Gene 2.1ST Array. We analysed the dataset with a new focus on integrating genetic risk. Firstly, IPF associated genes linked to DNAm were overlapped with SNP associated genes. A direct link between DNAm and GE for overlapping genes was assessed via DNMT (5-aza-2′-deoxycytidine, $10^{-10}$-$10^{-6}$ M) and TET (Ten-Eleven Translocation-in-C35, $5\times10^{-7}$, $1\times10^{-6}$, $5\times10^{-6}$ and $1\times10^{-5}$ M) inhibition in vitro, in PF from 3 IPF and 3 non-IPF donors. GE was assessed by qPCR. Secondly, we screened ±500 kb regions around each of the 35 IPF-associated SNPs for associated transcriptome changes, by linear modelling.

**Results** Four genes; DSP, STMN3, FAM13A, and TERC, were identified as SNP-associated genes that overlapped with DNAm-associated differentially expressed genes. TET inhibition did not show any impact on GE. DNMT inhibition showed significant reduction in expression of FAM13A in non-IPF cells. Our analysis of ±500 kb regions around IPF-associated SNPs revealed 64 genes differentially expressed between AF and PF in IPF, highlighting cell-type specific regulatory effects of genetic risk loci.

**Conclusion** Our integrative approach highlights an overlap between genetic risk loci and DNAm-associated GE changes in IPF. We observe fibroblast subtype-specific differences in GE near IPF-associated SNPs, though whether these effects are mediated by DNAm is to be further studied. These findings support the potential importance of epigenetic mechanisms in IPF pathogenesis and highlight the need for cell-type specific epigenetic analyses.

## S87 TARGETING AKAP13 RHOGEF ACTIVITY AMELIORATES PRO-FIBROTIC PHENOTYPES DRIVEN BY THE IPF ASSOCIATED AKAP13 RISK VARIANT

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10.1136/thorax-2025-BTSabstracts.93

**Background and Aims** IPF is a progressive, incurable scarring disease of the lung. A common genetic variant near AKAP13 has been associated with IPF susceptibility and elevated gene and protein expression. How this common variant contributes to the pathogenesis of IPF is not known. AKAP13 is a multi-functional scaffold protein, with high expression in lung epithelial cells and integrates intracellular signalling through its interactions with RhoA and protein kinase A (PKA). A13 is a selective small-molecule inhibitor of AKAP13's RhoGEF domain. This study investigates how an AKAP13 variant alters epithelial signalling and evaluates the therapeutic potential of targeting AKAP13 using A13.

**Methods** CRISPR-Cas9 editing was used to introduce the AKAP13 variant into immortalized human bronchial epithelial cells (iHBECs). Overexpression constructs modelled a truncated AKAP13 isoform lacking the PKA-binding domain. Real-time adhesion and proliferation were measured (xCELLigence). Cell transcriptional profiling is performed using ncounter. Intracellular cAMP was measured by FRET. RhoA activity was measured by G-LISA, and SMAD2 phosphorylation was measured by immunoblotting, both in the presence or absence of LPA stimulation. IPF patient-derived precision-cut lung slices (PCLS) were treated with A13 and analysed for profibrotic gene expression by real-time PCR analysis and SMAD2 nuclear translocation using immunofluorescence and cellpose single cell segmentation.

**Results** iHBECs carrying the AKAP13 variant showed increased expression of truncated isoform of AKAP13. These cells exhibited a ~50% increase in cell adhesion and ~30% reduction in proliferation compared to cells not expressing the AKAP13 variant. These cells showed a profibrotic gene signature had increased RhoA activation and SMAD2 phosphorylation following LPA stimulation. Furthermore, cells harbouring the AKAP13 variant displayed a reduced capacity to generate intracellular cAMP. A13 treatment reversed the pro-adhesive phenotype and significantly reduced RhoA activation. In IPF-derived PCLS (n=4), A13 suppressed *SERPINE1*, *CCN2*, and *MMP7* expression and reduced SMAD2 nuclear translocation.
**Conclusions** Presence of an AKAP13 variant disrupts epithelial homeostasis and promotes pro-fibrotic signalling. Inhibition of AKAP13's RhoGEF domain with A13 restores epithelial function and attenuates fibrotic activation, supporting AKAP13 as a therapeutic target in IPF.

## S88 EXPLORING THE EFFECT OF PKN2 KNOCKDOWN ON IDIOPATHIC PULMONARY FIBROSIS

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**Background** Idiopathic pulmonary fibrosis (IPF) is a chronic interstitial lung disease. A multi-centre genome-wide meta-analysis involving participants from the US, UK, and Spain identified Protein Kinase N2 (PKN2) as significantly associated with the decline in forced vital capacity in IPF patients. Although PKN2 has been linked to the proliferation of mouse embryonic fibroblasts, its role in IPF remains largely unexplored. We hypothesise that PKN2 contributes to the pro-fibrotic activity of fibroblasts, thereby promoting fibrosis. We aim to investigate how PKN2 knockdown influences $TGF\beta_1$-mediated profibrotic processes in human lung myofibroblasts (HLMFs), to better understand its impact on fibroblast function and lung function decline.
**Methods** PKN2 expression in lung tissue and in total lung cells was examined using qRT-PCR and Human Gene 2.1 ST genome-wide microarray data. The effect of PKN2 knockdown (siRNA) on HLMFs was examined by scratch wound assays, RNA-sequencing (n=5) and liquid chromatography-mass spectrometry (LC-MS) proteomics (n=3).
**Results** PKN2 mRNA is expressed in bulk lung tissue and specific cell types such as epithelial cells, airway smooth muscle cells and HLMF cells, with affymetrix gene expression data showing significant downregulation of PKN2 expression in IPF fibroblasts versus non-IPF fibroblasts in airway (p=0.0044) and parenchymal lung fibroblasts (p=0.0011).

Successful downregulation of PKN2 was achieved using siRNA (n=7, p=0.0402). RNA-sequencing shows that PKN2 knockdown in HLMFs promotes a pro-fibrotic genetic profile, with upregulation of collagens and extracellular matrix (ECM) components at gene level. Knockdown was associated with dysregulation of ECM, adhesion and migration gene pathways.

LC-MS analysis confirms successful downregulation of PKN2 at protein level. PKN2 knockdown leads to dysregulation of fibrosis associated proteins and pathways including integrin-signalling and regulation of WNT signalling pathways.

Functional assays showed that PKN2 knockdown does not impact HLMF proliferation (n=3, p=0.7439). PKN2 knockdown accelerates HLMF wound healing 24-hours post-injury (n=4, p=0.0061).
**Conclusion** Our findings confirm PKN2 expression in lung and IPF-associated cells, with lower expression in IPF and upon $TGF\beta_1$ stimulation. Knockdown of PKN2 enhances profibrotic processes, including wound healing and disrupts the expression of fibrosis associated genes, proteins and pathways. Collectively suggesting that PKN2 plays a role in protecting against fibrosis within the lungs.

## S89 THE ROLE OF TRPM7 IN HUMAN LUNG MYOFIBROBLAST PROFIBROTIC FUNCTION

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10.1136/thorax-2025-BTSabstracts.95

**Introduction** Idiopathic pulmonary fibrosis (IPF) is a progressive disease with no effective treatment. The melastatin-type transient receptor potential 7(TRPM7) bifunctional protein acts as an ion channel and a kinase. It is involved in $Ca^{2+}$/$Mg^{2+}$ homeostasis, cell growth and proliferation and has been linked to promoting profibrotic activity in cardiac fibroblasts, but its role in IPF and human lung myofibroblasts (HLMFs) pro-fibrotic function remains unclear. Therefore, we aim to investigate TRPM7 expression and function in HLMFs.
**Methods** TRPM7 mRNA expression (± TGFβ1) was assessed via qRT-PCR and Affymetrix Human Gene 2.1 ST genome-wide microarray in several lung cell types. TRPM7 currents were analysed using patch-clamp electrophysiology and manipulated with $Mg^{2+}$, Naltriben and TRPM7 inhibitor, waixenicin A (500nM). TRPM7 knockdown was achieved using siRNA, and fibrotic markers (αSMA, Collagen 1, and Collagen 6) were quantified by RT-PCR. MTS assay, flow cytometry and wound healing were utilised to assess the effects of waixenicin A on HLMF viability, proliferation and migration. DNA methylation was profiled with Illumina Infinium MethylationEPIC BeadChip.
**Results** TRPM7 is highly expressed in airway fibroblasts compared to airway epithelial cells (p<0.0001). However, in disease TRPM7 is significantly downregulated, IPF HLMF (p<0.05), IPF airway fibroblasts (p<0.0005) and IPF parenchymal fibroblasts(p<0.05) compared to the healthy controls. Similarly, treatment with TGFβ1 significantly reduced TRPM7 mRNA expression in HLMFs (p<0.05, n=11). Functional TRPM7 currents are present in HLMFs and can be blocked with the addition of magnesium (6 mM) (n=6 IPF and n=13

NFC) and waixenicin A(500nM) (n=6). siRNA knockdown of TRPM7 inhibited TRPM7 function and decreased α-SMA, Collagen 1 and 6 mRNA (n=10). MTS showed waixenicin A has no cytotoxic effects on HLMFs (n=5). TRPM7 inhibition has no significant effect on proliferation of HLMFs but significantly impaired wound healing ($p<0.01$, n=7). In addition, changes in TRPM7 methylation ($p<0.01$) were observed in CpGs located in regulatory regions.

**Conclusion** We confirmed HLMFs express functional TRPM7 channels. Despite TRPM7 mRNA downregulation in IPF, its inhibition reduces fibrotic markers and impairs wound healing, indicating a role in fibroblast activation. Epigenetic modifications may contribute to TRPM7 suppression in IPF. Further research is needed to understand how TRPM7 regulation influences fibrosis.

## S90 THE IMPACT OF A HYPOXIC ENVIRONMENT ON ALVEOLAR MACROPHAGE IMMUNOMETABOLISM: IMPLICATIONS FOR LUNG FIBROSIS

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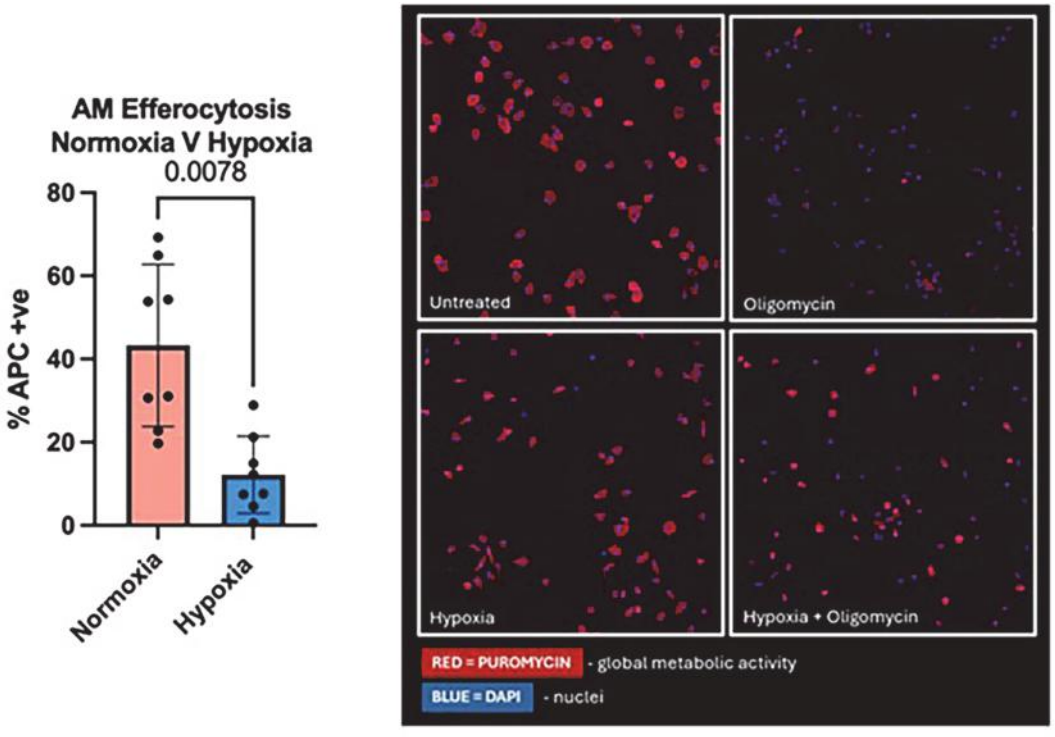
**Background** Alveolar macrophages (AMs) are known to influence the pathogenesis of idiopathic pulmonary fibrosis (IPF) and are said to express markers of hypoxic signalling in the fibrotic lung (PMID: 33805152). Hypoxia is suggested to directly affect fibroblasts and influence the fibrogenic cascade, however its impact on AM phenotype and function is unknown.

**Hypothesis** Hypoxic conditions alter the immunometabolic phenotype of AMs, impacting their role in lung homeostasis.

**Method** AMs were exposed to 1% oxygen for 48 hours and compared to normal culture conditions. AM function was measured by their ability to engulf apoptotic cells or 'efferocytosis'. Specific antibody binding to AM cell surface markers was measured by flow cytometry. Single Cell ENergetic metabolism by proflling Translation inHibition (SCENITH) is a method used to examine global metabolic activity in a cell. This method harnesses puromycin to measure all protein synthesis in a cell as a surrogate measure of ATP production (PMID: 35497494). AMs were treated with oligomycin, an inhibitor of ATP synthase and the electron transport chain, or 2-deoxyglucose, a non-metabolisable glucose analogue, and changes in metabolic activity were measured.

**Results** AMs exhibit a reduced capacity for efferocytosis in hypoxia ($p=0.0156$, n=7), alongside a reduction in scavenger receptor MerTK ($p=0.0312$, n=6). CD36 scavenger receptor surface expression is increased after exposure to hypoxia (0.0312, n=6). The global metabolic activity of AMs is reduced after exposure to hypoxia ($p=0.0147$, n=7). 2-deoxyglucose has no significant impact on metabolic activity of AMs in either condition. Oligomycin impairs metabolic activity in both normal cell culture conditions ($p<0.0001$, n=6) and in hypoxia ($p=0.0281$, n=6), indicating some reliance on oxidative phosphorylation regardless of oxygen supply.

**Conclusion** AMs exhibit changes in function, phenotype and metabolism in hypoxia. The loss of ability to remove dead or damaged cells by efferocytosis would lead to prolonged disruption of the alveolar epithelium in the lung and may potentiate excessive wound healing and fibrosis. Decreased efferocytosis in hypoxia may be attributed to dysregulated efferocytosis receptor expression or decreased metabolic activity. Further research is needed to dissect the mechanism behind this dysfunction and to model how it could influence the pathogenesis of IPF.




**Abstract S90 Figure 1**

## S91 DEFINING THE ANTIFIBROTIC MECHANISMS OF TREPROSTINIL IN PULMONARY FIBROSIS

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**Introduction** Idiopathic Pulmonary Fibrosis (IPF) is a progressive fibrosing interstitial lung disease with limited treatment options and a grave prognosis. Treprostinil, a synthetic prostacyclin analogue used in pulmonary hypertension, is under investigation for its antifibrotic potential. The antifibrotic mechanisms of Treprostinil are yet to be fully defined. Treprostinil activates the IP receptor to generate cAMP but may also target intracellular PPARβδ receptors. PPARβδ activation can inhibit the RhoA pathway which is amplified in IPF. These studies aim to explore the antifibrotic mechanisms of Treprostinil.

**Methods** Expression of IP and PPARβδ receptors in primary human lung fibroblasts (pHLFs) was confirmed via RT-qPCR and Western blotting. RhoA activation was measured using G-LISAä and TGFβ activity via TMLC coculture assay and Western blotting for phospho-SMAD2. Profibrotic gene expression was measured by RT-qPCR. Fibroblast proliferation was assessed using BrdU incorporation by ELISA and time-lapse live cell imaging. In IPF patient-derived precision-cut lung slices (PCLS), nuclear phospho-SMAD2 accumulation was assessed via immunofluorescence and hydroxyproline levels quantified using high-performance liquid chromatography in PCLS conditioned media.

**Results** Both IP and PPARβδ receptors were present in pHLFs, with PPARβδ more abundant. Treprostinil inhibited LPA-induced RhoA activation and this effect was not reversed by the presence of an IP antagonist. Treprostinil did not inhibit LPA-induced TGFβ activation in the TMLC assay or SMAD2 phosphorylation. However, Treprostinil depletes total SMAD2/3 likely via cAMP and this correlates with a rise in phospho-YAP. Fibroblast proliferation was also inhibited in a concentration-dependent manner by Treprostinil and there were additive effects when combined with nintedanib, pirfenidone, or nerandomilast. Treprostinil downregulated TGFβ-response genes in pHLFs including *FN* and *SERPINE1*, as well as nuclear phospho-SMAD2 and hydroxyproline content levels in the PCLS model.

**Conclusions** Treprostinil inhibits RhoA activation and this may be via PPARβδ activation. Treprostinil did not affect SMAD2 phosphorylation but did deplete total SMAD2/3 in a cAMP dependent manner in pHLFs. Antifibrotic effects of Treprostinil were detected in IPF derived PCLS. Further studies are ongoing to establish the underlying mechanism as there were additive effects of Treprostinil with established antifibrotic agents which may have important clinical implications.

# Enhancing outcomes in respiratory failure: from trials to real world practice

## S92 ESTABLISHING REAL WORLD QUALITY STANDARDS FOR DOMICILIARY NON-INVASIVE VENTILATION

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10.1136/thorax-2025-BTSabstracts.98

**Introduction** Chronic Obstructive Pulmonary Disease (COPD) places a significant burden on healthcare. Non-invasive ventilation (NIV) is an established treatment for acute respiratory failure in COPD, with comprehensive guidelines.[1] However, evidence for its use in the domiciliary setting is more recent, with no standardised criteria for initiating or monitoring treatment. Trials such as HOT-HMV[2] demonstrated clinical benefit but excluded patients with BMI >35 or obstructive sleep apnoea (OSA), highlighting limited real-world applicability. We hypothesised that testing local standards in real-world COPD populations could offer insight into realistic expectations for national domiciliary NIV services.

**Objectives** We developed local criteria for domiciliary NIV initiation and follow-up, assessing their feasibility in routine care to inform service design. We also characterised our COPD population to evaluate how well existing research[2] applies to real-world patients.

**Abstract S92 Table 1**

| Proposed Standard | Target% | Evaluation | Performance n (%) |
|---|---|---|---|
| • Hypercapnia at discharge (inpatients) | ≥90% with $pCO_2$ >6.5 kPa | Reviewed discharge (or closest) blood gases for hypercapnia. | 78/109 (72%) |
| • Pre-NIV assessment (outpatients) | ≥90% with sleep study and Capillary Blood Glucose (CBG) test | Confirmed both tests were/weren't performed prior to NIV initiation. | 14/31 (45%) |
| • NIV usage | ≥70% of days with >4h/night in first year | Usage data from Lumis devices assessed over first 365 days. | 20/36 (56%) |
| • Follow-up within 6 weeks | 100% seen within 6 weeks | Compared treatment start date to first clinic/phone review. | 21/167 (13%) |
| • CBG testing at first clinic if $SpO_2$ <92% | 100% with $SpO_2$ <92% should have CBG | Checked clinic letters for $SpO_2$ and corresponding CBG tests. | 24/48 (50%) |
| • Clinic attendance (first year) | ≥80% attend ≥3 respiratory clinics | Counted all respiratory clinic visits at ~3, 6, and 12 months. | 96/118 (81.36%) |
| • Reduction in hospital admission | Pre-NIV admissions vs Post-NIV admissions for patients with at least 12 months follow-up time at the point of data collection. | Compared unplanned admissions 12 months before and after NIV start. | Mean admissions/patient: 3 pre-NIV vs 2 post-NIV, n=107 patients, 33% reduction |

**Methods** This retrospective observational study used cross-sectional and longitudinal data from 181 COPD patients initiated on domiciliary NIV between 1st July 2021 and 1st July 2024 within an NHS trust. Seven local standards (table 1) were defined using clinical reasoning alongside available guidelines. Demographics were evaluated at initiation. Patients with ≥12 months of follow-up (n=107) were assessed for hospital readmission, comparing admissions in the year before and after NIV initiation.

**Results** Demographics: 62% female, mean BMI 34 (n=138), 48% BMI >35. 35% had OSA, 67% ≥1 additional respiratory diagnoses. Performance against quality standards was variable (see table 1). However, there was a 33% reduction in hospital admissions among patients with ≥12 months follow-up, and 81% attended ≥3 respiratory clinics in the first year on treatment.

**Conclusions** This study trialled proposed quality standards and examined the real-world COPD population. Our findings show that trial populations do not reflect real-world COPD cohorts, limiting research generalisability. Many standards proved overly ambitious, highlighting the need for evidence-based, achievable national guidelines. These findings support the need for a national audit to inform realistic, standardised domiciliary NIV pathways.

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## S93 CLINICIAN PERSPECTIVES ON THE NEED & FEASIBILITY FOR AN RCT COMPARING NASAL HIGH FLOW THERAPY TO CONTROLLED OXYGEN IN COPD PATIENTS INTOLERANT OF NIV


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**Background** Non-invasive ventilation (NIV) remains the gold-standard therapy for acidotic acute hypercapnic respiratory failure (AHRF) in COPD, but around 20% of patients are unable to tolerate it. In the event of NIV failure, invasive ventilation (IMV) is only rarely offered, and in-hospital mortality exceeds 40%. Currently, excluding IMV no proven therapy exists beyond controlled oxygen and supportive medical care. Nasal High Flow Therapy (NHFT) improves survival in hypoxic respiratory failure and improves gas exchange in AHRF, but there is no robust evidence for improved survival in NIV-intolerant patients with COPD. NHFT is increasingly used in this setting without RCT data. If practice becomes entrenched, clinical equipoise may be lost undermining the feasibility of a future trial. A similar trajectory occurred in early UK experience with NIV, where uncontrolled implementation without adequate evidence or governance led to unexpectedly high mortality and delayed service reform. (National COPD Audit 2003).

**Aims** We conducted a survey among 16 respiratory clinicians experienced in NIV/RSU care to understand; (a) need for a trial, (b) current NHFT use, (c) feasibility, (d) trial design, (e) appropriate endpoints.

**Results** All (n=16) agreed there is immediate need for a trial and 15 were willing to participate. A traditional RCT was deemed unfeasible due to the time-critical nature of intervention. Strong support was expressed for a Cluster RCT, randomising centres rather than individual patients. Considering the potential benefit and recruitment challenges, 70% favoured powering the trial to detect a 15% absolute 30 day mortality reduction, balancing clinical relevance with recruitment feasibility. 2 centres reported potential reservations/barriers to participation due to entrenched NHFT use and loss of equipoise. One centre was already using NHFT in NIV intolerant patients.

**Conclusions** There is an urgent need to evaluate NHFT in this high-risk, under-researched group. A cluster RCT, powered for a 30 day mortality, is feasible, but must be undertaken before widespread adoption makes such a study untenable.

## S94 THE LIVED EXPERIENCES OF PATIENTS USING POSITIVE AIRWAY PRESSURE (PAP) THERAPY: A NESTED PHENOMENOLOGICAL STUDY WITHIN THE 3DPIPPIN RANDOMISED CONTROLLED TRIAL


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10.1136/thorax-2025-BTSabstracts.100

**Introduction and objectives** Positive Airway Pressure (PAP) therapy requires long term commitment from patients. Understanding the lived experience of PAP therapy users is crucial to support successful treatment and identify research priorities. Published data predominantly explores the negative experiences of PAP.

This study aims to explore the lived experiences of patients using PAP therapy.

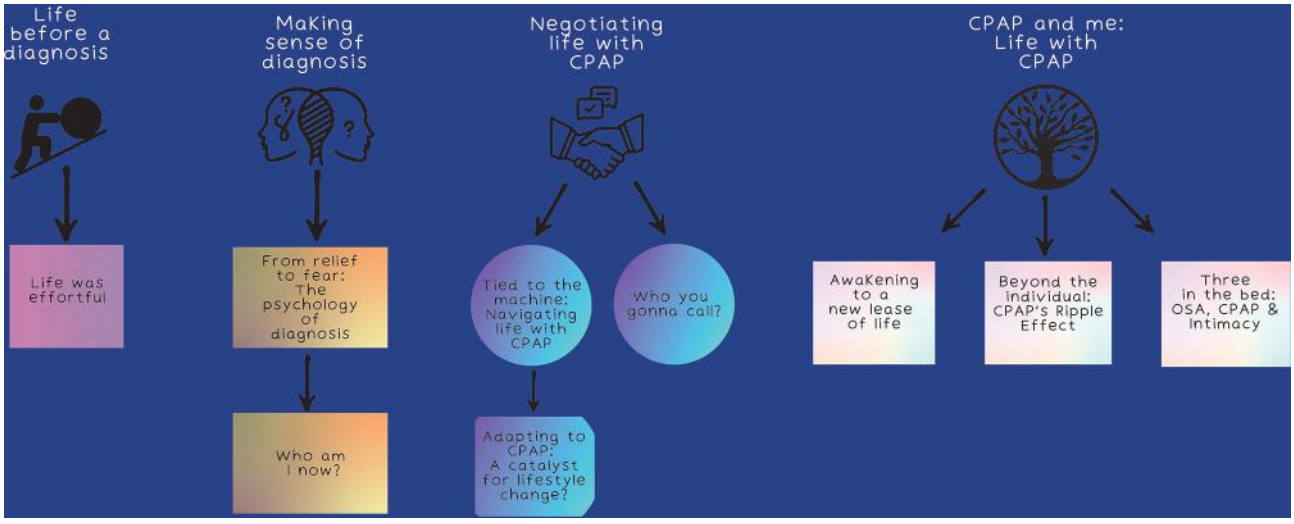
**Methods** This study employed an interpretive phenomenological approach. Heideggerian theory underpins the research, recognising the researcher's beliefs influence meaning thus allowing for rich analysis.

This was a nested study: participants were recruited from within a randomised controlled trial (RCT). Purposive sampling was used for maximal diversity.

Data were collected through 1-to-1 semi-structured interviews and analysed using Braun and Clarke's six-phase Reflexive Thematic Analysis. Analysis was conducted inductively through an interpretivist lens and managed using qualitative data analysis software (NVivo 14).

**Results** Twenty-nine participants were recruited, of which 45% (13) were women and 48% (14) were concordant with PAP therapy. Further, 62% (18) were Caucasian, 17% (5) Asian, and 21% (6) Black. Age groupings: 24% (7) were over 65 years, 52% (15) were 40–64 yrs and 21% (6) were 18–34 yrs.

The findings are presented as four overarching themes which capture and present the journey of people's lived experience with OSA and PAP therapy: 1) Life before a diagnosis, 2) Making sense of diagnosis, 3) Negotiating life with CPAP and 4) CPAP and me: Life with CPAP. Seven themes and two sub-themes are presented and displayed in the concept map (figure 1). This phenomenology provides insights into the lived experience that could inform individual clinical encounters and service design. Clinical practice often frames success




**Abstract S94 Figure 1**

purely in terms of CPAP concordance. This research shows that meaningful use in the context of people's lives and relationships is equally important.

**Conclusions** This phenomenology has brought together the existential and practical aspects of living with CPAP. It highlights the complex ways in which OSA and CPAP influence the individual's lived experience and the effect on society. The value of phenomenological research in applied clinical contexts is highlighted and its potential to inform more compassionate, person-centred care.

## S95 REMOTE MONITORING TO IMPROVE LOW ADHERENCE TO NON-INVASIVE VENTILATION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA/OBESITY HYPOVENTILATION SYNDROME (READ-NIV TRIAL)

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10.1136/thorax-2025-BTSabstracts.101

**Background** Chronic hypercapnic respiratory failure in patients with Obstructive Sleep Apnoea/Obesity Hypoventilation Syndrome (OSA/OHS) is commonly treated with non-invasive ventilation (NIV). However, adherence to NIV can be challenging, with substantial non-compliance occurring due to factors such as discomfort, aerophagia and claustrophobia. The primary aims of this study were-to assess whether remote monitoring could improve and optimise NIV adherence, to test patients' willingness to use remote monitoring devices, and to evaluate the associated healthcare resource usage and improvement in symptoms.

**Methods** The READ-NIV trial was a prospective, randomised, and controlled (usual care) trial with a two-arm parallel group design, testing remote monitoring of home NIV (T4P device, SRETT, Paris, France) for 3 months in patients with OSA/OHS who have previously had low NIV adherence (<4 hours/night). The trial was approved by the research ethics committee/Health Research Authority (HRA) [Integrated Research Application System (IRAS) ID: 270108], as well as by Guy's & St Thomas' NHS Foundation Trust R&D Department The study was registered at ClinicalTrials.gov (NCT04884165).

**Results** A total of 32 participants were enrolled and randomly assigned to intervention (n=16) or to usual care (n=16). Both study arms were similar in terms of age 61.5 (49.2; 70.2) *vs* 66.5 (51.0; 71.0) years, gender (male: female, 10:6 *vs* 11:5) and body mass index, BMI 37.7 (34.0 ;48.4) *vs* 42.8 (39.7; 48.2) kg/m$^2$. In the intervention arm, ΔNIV usage was +0.3 hours (95% CI -0.1; 3.2) hours/night; p=0.48). In the control arm, ΔNIV usage was +0.5 hours (95% CI -0.5; 2.0); p=0.32). The difference in NIV usage between groups at 12 weeks was not significant: -0.2 hours (95% CI: -2.3; 2.0) hours/night; p=0.90). Quality of life outcomes did not differ significantly between the intervention and usual care arms at the end of the trial.

**Conclusion** This randomised controlled trial aimed to improve adherence over 12 weeks using remote monitoring of NIV in patients with OSA/OHS. The intervention was associated with a modest but non-significant increase in adherence. These findings highlight the challenges of remote monitoring of patients with low long-term adherence.

## S96 WEIGHT MANAGEMENT IN PATIENTS WITH DECOMPENSATED OBESITY HYPOVENTILATION SYNDROME: UNDERSTANDING THE CHANGING LANDSCAPE IN THE ERA OF WEIGHT LOSS INJECTIONS

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**Background** Obesity-hypoventilation syndrome (OHS) is defined as a combination of obesity (BMI of ≥30 kg/m$^2$), raised $PaCO_2$ when awake, and breathing abnormality during sleep with or without obstructive sleep apnoea (OSA).[1]Patients can present to hospital in acute hypercapnic respiratory failure. The mainstay of acute treatment of such patient remains stabilisation with acute non-invasive ventilation (NIV) and consideration of home ventilation. However, long term management should include weight-loss intervention.[1] With the

improved evidence base supporting weight-loss pharmacotherapy, and improved access to such treatments,[2] it is crucial that patients appropriate for these interventions are identified. Currently little is known about the number of OHS patients that meet eligibility for these drugs.

**Methods** We conducted a retrospective analysis of patients admitted to a single-centre Respiratory Support Unit (June-December 2024) with OHS requiring acute NIV, including patients with comorbid conditions (e.g. COPD) but where OHS was the principal diagnosis. Clinical notes were reviewed for weight loss interventions offered during admission. Tirzepatide eligibility by both NHS England 2025 criteria (BMI $\geq$40 with 4 of 5 obesity related health conditions; diabetes, hypertension, hypercholesterolaemia, ischaemic heart disease, OSA) and proposed 2026 criteria (BMI $\geq$30 with 3 of those 5 conditions).

**Result** Among 377 admissions, 35 patients (9.3%) had OHS and were included. Only 3 (9%) were offered weight loss intervention/advice during admission. One patient was offered referral to a structured weight management programme. 10 patients (29%) met the 2025 criteria for Tirzepatide, whilst 21 (60%) would meet the proposed 2026 criteria.

**Conclusion** These findings demonstrate an emphasis on acute treatment for decompensated OHS but with a lack of long-term weight management strategies, despite evidence linking weight loss to improved outcomes. With the advent of weight-loss pharmacotherapy identifying appropriate patients promptly is essential. We have demonstrated that around 1/3 patients presenting with decompensated OHS meet current criteria for Tirzepatide. With the proposed expansion of criteria in 2026, it is likely that 2/3 patients will be eligible. Respiratory and primary care teams urgently need to develop robust pathways to ensure appropriate access to evidence based treatments for this overlooked patient group.

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## S97 FACTORS ASSOCIATED WITH OVERALL SURVIVAL IN PATIENTS WITH MOTONEURON DISEASE AND RESPIRATORY SYMPTOMS

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10.1136/thorax-2025-BTSabstracts.103

**Background** Motoneuron disease (MND) is a rare, progressive neuromuscular condition with usually guarded prognosis. Long-term non-invasive ventilation (LT-NIV) can prolong overall survival; however, due to disease heterogeneity it is unclear which patient might benefit from LT-NIV. The aim of this project was to analyse the overall survival (OS) of patients with MND referred to our tertiary long-term ventilation service.

**Methods** We studied 73 patients with established MND (68 $\pm$12 years, 73% men). Overall survival from the referral to our service was analysed with Cox-regression and Log-rank tests.

**Results** Amyotrophic Lateral Sclerosis (ALS) was the most common MND (n=63). Patients with ALS tended to have lower OS compared to non-ALS MND (median survival 25 vs. 51 months, p=0.06). In patients with ALS, whilst bulbar onset was not associated with reduced overall survival (p=0.18), the 5 year survival of patients with bulbar ALS was 28% compared to non-bulbar patients (42%). In non-hypercapnic patients with ALS (pCO2<6 kPa, n=42), LT-NIV (n=13) did not significantly prolong OS (p=0.99). In patients with daytime hypercapnia, 2 patients refused LT-NIV. Their median survival was 5 months versus 22 months who accepted LT-NIV. Age, sex, riluzole use or comorbidities did not affect OS (all p>0.05).

**Discussion** It is important to distinguish patients with ALS and non-ALS MND, as the former is associated with worse prognosis, especially in bulbar-onset subtype. LT-NIV is associated with survival benefit in patients with hypercapnia.

# Co-morbidity and novel treatments in COPD

## S98 TWO DECADES, TWO DESTINIES: WHEN CHRONIC OBSTRUCTIVE PULMONARY DISEASE HEARTS BEAT DIFFERENTLY – THE DIVERGENT MORTALITY TRAJECTORIES OF ATRIAL FIBRILLATION VS OTHER ARRHYTHMIAS

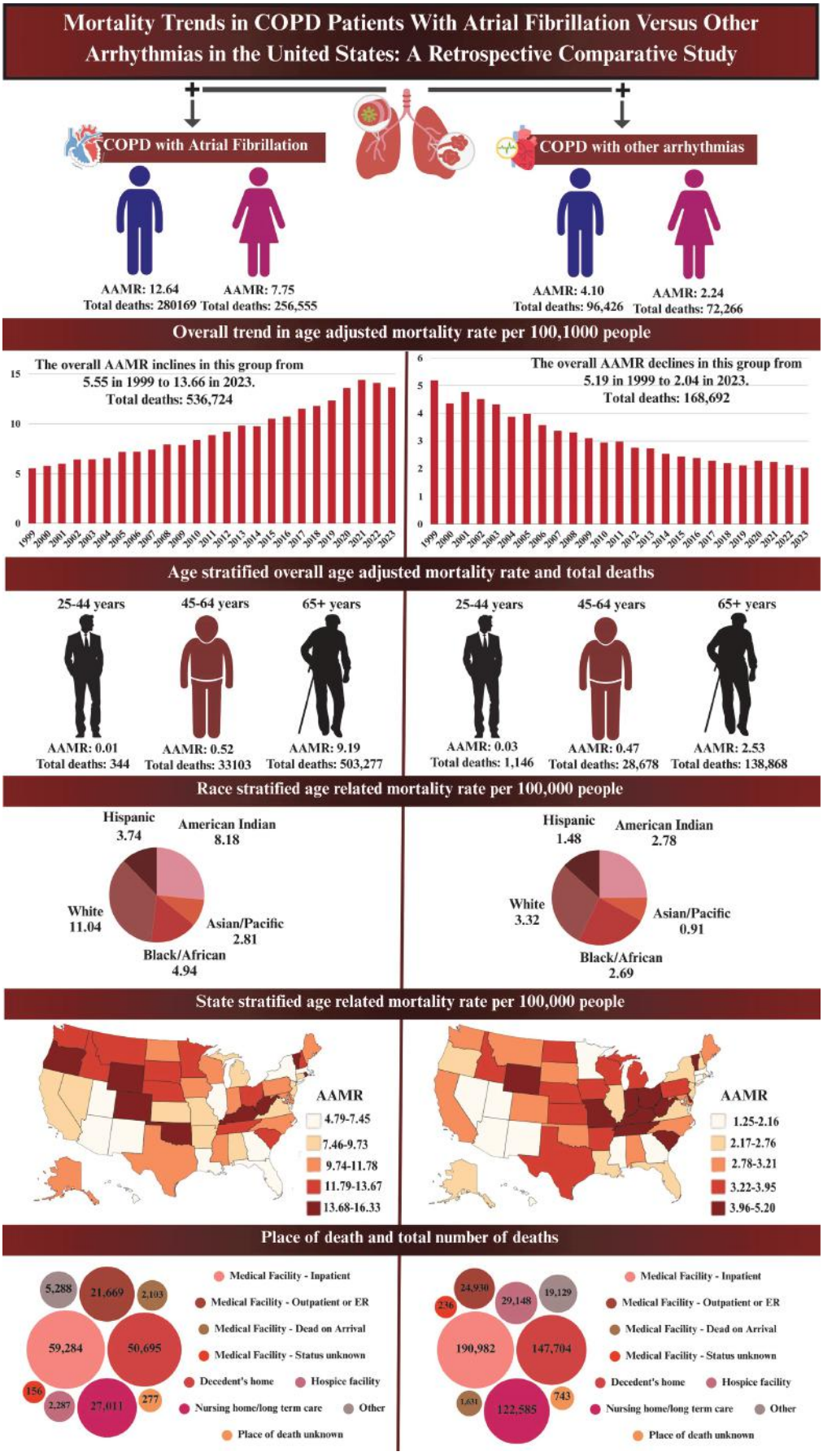
[1]MR Sarfraz, [2]MF Hemida, [3]A Ali, [4]S Ishtiaq, [5]K Patel, [2]M Hussein, [6]P Tabasum, [7]A Basit Kayani, [7]H Mehmood, [8]I Mushtaq, [9]S Rehman. *[1]Allied Hospital, Faisalabad Medical University, Faisalabad, Pakistan; [2]Alexandria Faculty of Medicine, Alexandria, Egypt; [3]Shrewsbury and Telford Hospital NHS Trust, Shrewsbury, UK; [4]Rawalpindi Medical University, Rawalpindi, Pakistan; [5]C. U. Shah Medical College, Surendranagar, Gujarat, India; [6]Peoples University of Medical and Health Sciences for Women, Nawabshah, Pakistan; [7]Hitec institute of medical science, Taxila, Pakistan; [8]Guys and St Thomas NHS Foundation Trust, London, UK; [9]Bahria University Health Sciences, Karachi, Pakistan*

10.1136/thorax-2025-BTSabstracts.104

**Background** While arrhythmias are recognized as potential causes of death in chronic obstructive pulmonary disease (COPD) patients. However, temporal trends in arrhythmia-related mortality among COPD patients remain unexamined. Therefore, we conducted a comparative study evaluating mortality trends between atrial fibrillation (AF) and other arrhythmias in COPD patients.

**Methods** A retrospective analysis of was conducted from 1999–2023, using the CDC WONDER database comparing COPD patients with AF (ICD-10: I48) versus other arrhythmias (ICD-10: I47, I49). Age-adjusted mortality rates (AAMRs) per 100,000 population were stratified by demographic variables for adults $\geq$25 years. Joinpoint regression estimated average annual percent changes (AAPC) in mortality trends.

**Results** From 1999–2023, 537,088 COPD-AF deaths were recorded (280,378 Men; 256,710 Women). AAMRs increased significantly from 5.55 to 13.66 (AAPC: +3.87%). Conversely, 168,770 COPD patients with other arrhythmias died (96,472 Men; 72,298 Women), with AAMRs declining significantly from 5.19 to 2.04 (AAPC: -3.56%). Men consistently showed higher mortality rates in both COPD with AF and other arrhythmias. In COPD-AF, AAMRs increased significantly for both genders (p<0.000001): men (7.96 to 16.95; AAPC: +3.23%) and women (4.17 to 11.19; AAPC: +4.18%). Conversely, in the COPD with other arrhythmias cohort, mortality rates decreased significantly (p<0.000001) for both men (AAMR: 7.68 to 2.67; AAPC: -4.05%) and women (AAMR: 3.58 to 1.52; AAPC: -3.16%). Inpatient medical facilities were the most common place of death for




**Abstract S98 Figure 1**

both groups, though COPD-AF patients had fewer inpatient deaths (59,284) than those with other arrhythmias (190,982). Both cohorts showed a notable shift toward increased home deaths over the study period. Racially, Whites had the highest AAMRs in both groups (AF: 11.04; other: 3.32), followed by American Indians (AF: 8.18; other: 2.78). Regionally, the Midwest showed highest mortality with opposing trends: upward for AF (AAPC: +4.89%) and downward for other arrhythmias (AAPC: -3.30%) $p<0.000001$. At state level, Vermont had the highest COPD-AF mortality (AAMR: 16.33), while Ohio had the highest AAMR for other arrhythmias (5.20).

**Conclusion** COPD-AF mortality increased dramatically while other arrhythmia mortality declined significantly. Men showed consistently higher mortality with notable demographic disparities. These opposing trends suggest AF represents an emerging threat requiring targeted interventions.

## S99 CARDIAC ARRHYTHMIAS DURING STABLE COPD

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10.1136/thorax-2025-BTSabstracts.105

**Background** Cardiac disease is common in those with COPD, who have double the odds of cardiac arrhythmias compared to those without COPD.[1] We used ambulatory cardiac monitoring to investigate if arrhythmias occurred during periods of disease stability in people with COPD.

**Methods** Patients enrolled within the London EXCEL COPD cohort were reviewed at disease stability: had blood samples taken, performed post-bronchodilator spirometry, and instigated 7-day ambulatory cardiac monitoring (Eclipse™ Pro, Spacelabs Healthcare, USA). We used longitudinal data for the number of exacerbations in the prior 12 months. Atrial fibrillation (AF)-like activity was defined as irregular ventricular response to atrial driven activity with no discernible p waves for <30s. Frequent premature atrial complexes (fPAC) were defined as any period of ≥30 premature atrial complexes (PACs)/hour. Frequent premature ventricular complexes (fPVC) were defined as premature ventricular complexes (PVCs) being >20% of total complexes.

**Abstract S99 Table 1** Presence of arrythmias during 7-day cardiac monitoring in stable COPD

| | N (%) (study population, n = 22) |
|---|---|
| ≥one SVT period | 12 (55) |
| ≥one NSVT period | 6 (27) |
| AF-like activity | 3 (14) |
| Either ≥one SVT period OR ≥one NSVT period | 14 (64) |
| Neither SVT nor NSVT | 8 (36) |
| Either ≥one SVT period OR ≥one NSVT period OR AF-like activity | 16 (72) |
| fPAC | 7 (32) |
| fPVC | 0 |

**Results** Interim results include twenty-two patients (median age: 73.3 (IQR:69.2 – 76.3) years, 11 (50%) male, median $FEV_1$ %predicted (n=21): 67.2 (IQR:52.5 – 69.3), median troponin: 4.1 (IQR: 3.2– 5.8) ng/L, median brain natriuretic peptide (BNP): 33.0 (IQR:22.3 – 83.8) ng/L) who completed ambulatory cardiac monitoring; median duration was 169.5 (IQR 167.0 –177.8) hours. Eight (36%) had ≥2 exacerbations in the previous 12 months. The frequency of arrythmias present is shown in table 1. There was no association with presence of supraventricular tachycardia (SVT), non-sustained ventricular tachycardia (NSVT), AF-like activity or fPAC and $FEV_1$%predicted, exacerbation frequency or BNP. Median troponin was higher in those with SVT than without ($p=0.022$).

**Conclusions** Cardiac arrhythmias are common during COPD disease stability with 16 (72%) patients having one or more of SVT, VT or AF-like activity and 7 (32%) having fPAC. This may reflect undiagnosed underlying structural heart disease, possibly worsened by COPD, and potentially benefitting from cardiac imaging assessment.

### REFERENCE

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## S100 RELATIONSHIP OF CARDIAC BIOMARKERS IN COPD AT STABLE STATE AND EXACERBATION

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10.1136/thorax-2025-BTSabstracts.106

**Introduction** Cardiac biomarkers are frequently raised in patients with COPD. Our aim was to understand the relationship between troponin and BNP at both stable state COPD and during an exacerbation.

**Methods** Patients with COPD were prospectively recruited from 14 GP practices as part of the STARR2 study.[1] Blood sampling was obtained at stable state, exacerbation (day 0), and 14 and 30 days later. Troponin and BNP levels were measured at each time point using commercial ELISA kits (R&D systems, Troponin LLD 125pg/mL and R&D systems, BNP LDD 312pg/ml).

Spearman's rank test was undertaken to assess the relationship between troponin and BNP at stable state, and the relationship between troponin and BNP rise for each exacerbation.

**Results** 65 patients were included in the study, 66% were male, median age was 72 (IQR 66.5–76.5) years. 13 patients (20%) had IHD, 5 (7.6%) known atrial fibrillation and 3 (4.6%) a diagnosis of heart failure.

A significant positive correlation was found between troponin and BNP at stable state ($r=0.53$; $p<0.001$) but no correlation was observed between peak troponin rise and peak BNP rise within 30 days of moderate COPD exacerbation ($r=0.071$; $p=0.57$). We identified 4 clusters of cardiac biomarker changes during exacerbation: 10 patients (15.4%) had a significant rise in both troponin and BNP, 11 patients (16.9%) had a significant rise in troponin only; 15 patients

(23.1%) had a significant rise in BNP only and 29 patients (44.6%) had neither a troponin nor BNP rise.

**Conclusion** To our knowledge this is the first study investigating the relationship between different cardiac biomarkers in COPD at stable state and exacerbation. BNP and troponin levels exhibited a similar pattern of baseline cardiac stress at stable state but no relationship was observed between troponin and BNP rise during moderate exacerbation. These results suggest that different endotypes of cardiac injury may exist at exacerbation. Further work is needed to understand if there are different pathophysiological mechanisms accounting for different clusters of cardiac injury at exacerbation.

## REFERENCE

1. Ramakrishnan S, *et al*. Blood eosinophil-guided oral prednisolone for COPD exacerbations in primary care in the UK (STARR2). *Lancet Respir Med*. 2024.

## S101 IMPACT OF DUPILUMAB IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH CARDIOVASCULAR OR METABOLIC COMORBIDITIES: BOREAS AND NOTUS TRIALS

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10.1136/thorax-2025-BTSabstracts.107

**Introduction and Objectives** Cardiovascular disease (CVD) and metabolic disease (metD) are common comorbidities in chronic obstructive pulmonary disease (COPD) and may modulate clinical outcomes and therapy response. In BOREAS and NOTUS, add-on dupilumab reduced exacerbations and improved lung function in patients with COPD and type 2 inflammation. Safety was consistent with the known dupilumab safety profile. This post hoc analysis assessed dupilumab efficacy in patients with COPD with/without CVD or metD.

**Methods** BOREAS (NCT03930732) and NOTUS (NCT04456673), phase 3 RCTs, enrolled patients (40– 85 years) with COPD, moderate-to-severe airflow limitation, and type 2 inflammation (screening blood eosinophils $\geq$300 cells/μL) on triple therapy. Patients received dupilumab 300 mg or placebo q2w for 52 weeks. Endpoints: annualized moderate or severe exacerbation rates, and change from baseline at Week 52 in pre-bronchodilator forced expiratory volume in 1 second ($FEV_1$) and St. George's Respiratory Questionnaire (SGRQ) total scores (range: 0–100 points; lower scores indicating better quality of life) in the pooled intention-to-treat population with/without investigator-reported CVD or metD.

**Results** Of 1,874 patients, 1,253 (66.9%) had a history of CVD and 758 (40.4%) of metD. Dupilumab reduced exacerbation rates by 31–33%, with relative risk vs placebo (95%CI) of 0.69 (0.59, 0.82; $P<0.001$) with CVD, 0.67 (0.51, 0.89; $P=0.005$) without CVD, and 0.68 (0.54, 0.85; $P<0.001$) with metD, 0.69 (0.57, 0.84; $P<0.001$) without metD. Dupilumab improved Week 52 pre-bronchodilator $FEV_1$ across all subgroups by LS mean difference vs placebo (95%CI) of 55 mL (18, 92; $P=0.004$) with CVD, 104 mL (42, 166; $P=0.001$) without CVD, and 62 mL (12, 112; $P=0.015$) with metD, 77 mL (35, 119; $P<0.001$) without metD. Dupilumab also reduced Week 52 SGRQ total scores by LS mean difference vs placebo (95%CI) of −3.1 points (−4.9, −1.2; $P=0.001$) with CVD, −3.3 points (−6.1, −0.4; $P=0.026$) without CVD, and −2.5 points (−4.8, −0.1; $P=0.040$) with metD, −3.9 points (−6.0, −1.9; $P<0.001$) without metD. All interaction $P$ values for data presented were >0.05.

**Conclusion** Dupilumab reduced moderate or severe exacerbation rates, and improved lung function and quality of life in patients with COPD and type 2 inflammation, regardless of comorbid CVD or metD.

## S102 THE POTENTIAL EFFECT OF GLUCAGON-LIKE PEPTIDE 1 RECEPTOR AGONISTS (GLP1S) ON CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) EXACERBATIONS, A CASE CONTROL ANALYSES

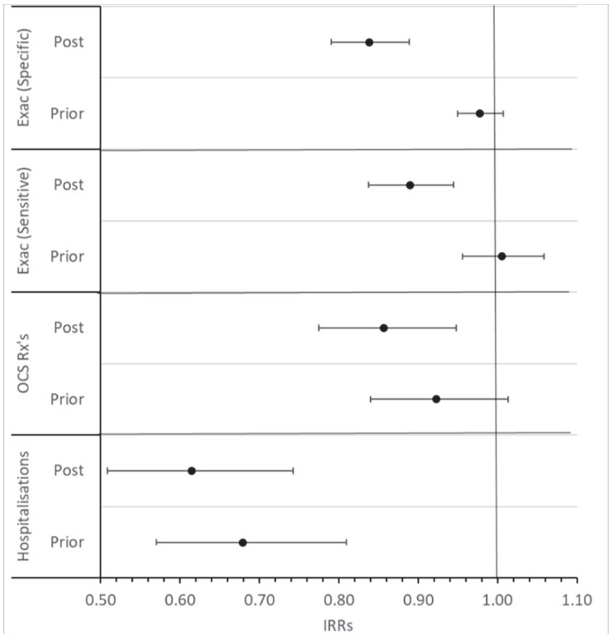
[1]B Alcázar, [2]H Heatley, [3]A Kaplan, [4]TT Lee, [5]MS Koh, [6]N Sattar, [2]J Townend, [2]D Skinner, [2]V Carter, [2]D Price. *[1]Universidad de Granada, Granada, Spain; [2]Observational and Pragmatic Research Institute, Singapore, Singapore; [3]Family Physician Airways Group of Canada, Ontario, Canada; [4]National University Health System, Singapore, Singapore; [5]Singapore General Hospital, Singapore, Singapore; [6]University of Glasgow, Glasgow, UK*

10.1136/thorax-2025-BTSabstracts.108

**Introduction** Reducing the risk of exacerbations is a key objective of COPD management. Recently published studies have demonstrated that GLP-1RAs used for weight loss also have benefits on respiratory outcomes within asthma.[1]

**Aims** The aim of this study was to use real-world data from the Optimum Patient Care Research Database (OPCRD) to determine whether respiratory benefits might extend to a COPD population & Type 2 Diabetes.

**Methods** Our exposed cohort were prescribed a GLP-1RA, and had a diagnosis of COPD, type 2 diabetes, were over 40 years old at the time of the first GLP-1RA (index date). We identified an age, sex, smoking status and BMI matched control cohort of patients with COPD and diabetes who were contributing data to OPCRD at the time of their matched




**Abstract S102 Figure 1**

patients index date, their COPD treatments and number of exacerbations in the year prior to the GLP-1RA prescription.

Exacerbation outcomes included a) specific exacerbation measure (chest infection type diagnosis with an antibiotic or steroid prescription), b) sensitive exacerbation measure (antibiotic or steroid prescription without a chest infection type diagnosis), c) OCS prescriptions and d) hospital admissions in the year following GLP1 initiation.

**Results** The cases mean age was 66, 58% were male, the mean BMI was 36.25. Cases GOLD (2023) group classification were 16% A, 55% B, and 29% E. Controls were comparable. Patients receiving a GLP-1RAs had significantly lower risk of exacerbations (specific IRR 0.84: 0.79–0.89 p<0.001, sensitive IRR 0.89: 0.84–0.95 p=0.003). Cases had lower OCS prescriptions (IRR 0.86: 0.77–0.95 p<0.001) and respiratory hospitalisations (IRR 0.61: 0.41–0.74 p<0.001) compared with controls.

**Conclusions** This real-world study provides evidence that GLP-1RAs therapy maybe associated with a lower risk of COPD exacerbations in patients with comorbid T2D suggesting a potential protective effect of GLP-1 RAs on respiratory health. Furthermore, an approximately 20% lower risk in exacerbations and corticosteroid prescriptions, is clinically meaningful, especially in a population with high multimorbidity burden. Large scale randomised trials are now needed to test this potential.

## REFERENCE

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## S103 THE EFFECTS OF SODIUM/GLUCOSE CO-TRANSPORTER-2 (SGLT-2) INHIBITORS IN COPD PATIENTS' ADMISSION FREQUENCY, DURATION AND USE OF ACUTE NIV. A SINGLE-CENTRE 48-MONTH RETROSPECTIVE OBSERVATIONAL STUDY IN A UK TERTIARY CARE CENTRE

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10.1136/thorax-2025-BTSabstracts.109

**Background** SGLT-2 inhibitors are used extensively in the management of diabetes and heart failure given their powerful anti-hyperglycaemic and diuretic effects. Recent research has shown the multifaceted immunomodulatory and anti-inflammatory effects of SGLT2-inhibitors with recent research suggesting favourable outcome in patients with COPD.

**Objective** Our aim was to conduct a retrospective observational study into the relationship between SGLT-2 inhibitors and their effects on length of stay, frequency of admissions and use of acute NIV in patients admitted with an acute exacerbation of COPD between April 2022 and April 2024.

**Methods** Patients with COPD confirmed by spirometry and discharged with a primary diagnosis of 'exacerbation of COPD, infective or non infective' were identified from electronic records.

Patients with concurrent diagnoses during the admissions such as primary lung cancer and its complications, acute ischaemic heart disease or acute heart failure, complications from acute renal failure were excluded.

**Results** 627 patients, representing 1197 admissions, were identified. 32 patients received SGLT-2 inhibitors. Those receiving SGLT-2 inhibitors were associated with a 26% reduction in admission duration (CI 0.62–0.87; p≤0.001), accounting for age, sex, FEV1 and concurrent diagnosis of congestive heart failure and Type 2 Diabetes.

There was no significant reduction in the frequency of admissions or in the use of acute non-invasive ventilation in the SGLT-2 inhibitor cohort.

**Conclusion** Our study has a revealed that SGLT-2 inhibitors significantly reduce the duration of in-hospital admission for those with COPD. However, that there was no effect on the frequency of admissions or reduction acute NIV in this group. Nonetheless, this offers a tantalisingly new direction in the management of COPD patients.

# Deep dive into the asthma airways

## S104 BLOOD AND AIRWAY EOSINOPHILS IN SEVERE EOSINOPHILIC ASTHMA ARE PHENOTYPICALLY AND FUNCTIONALLY DISTINCT

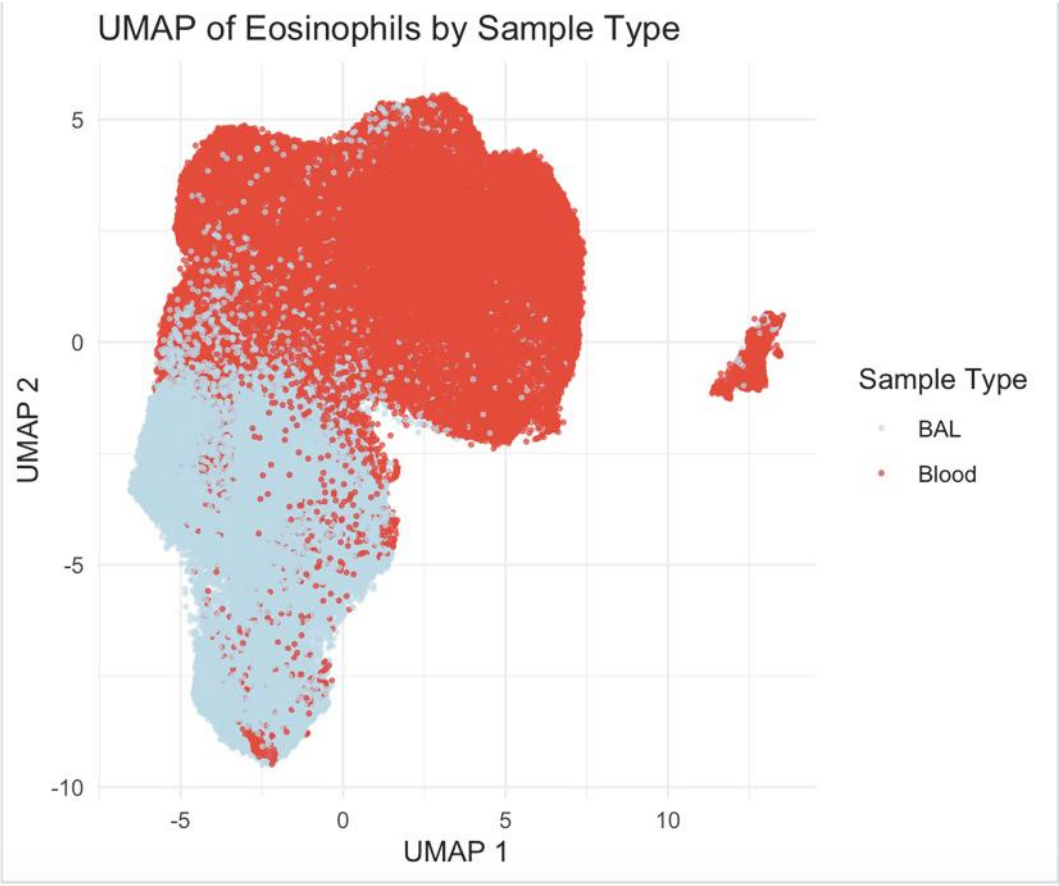
[1]J Gates, [2]P Khooshemehri, [2]S Cass, [2]J Baker, [1]A del Rosario, [2]V Kanabar, [1]J Dhariwal, [1]AM Nanzer, [2]RT Martinez Nunez, [1]DJ Jackson. *[1]Guys Severe Asthma Centre Guys and St Thomas NHS Trust, London, UK; [2]School of Immunology and Microbial Sciences Kings College London, London, UK*

10.1136/thorax-2025-BTSabstracts.110

**Introduction** Severe asthma is typically characterized by high levels of eosinophilic inflammation. Evidence from animal models and studies of mild-moderate asthma suggest there may be different inflammatory profiles of blood and bronchoalveolar lavage (BAL) eosinophils, but these differences remain poorly defined. Whether such differences exist in severe eosinophilic asthma (SEA) is unknown, however an understanding of such phenotypic and functional characteristics may be clinically relevant and inform biologic treatment choice.

**Methods** Paired peripheral blood and BAL samples were collected from 11 patients with SEA. CyTOF was performed on whole blood and BAL cells. Eosinophils were gated based on CD66b, CD294 and CD125 expression and then further subtyped based on functional and phenotypical markers. Following UMAP visualisation (figure 1), mass cytometry data underwent FlowSOM clustering to identify phenotypically distinct subpopulations. Expression levels of lineage and functional markers were compared between blood and BAL eosinophils. Relative cluster distributions were assessed using compositional analysis, and differential expression was statistically evaluated.

**Results** UMAP visualization revealed distinct clustering patterns of blood and BAL eosinophils, indicating phenotypic divergence (p<0.001). FlowSOM clustering identified 6 main metaclusters, 4 of which were enriched in BAL eosinophils, and 2 in blood eosinophils. Heatmap analysis highlighted differential expression of CD196 across the clusters of BAL eosinophils. Eosinophils with low CD196 expression had high IL-13 expression, suggesting a more migratory phenotype. The 2 main metaclusters of blood eosinophils were differentiated by CD45RA expression or high TSLPR expression. Additionally, the blood microenvironment was different to the BAL, with




**Abstract S104 Figure 1**

significantly higher levels of CCL11, CCL26, IP-10, MCP-4 and MIP3a (all $p<0.05$), whereas BAL showed significantly higher levels of GROa($p=0.03$) and CXCL8($p=0.01$).

**Conclusion** Simultaneous measurements of BAL and blood eosinophils from patients with SEA demonstrate specific compartment related differences in marker expression, with multiple subgroups of eosinophils present in each compartment. How these differences relate to baseline clinical features and response to anti-IL5R therapy is an area of ongoing investigation.

## S105 THE AIRWAY TRANSCRIPTOME IN TYPE2 CYTOKINE BIOMARKER-HIGH AND -LOW SEVERE ASTHMA

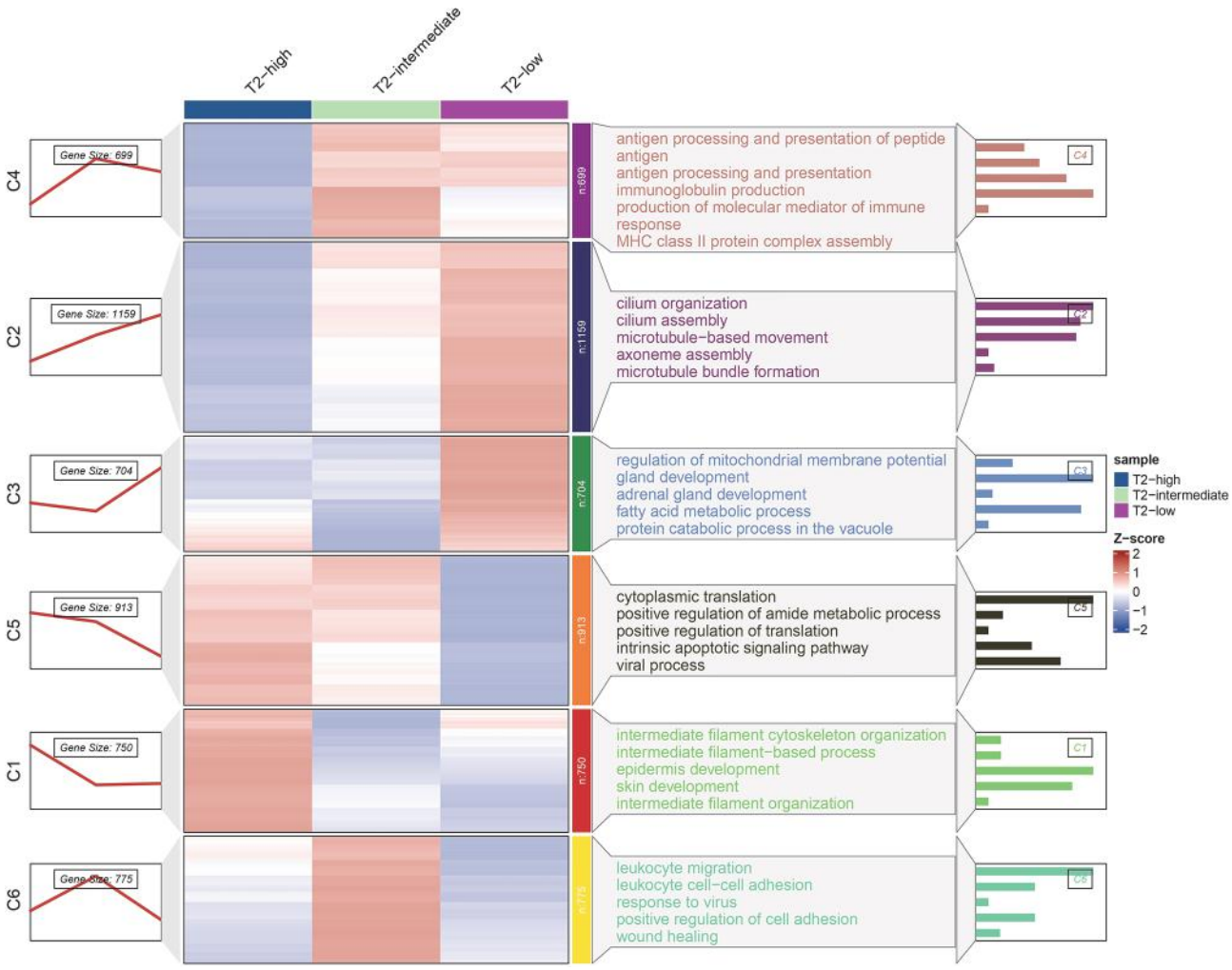
[1]J Shen, [2]R Chaudhuri, [2]S Bicknell, [3]A Mansur, [1]R Shrimanker, [1]I Pavord, [4]S Fowler, [5]V Brown, [5]L McGarvey, [6]P Howarth, [6]J Arron, [5]L Heaney, [7]D Choy, [1]E Marchi, [1]T Hinks, [8]P Bradding. *[1]NIHR Oxford Respiratory BRC, Nuffield Department of Medicine, University of Oxford, Oxford, UK; [2]Institute of Infection and Immunity, University of Glasgow, Glasgow, UK; [3]University of Birmingham and Heartlands Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK; [4]School of Biological Sciences, Faculty of Biology, Medicine and Health, University of Manchester, Manchester Academic Health Science Centre and NIHR Manchester Biomedical Research Centre, Manchester University Hospitals NHS Foundation Trust, Manchester, UK; [5]Wellcome-Wolfson- Centre for Experimental Medicine, Queen's University Belfast, Belfast, UK; [6]School of Clinical and Experimental Sciences, University of Southampton, NIHR Southampton Biomedical Research Centre, Southampton, UK; [7]Genentech, Inc., San Francisco, USA; [8]Department of Respiratory Sciences, University of Leicester, Leicester Respiratory NIHR BRC, Glenfield Hospital, Leicester, UK*

10.1136/thorax-2025-BTSabstracts.111

**Background** Severe asthma is a heterogeneous disease. The mechanisms underlying airway inflammation, particularly when type 2 (T2) cytokine activity is suppressed, remain poorly understood. This study aimed to identify the airways molecular pathways of T2 biomarker-high and biomarker-low severe asthma under biomarker-based optimised corticosteroid therapy.

**Methods** We analysed clinical data and transcriptomic data from the bronchial brushings and biopsies in the RASP-UK multi-centre severe asthma cohort. We included 18 corticosteroid-resistant T2 biomarker-high (T2-high), 23 corticosteroid-responsive T2 biomarker-intermediate (T2-intermediate) and 11 T2 biomarker-low (T2-low) patients with severe asthma and 20 healthy controls pre- and post-treatment with inhaled corticosteroids.

**Results** Severe asthma, independent of confounding by therapeutic corticosteroids, was characterised by upregulation of mucins (*MUC5AC*, *MUC2*), lysozyme, *CEACAM5*, *SYNCRIP*, typical T2-genes (*POSTN*, *IL33*), the transcription factor *FOS*, and *ITGB8*, which regulates *TGF*β activation and airway remodelling. FeNO and blood eosinophils share many common transcriptome signatures, such as *CCL26* and *MMP1*. However, some genes were associated only with eosinophils (e.g., *EDN2*) or FeNO (e.g., *POSTN*, *CST1*). Clinically, patients with T2-high asthma exhibited poorer pulmonary function, while those with T2-low asthma had an earlier disease onset, higher serum IgE levels, and experienced more frequent severe exacerbations. At the molecular level, T2-high asthma showed upregulation of T2-dependent genes, keratins, adaptive immune response, impaired ciliary function, and




**Abstract S105 Figure 1** Heatmap of gene cluster expression patterns across T2-high, -intermediate, and -low asthma. Heatmap of gene cluster expression patterns across T2-high, -intermediate, and -low asthma. Genes were grouped into distinct co-expression clusters (C1-C6) using *k*-means clustering based on shared expression profiles. Line plots to left show average expression profile of each gene cluster across phenotypes. Heatmap displays Z-score-normalized gene expression values with rows corresponding to genes and columns grouped by phenotype. Red indicates higher expression and blue indicates lower expression. Top enriched Gene Ontology (GO) biological process terms for each cluster are shown to right. Coloured bars reflect statistical significance of enrichment

epithelial development. T2-low asthma was marked by upregulation of Th1- and Th17-associated genes (*IDO1*, *CXCL10*, *GBP1*, *LAG3*), neuroimmune genes (*SGC2*, *CALCA*), interferon-γ signalling, antigen processing, and enrichment of neutrophils and mast cells. T2-intermediate asthma exhibited a mixed molecular profile, sharing features of both T2-high and T2-low endotypes. It was characterised by expression of the antimicrobial peptide *BPIFA1*, viral response signatures, mast and natural killer (NK) cell enrichment, and activation of transcription factors involved in pathogen defence (*NFKB1*, *TBX21*, *IRF1*, *IRF2*, *BATF*, *STAT2*). This group also showed heightened immune responses, impaired mitochondrial function, and enhanced antigen processing pathways.

**Conclusions** This study delineates distinct molecular signatures and pathways in three major severe asthma phenotypes, independent of corticosteroid effects. These findings offer insights for personalised asthma management and the development of targeted biologic therapies.

On behalf of the RASP-UK.

## S106 SPATIAL TRANSCRIPTOMIC PROFILING OF AIRWAY INFLAMMATION AND STRUCTURAL CHANGES IN ASTHMA

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The different mechanisms underlying distinct phenotypes of asthma remain poorly understood. While scRNA sequencing has improved our understanding of the cellular diversity in asthma, it lacks spatial context. To address this limitation, we applied high-resolution spatial transcriptomics to airway biopsies from healthy individuals and clinically stratified asthmatic patients from two specific phenotypes.

**Methods** We recruited 42 participants to a bronchoscopy study: 16 healthy control participants, 13 with early-onset atopic asthma with strong host dust mite sensitisation, 13

with adult-onset eosinophilic asthma. Bronchial biopsies were analysed by 10X Xenium with 389 predesigned and custom selected probes focussing on type 2 immune and remodelling pathways.

**Results** Early-onset asthma *vs* late-onset asthma had mean age of onset 5.6 *vs* 45 years, worse lung function ($FEV_1$74% *vs* 90% predicted), lower FeNO (30 ppb *vs* 80 ppb), lower blood eosinophils (0.3 *vs* 0.44 cells/mL) higher total IgE (376 IU/L *vs* 66 IU/L). We performed quality control, segmentation, and cell type annotation using Seurat and Xenium software. Healthy tissues showed higher transcript capture per cell and more consistent segmentation compared to asthmatic tissues. Cell type annotation revealed the spatial distribution of key cell types, including T cells, B cell lineage cells, fibroblasts, and smooth muscle cells, in both asthmatic and healthy samples.

Within epithelial cells genes differentially upregulated in late-onset asthma included *POSTN, NOS2, CCL26*, and *TSLP* consistent with strong epithelial type 2 inflammatory signalling. By contrast early-onset, highly atopic asthma was associated with upregulation of genes including *DMBT1* (which can suppress IL4, IL5 and cilial motility), *LTF*, genes involved in mucin hypersecretion including *MUC5B, SOX9* (a transcription factor involved in goblet cell metaplasia) and *KDR* (the *SOX9*-modulated VEGF receptor), the wnt b-catenin signalling modulator *SFRP1,* and the mast cell receptor *KIT*. Whilst the alarmin *TSLP* was upregulated in late-onset asthma, *IL33* expression was higher in health, linking its expression more closely with therapeutic steroids than asthma pathology. Neighbourhood analyses are ongoing to reveal spatial interactions between cell types.

**Conclusion** Spatial transcriptomics identifies features of disease-specific inflammation and remodelling underlying differences between phenotypes, and enabling characterisation of spatial relationships between epithelial, inflammatory and stromal cells.

## S107 CAN PLASMA CELL-FREE DNA METHYLATION PREDICT RESPONSE TO MEPOLIZUMAB IN ASTHMATIC INDIVIDUALS?

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**Rationale** Monoclonal antibody therapies are used to treat severe asthma. Mepolizumab was the first anti-cytokine therapy introduced, and it targets IL-5. It effectively reduces steroid use and exacerbations in some severe asthma patients but approximately one third of treated patients do not havea clinically significant reduction inexacerbations and oral steroid use at 12 months post-treatment. An early biomarker that was predictive of clinical response at one year would reduce patient burden, improve management and have associated cost saving.Cell free DNA (cfDNA) are DNA fragments released into bodily fluids carrying cell/tissue-specific epigenetic patterns called DNA methylation (DNAm) that reflect their origin. cfDNAm offers tissue specific specificity to systemic samples. We have shown DNAm changes in the nasal epithelium after Mepolizumab treatment [PMID: 38386780] and distinct plasma cfDNAm profiles between asthmatic and non-asthmatic individuals.

**Objective** To identify cfDNAm signatures that differ between Mepolizumab responders (R) and non-responders (NR) pre-treatment and after 3 months of treatment.

**Methods** cfDNA was extracted (Maxwell® RSC ccfDNA Plasma Kit) from 1 ml blood plasma of 14 asthmatic individuals (9 R; 5 NR identified at 12 months) at baseline and 3 months of Mepolizumab treatment [part of Investigating Poor Response to Monoclonal therapy in Asthma study]. DNAm was profiled using the Illumina EPIC Array. Linear regression assessed differential cfDNAm between R and NR.

**Results** A single DNAm locus was identified with a significant (Benjamini-Hochberg $p<0.05$) decrease (~8%) in methylation between baseline and 3 months among Mepolizumab responders, while non-responders exhibited an increase (~4%). A 213bp region around this locus with 4 adjacent loci was identified to be evaluated as potential early response biomarker. Linear regression analysis within each time point identified DNAm changes (pre-treatment = 1838; 3 months post treatment =1283, nominal $p<0.001$) between R and NR. The two most significant and consistent loci have been selected to evaluate as potential biomarkers.

**Conclusion** Plasma cfDNAm signatures have been identified for further investigation as early predictors of patient response to Mepolizumab. Evaluation of their potential as biomarkers using pyrosequencing in a blinded blood plasma sample cohort of Mepolizumab treated severe asthmatic individuals is ongoing.

## S108 MEPOLIZUMAB TREATMENT INFLUENCES AIRWAY REMODELLING AND IMPROVES EPITHELIAL CELL BIOLOGY IN SEVERE ASTHMA AS DETERMINED BY SINGLE CELL TRANSCRIPTOMICS

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**Introduction** Severe asthma is characterised by inflammation and structural remodelling of the airways, typically resistant to standard therapies. Mepolizumab, an anti-IL-5 biologic, reduces eosinophil levels and improves asthma control. Limited evidence exists investigating the effect of mepolizumab on airway remodelling and bronchial epithelium. We aimed to characterise the bronchial epithelial cell response to mepolizumab treatment at a single cell level with a focus on its potential role in modulating remodelling.

**Methods** Twenty patients with severe asthma (blood eosinophils $\geq$300 cells/uL at baseline) were recruited (NCT05144087). Good quality paired airway samples were obtained from 12 patients through bronchoscopy before and after 6 months of mepolizumab treatment. We performed single-cell RNA sequencing (scRNA-seq) on bronchial brushes using 3' chemistry. Data processing was done using Cell

Ranger, seurat DESeq2 paired analysis and CellChat. Pathway enrichment analyses were performed using gene set enrichment analysis (GSEA).
**Results** The IL5-Ra (alpha subunit of the IL5 receptor) gene was highly expressed in ciliated epithelial cells. After mepolizumab treatment, distinct transcriptional changes were seen in ciliated cells, basal cells, and ionocytes, with the highest number of differentially expressed genes (DEGs) in ciliated cells (325 DEGs). We found significantly decreased *SMAD2* mRNA levels and suppression of 28 SMAD2-dependent genes as well as changes in the expression of genes related to cell-cell junction (5 genes) and remodelling (20 genes). Overall, we observed a positive induction of pathways related to epithelial integrity and barrier function after mepolizumab treatment. Inferred cell-cell communication networks revealed a reduction in structural and remodelling pathway intercellular activity, showing reduced markers of epithelial damage and subepithelial fibrosis.
**Conclusion** In addition to targeting eosinophils, mepolizumab treatment also modifies the transcriptional profile of specific bronchial epithelial cell populations. Our data indicate that mepolizumab suppresses remodelling signals and restores epithelial barrier integrity and homeostasis, potentially via reducing the expression of *SMAD2*, a key transcription factor in TGF-beta signaling. Overall, our findings support a role for IL-5 in modulating airway structural cells and reveal previously unrecognised mechanisms by which anti-IL5 therapy may confer clinical benefit in severe asthma.

### S109 B CELL RECEPTOR REPERTOIRE SEQUENCING TO CHARACTERISE ANTIBODY SUBCLASS EXPRESSION AND SOMATIC HYPERMUTATION IN SEVERE EOSINOPHILIC ASTHMA AND EGPA

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**Introduction** Severe eosinophilic asthma (SEA), eosinophilic granulomatosis with polyangiitis (EGPA) and nasal polyposis (NP) are immune-mediated diseases characterised by eosinophilic inflammation. However, there is also increasing interest in the potential pathological roles of auto-antibodies in these diseases.
**Objectives** Understanding the antibody repertoire in these conditions may provide valuable insights into disease mechanisms, and contribute to our understanding of potential roles of targeted biologic therapies.
**Methods** We conducted B cell receptor repertoire sequencing using peripheral blood from 43 patients, comprising SEA with nasal polyps (SEA+NP), SEA without nasal polyps (SEA-NP), and EGPA, along with 16 healthy controls (HCs).
**Results** Compared to HCs, patients with EGPA exhibited increased relative proportions of IgA1, IgG1, IgG2, and IgG4 subclasses. Similarly, SEA-NP patients demonstrated a significantly higher proportion of IgG2 compared to HCs. Notably, the IgG4 subclass was significantly elevated across all patient groups compared to HCs. Patients receiving anti-IL-5/5R biologic treatments showed increased relative proportions of IgA2, IgG2, and IgG3 subclasses compared to untreated patients. Conversely, the proportions of IgE and IgM were decreased in patients on anti-IL-5/5R biologic therapies.

Variation across participant groups in mean somatic hypermutation was evident with an increased mutation frequency in IgG3 in SEA-NP patients and decreased mutation frequency in IgA2 in SEA+NP and EGPA patients. No differences were evident in mutation frequencies between those patients on and not on anti-IL-5/5R biologics, except for IgM.
**Conclusions** Our preliminary data is consistent with a pattern of a more mature B cell component in the periphery and with the T2 inflammatory response observed in SEA and EGPA. Analysis of patterns of somatic hypermutation among clonally expanded sequences may be necessary to determine whether the IgE and IgG4 compartments in the peripheral blood are subject to antigen-driven clonal expansion.

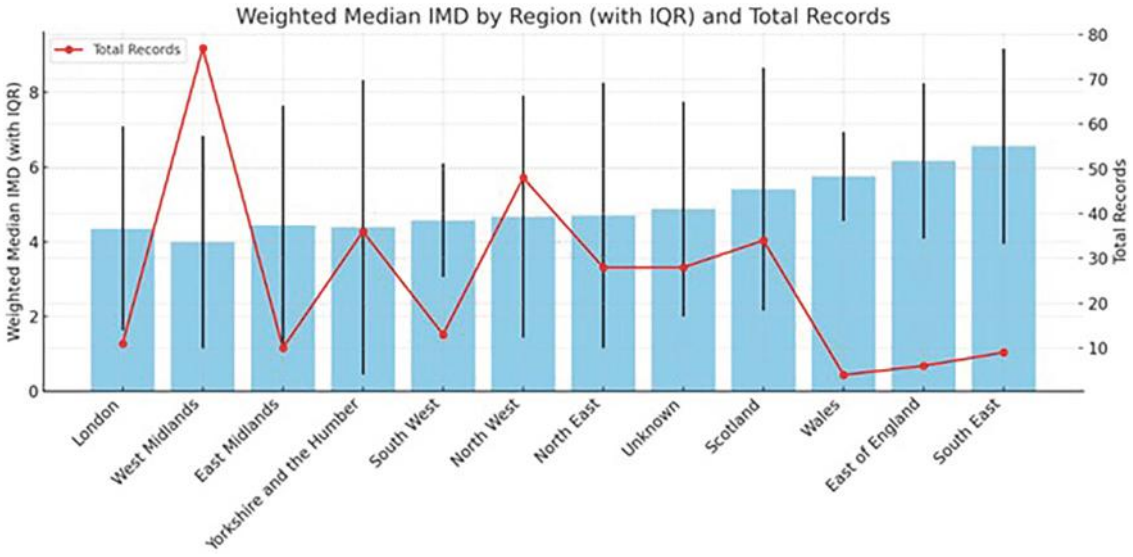
## Occupational hazards of exposure to particulates

### S110 LOCATION AND DEPRIVATION IN SILICOSIS CASES: DATA FROM THE SWORD SCHEME 1996–2024

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**Introduction** Silicosis is a preventable pneumoconiosis, and cases continue to be reported in the UK. Silicosis more prevalent in areas with industries where silica exposure may occur, and in communities with higher levels of deprivation. Geographical location of UK silicosis cases and associated levels of deprivation has not been described previously.
**Methodology** The Surveillance for Work and Occupationally-Related Disease (SWORD) scheme allows UK chest physicians to report diseases including silicosis. All silicosis cases reported to SWORD between 1996 and the end of 2024 were reviewed. Pseudonymised demographic data (gender, age, first three digits of postcode), reporter information, and diagnosis were reviewed. Geographical location of cases was examined by postcode and by reporting centre, grouped by UK region. Postcode data were mapped to individual lower layer super output area (LSOA) and corresponding index of multiple deprivation (IMD) by decile.
**Results** In total 318 cases of silicosis were reported to SWORD between 1996 and 2024. Mean age was 59.82 (range 24–99), and most (n=305, 96%) were men. Data on postcode were available for 304 cases and for 303 cases on reporting centre. Based on postcode location, cases of silicosis were found in the Midlands (n=87), North West (n=48), Yorkshire and Humber (n=36), Scotland (n=34), North East (n=28), South West (n=13), London (n=11), South East (n=9), East of England (n=6), and Wales (n=4). Reporting centre locations included the Midlands (n=77, 25%), North West (n=56, 18%), South East (n=51, 17%), Yorkshire and Humber (n=42, 14%), Scotland (n=33, 11%), North East (n=27, 9%), South West (n=11, 4%), Wales (n=5, 2%), and East of England (n=1, 1%). Median IMD decile for cases reported ranged from 4–6 (figure 1). Numbers of cases of silicosis were higher in lower IMD decile areas including the Midlands, Yorkshire, and the North West.
**Conclusion** Despite being preventable silicosis continues to occur in the UK. Most cases were from low/middle IMD




**Abstract S110 Figure 1**

deciles and from regions with higher levels of deprivation suggesting silicosis disproportionately affects people with higher deprivation in the UK. More research should focus on the complex factors increasing the risk of disease, and on improved disease prevention, for these groups.

## S111 SARCOIDOSIS AND SILICA: A CASE-CONTROL STUDY OF OCCUPATIONAL EXPOSURES (SANDSTONE STUDY)

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**Background** Sarcoidosis remains a disease of unknown cause, although current hypotheses suggest environmental triggers cause a dysfunctional immune response in genetically predisposed individuals. The role of occupational exposures, particularly respirable crystalline silica (RCS) in pulmonary sarcoidosis (PS) remains unclear, although consistent association is made.

**Aim** To assess if occupational RCS exposure is associated with PS.

**Method** The SANDSTONE study was a multicentre UK-regional (West Midlands) case-control study of men diagnosed with PS since 2000, compared to hospital outpatient controls. Participants completed lifetime occupational histories (LOH) and limited clinical data was collected from medical records. LOH were coded against the UK SOC 2020 and a UK-specific RCS job exposure matrix (SIMBO-JEM) applied. LOH and subsequently identified exposures for cases were limited to jobs prior to diagnosis. Multivariable Firth logistic regression controlled for age at diagnosis, ethnicity, first degree family history of sarcoidosis, smoking, highest educational attendance, rurality, and deprivation.

**Results** A total of 171 cases and 94 controls participated, with median ages of 51 years (IQR 42–61) and 62 years (IQR 53–70), respectively ($p<0.001$). Occupational exposure to RCS was significantly associated with increased odds of pulmonary sarcoidosis, with an adjusted odds ratio (OR) of 1.85 (95% CI 1.01 – 3.39; p-value 0.045). A non-statistically significant trend that RCS exposure within 10 years from first exposure (OR 5.41, 95% CI 0.91–32.04, $p=0.063$) or within 10 years from last RCS exposure (OR 2.61, 95% CI 0.85–8.00, $p=0.093$) was associated with PS.

**Discussion** RCS exposure is associated with PS. Although not statistically significant, exposure within 10 years of diagnosis, whether first or last, was linked to increased odds of PS. The relatively short latency period may suggest a distinct disease process from that observed in silicosis.

## S112 DESCRIPTION OF 32 CASES OF ARTIFICIAL STONE SILICOSIS FROM A SINGLE UK CENTRE

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**Background** The emerging global epidemic of silicosis in artificial stone worktop fabricators has been reported in at least 8 countries, with >1000 cases identified in both Australia and Spain. Cases were first identified in the UK in mid-2023[1] but are under-recognised due to (i) asymptomatic early disease (ii) inadequate health surveillance (iii) lack of awareness by clinicians (iv) misclassification as sarcoidosis. Early diagnosis is important for many reasons including prevention of progressive disease.

**Methods** We retrospectively reviewed medical records of patients with silicosis attributed to artificial stone exposure from our occupational lung disease clinic. We observed that acute/accelerated disease radiological features were not confined to their defined duration of exposure categories, we therefore categorised cases based on three (exclusive) radiological categories (simple, complicated and acute/accelerated).

**Abstract S112 Table 1** Selected demographics and clinical characteristics of 32 patients with artificial stone silicosis

| | | Radiological appearances | | |
|---|---|---|---|---|
| **N (%) unless otherwise stated** | **Total (n=32)** | **Simple silicosis* (n=12)** | **Complicated silicosis** (n=5)** | **Acute/accelerated silicosis*** (n=15)** |
| **Male** | 32 (100%) | 12 (100 %) | 5 (100 %) | 15 (100 %) |
| **Age in years (median (range))** | 39y (23-61) | 38y (26-61) | 48y (27-65) | 33y (23-56) |
| **Born in UK** | 10 (31%) | 6 (50%) | 2 (40%) | 2 (13%) |
| **Smoking status** | | | | |
| **Current** | 9 (28%) | 2 (17%) | - | 7 (47%) |
| **Ex** | 18 (56%) | 7 (58%) | 5 (100%) | 6 (40%) |
| **Never** | 5 (16%) | 3 (25%) | - | 2 (13%) |
| **Duration of exposure in years (median (range))** | 14y (2-53)* | 16y (3-53) | 25y (4-42)* | 13y (2-45) |
| **<10** | 10 (16%) | 3 (25%) | 1 (25%) | 6 (40%) |
| **10-20** | 7 (22%) | 4 (33%) | - | 3 (20%) |
| **>20** | 14 (44%) | 5 (42%) | 3 (75%) | 6 (40%) |
| **Artificial stone exposure only** | 11(34%) | 4 (33%) | 1 (20%) | 6 (40%) |
| **Employment status at referral** | | | | |
| **Employed exposed** | 8 (25%) | 2 (17%) | 2 (40%) | 4 (27%) |
| **Employed unexposed** | 7 (22%) | 4 (33%) | - | 3 (20%) |
| **Unemployed** | 17 (53%) | 6 (50%) | 3 (60%) | 8 (53%) |
| **Bloods** | | | | |
| **Lymphopenia (<1.30 10*9/L)** | 13/28(46%) | 5/10 (50%) | 1/4 (25%) | 7/14 (50%) |
| **Elevated serum ACE (>64 U/l)** | 22/27(81%) | 9/10 (90%) | 2/3 (67%) | 11/14 (79%) |
| **Positive Anti-Nuclear Antibodies** | 9/27 (33%) | 2/10 (20%) | 1/3 (33%) | 7/14 (50%) |
| **Referral route** | | | | |
| **Respiratory** | 19 (59%) | 5 (42%) | 5 (100%) | 9 (60%) |
| **Self** | 11 (34%) | 5 (42%) | - | 6 (40%) |
| **Medicolegal** | 1 (3%) | 1 (8%) | - | - |
| **Occupational Health** | 1 (3%) | 1 (8%) | - | - |

**Key: ACE: angiotensin converting enzyme** * missing n=1

*Simple silicosis defined as presence of pulmonary nodules <10mm diameter

**Complicated silicosis (progressive massive fibrosis) defined by presence of pulmonary nodules with a conglomerate mass ≥10mm

*** Acute/accelerated silicosis include the presence of centrilobular diffuse ground glass nodules

**Results** We identified 32 men with artificial stone silicosis; median age was 39 years (range 23–61). Fifteen had CT features of acute/accelerated silicosis and suggests very high exposures. These patients were younger, more likely to be born outside the UK, to be current smokers and to have shorter exposure histories. Eleven patients had self-referred, contacting our service directly via word-of-mouth or after seeing media articles. Of the 19 referred from respiratory specialists, seven had been treated for sarcoidosis for between 6 months and 5 years prior to referral. Three had been treated for presumed tuberculous based on imaging and two presented with acute autoimmune disease requiring ITU support. A single referral from occupational health had undergone spirometry but no imaging. Prevalence of lymphopenia, elevated serum angiotensin converting enzyme levels and positive antinuclear antibodies were 46%, 81% and 33% respectively. Unemployment in people with silicosis is high even at presentation (50%). (table 1)

**Conclusion** Artificial stone silicosis disproportionally affects a young, vulnerable, migrant workforce often exposed to very high levels of silica. Clinicians should enquire about occupational exposures and be alert to the clinical features which overlap with sarcoidosis and other conditions. The UK health surveillance programme should incorporate early imaging given a significant proportion of cases have onset of disease within 10 years of exposure.

## REFERENCE

1. Feary, *et al. Thorax*. 2024 Sep 18;**79**(10):979–981.

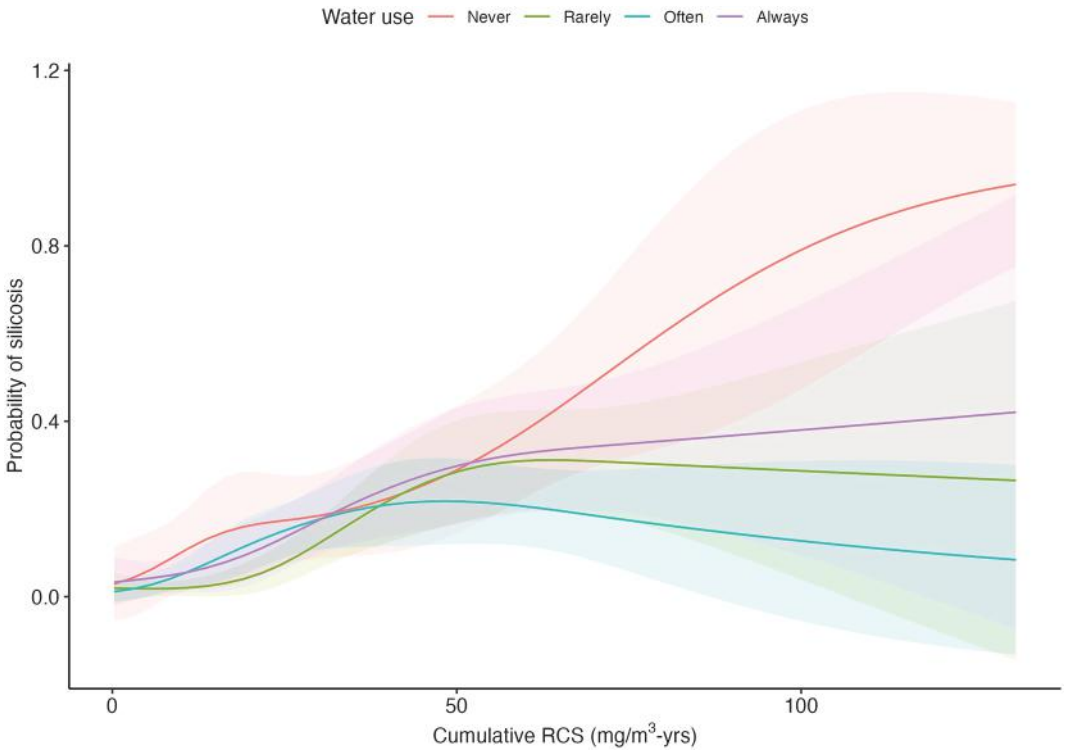
## S113 SILICOSIS PREVALENCE AND ASSOCIATION WITH CUMULATIVE SILICA EXPOSURE AMONG CURRENT AND EX- UNDERGROUND SMALL-SCALE MINERS IN NORTHERN TANZANIA

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**Introduction** High silicosis prevalence has been reported among 50 million small-scale miners globally. We aimed to assess the association of silicosis with cumulative silica exposure in current and ex-small-scale Tanzanite miners and allied workers.

**Methods** All miners attending a screening event (11th-24th March 2024), including a nested random stratified sample of 23 mines, completed a questionnaire and chest Xray (CXR) and were offered testing for TB and HIV. CXRs were read by a blinded, experienced and trained International Labour Organisation (ILO) Classification reader; ≥ 1/0 profusion or large opacities defined silicosis. Previously




**Abstract S113 Figure 1** Predicted probability of ILO ≥ 1/0 silicosis, according to cumulative silica exposure in groups reporting different levels of water suppression (n = 1083)

published exposure measurements were used to assign job titles to high and low silica dust roles. Cumulative silica exposures were then estimated based on duration of current and previous job roles. Inverse probability weights were used to adjust for selection bias. Univariable logistic regression with cumulative silica exposure included as a restricted cubic spline was used to model the risk of silicosis. A sensitivity analysis assess the effect of reported water suppression on silicosis risk.

**Results** Of 1291 current miners, ex-miners and persons who pan mine tailings, 1239 (96%) had reportable CXRs. The unweighted silicosis prevalence among current miners was 126/1083, 11.6% (95% CI 9.7%, 13.5%); weighted prevalence was 13.4% (95% CI 11.1%, 16.0%). Silicosis prevalence among ex-miners was 31/86, (36.0%, 95% CI 25.9%, 46.2%) and among persons who pan mine tailings was 6/77 (7.8%, 95% CI 2.9%, 16.2%). Among all groups, silicosis and more advanced disease were associated with duration and cumulative respirable silica exposure. Among miners and ex-miners with ≥ 20 years work, silicosis prevalence was 62/206 (30%) and 16/20 (80%), respectively. Among current miners, reported use of any water suppression appeared protective when compared to never water suppression, particularly at higher ranges of cumulative silica exposures (figure 1).

**Conclusion** Silicosis prevalence was high among current and ex-miners and was associated with cumulative silica exposures. Silicosis was also observed among workers panning mine tailings. The application of any water appears protective against silicosis. Possible sources of bias include job role and diagnosis misclassification and selection bias.

## S114 LONGITUDINAL CHANGE IN RESPIRATORY FUNCTION IN FIREFIGHTERS; RESULTS FROM THE GRENFELL FIREFIGHTER STUDY

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10.1136/thorax-2025-BTSabstracts.120

**Introduction** The Grenfell Tower fire in 2017 exposed firefighters to unusually high levels of toxic smoke. The long-term effects of smoke particulates have not been extensively studied. Although literature exists surrounding the 9/11 fire and large wildfires, findings of decline in respiratory health are not generalisable to the UK urban setting and require further research. The Grenfell Firefighter Study (GFS) was established to investigate the health risks of firefighting, and specifically the association between occupational fire smoke exposure at the Grenfell Tower fire and longitudinal lung function.

**Methods** 685 firefighters from the London Fire Brigade (LFB) were recruited to the GFS. At a single study visit, spirometry was performed and a symptom questionnaire completed. Historical spirometry records from occupational health were obtained. Exposure was defined by attendance at the Grenfell Tower fire: fire phase (first 23h), recovery phase (day 2–13) or non-attendance. Historical routine spirometry records were obtained from LFB. Piecewise linear mixed models were used to analyse the slopes of absolute forced expiratory volume in

**Abstract S114 Table 1** Spirometry and reported respiratory symptoms by Grenfell Tower fire attendance

| | Grenfell Tower Fire Category | | |
|---|---|---|---|
| | Non-attendees n=124 | Recovery Only n=165 | Fire phase n=332 |
| $FEV_1$ (mean (standard deviation)) | | | |
| Absolute, L | 4.12 (0.62) | 4.01 (0.56) | 3.97 (0.59) |
| % predicted | 100.2 (11.74) | 97.55 (10.93) | 97.46 (11.85) |
| Z score | 0.01 (0.90) | -0.19 (0.83) | -0.20 (0.90) |
| FVC (mean (standard deviation)) | | | |
| Absolute, L | 5.34 (0.81) | 5.23 (0.70) | 5.17 (0.75) |
| % predicted | 103.36 (11.77) | 101.29 (10.52) | 100.84 (11.24) |
| Z score | 0.25 (0.91) | 0.10 (0.82) | 0.06 (0.87) |
| Reported respiratory symptoms <48hrs of the Grenfell Tower fire | - | 16 (9.7%) | 100 (30.1%) |

Key: FEV1: Forced Expiratory Volume in one second; FVC: Forced Vital Capacity

one second ($FEV_1$) before and after the Grenfell Tower fire using splines, adjusting for age and height. Other models also included exposure and the interaction of time and exposure.

**Results** We had usable spirometry from their study visit for 621 male firefighters (mean age 44.8 years, 88% Caucasian, 33.2% ever smokers). Mean $FEV_1$ was 3.97L (SD 0.59), 4.01L (0.56) and 4.12L (0.62) for the fire phase, recovery, and non-attendees respectively. Respiratory symptoms were reported in a dose response manner.(table 1) Preliminary analyses showed annual declines in absolute $FEV_1$ before and after the Grenfell Fire of 20.9 ml (95% CI -24.8, -17.1) and 58.5 ml (95% CI -63.1, -53.8) respectively. Further models explore the role of exposure and a time-exposure interaction.

**Conclusion** Differences in FEV1 at assessment between the groups were clinically small. Initial analyses show a steeper decline in absolute FEV1 after the fire, however further analyses exploring the role of exposure and time-exposure interaction will show if the differences before and after the fire are dependent on the role of the firefighter or reflective of ageing.

## S115 ASBESTOS-RELATED RESPIRATORY DISEASE IN UK ARMED FORCES PERSONNEL: REPORTS FROM THE SWORD SURVEILLANCE SCHEME (1996–2023)

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10.1136/thorax-2025-BTSabstracts.121

**Introduction** The extensive use of asbestos in naval vessels, aircraft, and other UK military infrastructure during the 20th century has resulted in elevated risks of pleural and parenchymal asbestos-related diseases among military personnel. This study aimed to examine the causes and trends of such diseases as reported through a national voluntary reporting scheme.

**Methods** Surveillance of Work-Related and Occupational Respiratory Disease (SWORD) scheme, part of the THOR (The Health and Occupation Reporting) network, collects notifications from UK respiratory physicians on occupational lung diseases to support epidemiological surveillance. We analysed cases where (1) reported agent was asbestos, (2) occupation was linked to the military, and (3) diagnosis included benign pleural disease, mesothelioma, bronchial cancer, or asbestosis.

**Results** There were n=356 cases (1996–2003; estimated actual cases=1,570), n=404 disease episodes (plaques=48% diffuse pleural thickening=13%, mesothelioma=29%, asbestosis=18%, bronchial cancer=5%). Cases peaked 1999–2003 (n=91), declining steadily to n=11 (2020–23). Most cases (89%) were linked to Royal Naval (RN) service. Among 52 RN asbestosis cases, marine engineers, stokers, artificers, and electricians were most frequently affected, though 15% of cases occurred in non-engine room roles (e.g., aircraft maintenance, food preparation, stevedoring, and radio-telecommunications).

**Conclusion** Asbestos respiratory disease remains a concern, though cases have declined significantly, reflecting improvements in exposure control and safety measures. Ongoing surveillance is essential, particularly for veterans in non-engineering roles who have expereinced significant risk.

## S116 BIOLOGICAL THERAPY IN SEVERE OCCUPATIONAL ASTHMA

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10.1136/thorax-2025-BTSabstracts.122

**Background** Occupational asthma (OA) accounts for 16% of adult-onset asthma. OA is overrepresented in severe asthma cohorts, and outcomes in severe OA may be worse than in non- OA particularly where exposures continue. There is limited data on use of biologics in OA, with only one published study on omalizumab. We present real-world data from a single UK centre on biologic use in patients with severe OA.

**Methods** Patients receiving biologic therapy for severe asthma were screened for a diagnosis of OA. OA was diagnosed in a specialist centre with objective evidence of a positive work effect either by serial peak flow analysis and/or specific inhalation challenge. Data collected included demographics, spirometry at diagnosis, biomarkers, causative agent, comorbidities, biologic prescribed, and clinical outcomes. Asthma control was assessed with the Asthma Control Questionnaire.

**Results** Of 335 patients on biologics for severe asthma, 11 (3.3%) had OA. Mean age was 58 years (SD 6.9), and 36% were female (table 1). None had a prior asthma history, and all exhibited a Th2-high phenotype. None were current smokers; 64% were ex-smokers. Median exposure duration to the causative agent was 10 years (IQR 6–18). Causative agents included; isocyanates (n=3), epoxy resins (n=2), cleaning products (n=3), and wheat flour (n=2). Most patients were prescribed Tezepelumab (n=6) and over 60% had trialled at least 1 biologic. Two discontinued biologics (one due to adverse effects, one by choice), both with ongoing poor asthma control, both patients had already been removed from causative agent. Of the remaining patients, symptom control was evenly split between well-controlled and sub-optimally controlled. Two remained exposed to the causative agent (18%), and fewer than half were still employed.

**Conclusion** Biological therapy offers clinical benefit in patients with severe OA when used alongside standard asthma treatment and exposure control. Isocyanate-related OA was associated with persistent symptoms despite cessation of exposure, suggesting a more treatment-resistant phenotype. Over half were unemployed suggesting poor employment outcomes for severe OA. Further research to explore prevalence of OA

**Abstract S116 Table 1** Clinical Characteristics of Patients with Severe Occupational Asthma (n = 11)

| Variable | Value |
|---|---|
| Age, years (mean ± SD) | 58 ± 6.9 |
| Female sex n,% | 4 (36) |
| Smoking status n,% | |
| • Current | 0 (0) |
| • Ex-smoker | 7 (64) |
| • Never | 4 (36) |
| Prior asthma n,% | 0 (0) |
| History of atopy n,% | 8 (73) |
| $FEV_1$, L (mean ± SD) | 2.09 ± 0.44 |
| $FEV_1$,% predicted (mean ± SD) | 66% ± 8.3 |
| Fractional exhaled nitric oxide (FeNO), ppb (mean ± SD) | 71 ± 31 |
| Serum eosinophils, $\times 10^9$/L (median, IQR) | 0.43 (0.30–1.00) |
| Causative agent identified n,% | |
| Cleaning products | 3 (27) |
| Isocyanates | 3 (27) |
| Epoxy | 2 (18) |
| Wheat | 2 (18) |
| Hydraulic fluid | 1 (9) |
| Duration of exposure, years (Median, IQR)) | 10 (6–19.8) |
| Currently exposed to agent n,% | 2 (18) |
| Employment status: employed n,% | 5 (45) |
| Current biological therapy n,% | |
| • Omalizumab | 0 (0) |
| • Mepolizumab | 0 (0) |
| • Benralizumab | 1 (9) |
| • Dupilumab | 1 (9) |
| • Tezepelumab | 6 (55) |
| • None | 2 (18) |
| Prior biological therapy use n% | |
| 0 | 4 (36) |
| 1 | 4 (36) |
| 2 | 2(18) |
| 3 | 1 (9) |
| Asthma control status n,% | |
| • Well-controlled | 4 (36) |
| • Sub-optimal | 4 (36) |
| • Poor | 3 (27) |

among severe asthma and better characterise treatment responses where exposures may continue is important for this underrepresented population.

# New insights in COPD pathogenesis

## S117 BIOMARKERS OF ACCELERATED AGING AND LOW EARLY-LIFE LUNG FUNCTION TRAJECTORY IN THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN (ALSPAC)

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10.1136/thorax-2025-BTSabstracts.123

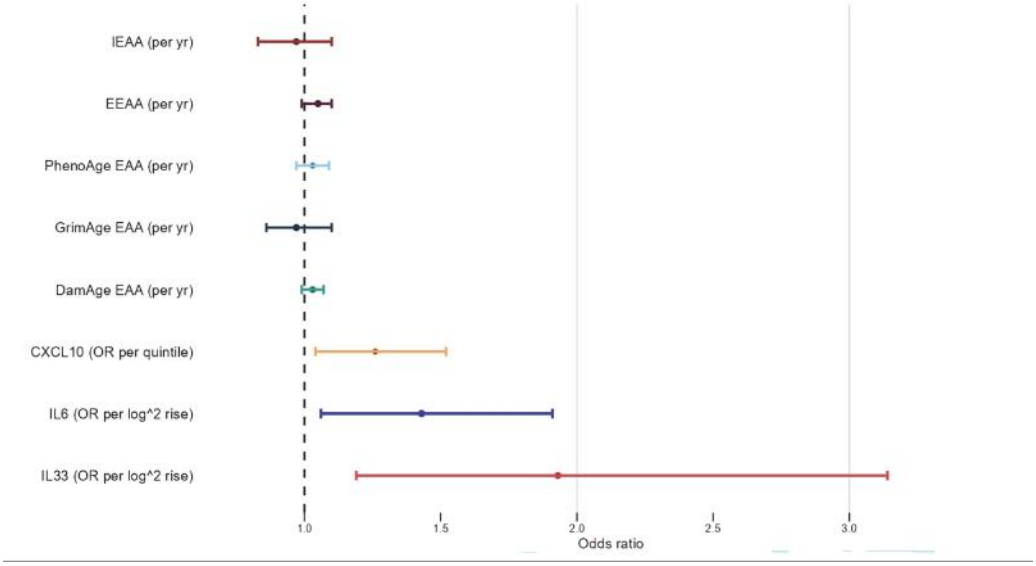
**Introduction** COPD is now recognised as a disease of multifactorial aetiology, with low lung function trajectory (LLFT) in early life a major risk factor. Accelerated aging and cellular senescence may be important to COPD pathogenesis. Epigenetic age acceleration (EAA) and senescence-associated plasma proteins are associated with lower adult lung function and COPD, but little is known of how they relate to high-risk early-life LLFT.

**Aim** To investigate whether childhood EAA and plasma concentrations of senescence-associated proteins are associated with subsequent LLFT into young adulthood.

**Methods** Lung function was measured at ages 8, 15, 24 and 30 years in the Avon Longitudinal Study of Parents and Children. Latent profile analysis was used to derive $FEV_1$ trajectory groups from participants with ≥2 measurements (n=5,232;% predicted for age, sex and height). The final model was selected based on goodness-of-fit and adequate group size.

DNA methylation was quantified at age 7 or 9 (n=1,144) using the Illumina 450 k array. EAA was estimated by five different approaches: Horvath intrinsic EAA (IEAA), Hannum extrinsic EAA (EEAA), DNAm PhenoAge, DNAm GrimAge and DamAge EAA.

Proteomics were assayed from blood plasma at age 9 (n=2,961) using the O-link Target 96 inflammation panel, with 13 senescence-associated proteins analysed as exposures.




**Abstract S117 Figure 1** Odds ratios for persistently low trajectory by EAA and plasma protein concentration

Logistic regression provided the odds ratio of LLFT per year of EAA, or $log^2$ increase in plasma protein concentration, after adjusting for sex and age at blood sampling.

**Results** Five parallel trajectories were identified, with persistently low (n=158; mean FEV1=72.6%) and below average (n=1423; mean FEV1=87.7%) modelled as LLFT and compared to average and above. We did not find evidence of an association between EAA and subsequent LLFT. FDR adjusted associations ($p<0.10$) with persistently low trajectory were evident for plasma concentrations of CXCL10 (OR per quintile 1.26 [1.04–1.52]), IL6 (OR per $log^2$ rise 1.43 [1.06–1.91]) and IL33 (OR per $log^2$ rise 1.93 [1.19–3.14]) (figure 1).

**Conclusions** Childhood EAA was not associated with subsequent LLFT in our sample. Increased plasma concentrations of CXCL10, IL6 and IL33 at age 9 were associated with persistently low trajectory from 8–30 years. These associations will be interrogated for causality with Mendelian Randomisation.

## S118 SPATIAL TRANSCRIPTOMIC PROFILING OF AIRWAY DISEASE IN COPD: INVESTIGATING THE IMPACT OF ANTIVIRAL THERAPY

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10.1136/thorax-2025-BTSabstracts.124

**Background** Chronic obstructive pulmonary disease (COPD) is the third leading cause of global mortality. Therapeutic options that effectively attenuate airway remodelling and disease progression remain limited. Epstein-Barr virus (EBV) has been implicated in immune dysregulation and lymphoid neogenesis in COPD, yet the immunological consequences of antiviral suppression in the airway are poorly defined.

**Aims** To determine whether antiviral therapy alters cellular composition and inflammatory signalling in the bronchial airway microenvironment of COPD patients.

**Methods** Spatial transcriptomics (10x Genomics Visium SD) was performed on paired bronchial biopsies (n=5) collected at baseline and following 8 weeks of treatment from a subset of patients enrolled in the EViSCO trial (NCT03699904), a randomised, placebo-controlled study of valaciclovir (1 g TID). Computational tools including Scanpy, Scanorama, and Cell2-Location were used for cell type deconvolution and differential gene expression analysis.

**Results** Unsupervised clustering identified distinct epithelial and immune populations with region-specific localisation. Enrichment analysis revealed global downregulation of prostaglandin transport and Toll-like receptor (TLR) pathways, indicative of reduced inflammatory signalling, particularly in T and NK cell-enriched regions. At individual patient level, we observed downregulation of genes associated with extracellular matrix components (e.g., collagen trimer, fibrillar matrix), immune activation pathways (e.g., leukocyte migration, chemotaxis), and epithelial remodelling processes (e.g., EMT, cell adhesion, proliferation). These changes suggest a transition from active inflammation and tissue remodelling toward a more quiescent, structurally resolved epithelial state. Reactome pathway analysis additionally showed downregulation of surfactant metabolism and related disease-associated genes post-treatment, potentially reflecting a resolution of inflammation-driven expression, restoring baseline levels of epithelial surfactant genes such as *SFTPA-D*, in alignment with broader suppression of immune and tissue remodelling pathways.

**Conclusions** This study provides the first spatial transcriptomic characterisation of the airway response to EBV-targeted antiviral therapy in COPD. The findings suggest that treatment suppresses key inflammatory and remodelling pathways, offering mechanistic insight into virus-associated airway pathology and its resolution.

## S119 DYSREGULATED INFLAMMATORY, IMMUNE AND FIBRINOLYTIC PATHWAYS ARE ASSOCIATED WITH COPD IN SUBJECTS WITH ALPHA-1 ANTITRYPSIN DEFICIENCY

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10.1136/thorax-2025-BTSabstracts.125

Alpha-1 antitrypsin deficiency (AATD) predisposes individuals to early-onset chronic obstructive pulmonary disease (COPD), yet the molecular differences that distinguish AATD patients who develop COPD from those who do not remain poorly defined. Our aim was to understand protein-level changes which may uncover mechanisms contributing to disease progression in this genetically susceptible population. Protein expression was measured in the serum of subjects with AATD in our Alpha-1 Registry, using the SomaScan® 7 k platform (SomaLogic). Analysis was limited to those with the PiZZ genotype (associated with most severe deficiency) and those without concurrent bronchiectasis. Comparative proteomic analysis was performed between n=98 subjects with a clinical diagnosis of COPD and n=51 without COPD. Log2-fold change was calculated, alongside p value and false discovery rate (FDR).

The top five upregulated proteins in COPD patients were AGER, CILP2, IGDCC4, IGFALS, and tetranectin, while the top five downregulated proteins were PLAT, cathepsin D, TAGLN, LILRA5, and complement component C7. These findings highlight distinct dysregulation in pathways related to chronic inflammation (e.g., AGER), extracellular matrix remodelling (CILP2, tetranectin), and immune regulation (LILRA5, C7). Notably, the downregulation of PLAT and cathepsin D suggests a disruption in fibrinolysis and lysosomal function, potentially impairing normal tissue turnover and immune clearance. Upregulation of AGER further supports a role for oxidative stress and epithelial injury in COPD pathogenesis within the AATD cohort.

Our study identified key protein expression differences between AATD individuals with and without COPD, implicating altered fibrinolytic balance, immune signalling, and inflammatory responses in disease development. These protein-level insights may inform future biomarker development and therapeutic strategies tailored to AATD-related COPD.

## S120 THE ROLE OF FIBROBLAST-DERIVED EXTRACELLULAR VESICLES IN EPITHELIAL CELL SENESCENCE IN COPD

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10.1136/thorax-2025-BTSabstracts.126

**Background** Cellular senescence contributes to accelerated lung ageing observed in COPD. It is characterised by the cessation of cell division, upregulation of cell cycle inhibitors, $p21^{CIP1}$ and $p16^{INK4a}$ and the downregulation of anti-ageing molecule sirtuin (SIRT).[1] Extracellular vesicles (EVs) derived from COPD small airway epithelial cells induce cellular senescence in healthy epithelial cells. This study investigated whether SAF-derived EVs from COPD patients induce senescence in a bronchial epithelial cell line and whether inhibiting EV uptake can attenuate this.

**Methods** EVs were isolated from non-smoker or COPD SAFs and fluorescently labelled with pkh67. Bronchial epithelial cell line BEAS-2B cells were exposed to PKH67-labelled EVs, and EV uptake was quantified by flow cytometry. Senescence-associated gene expression ($p16^{INK4a}$, $p21^{CIP1}$, SIRT1) was measured by qRT-PCR. To examine uptake mechanisms, cells were treated with Dynasore (dynamin inhibitor) or Pitstop (clathrin inhibitor) prior to EV exposure.

**Results** SAF-derived EVs were internalised by BEAS-2B cells in a time-dependent manner, with higher uptake for non-smokers than COPD-derived EVs by approximately 10% for small EVs and 20% for large EVs. COPD-derived EVs induced elevated expression of $p16^{INK4a}$ by 1.2-fold ($n=5$; $p < 0.05$) and $p21^{CIP1}$ by 2-fold ($n=5$; $p < 0.05$) and downregulation of SIRT1 expression from baseline to 0.8 ($n=4$; $p < 0.05$). Dynasore, but not Pitstop, significantly reduced EV uptake which leads to the inhibition of $p21^{CIP1}$ ($n=5$; $p < 0.05$) and $p16^{INK4a}$ ($n=5$; $p < 0.01$) expression induced by COPD EVs.

**Discussion** COPD SAF-derived EVs promote epithelial senescence via dynamin-mediated endocytosis, implicating EVs in fibroblast-epithelial signalling in COPD. The findings are consistent with previous studies demonstrating that EVs from diseased cells can transfer pro-senescent signals to recipient epithelial cells. Blocking EV uptake effectively attenuated this senescent response, suggesting a potential therapeutic strategy to disrupt fibroblast-driven airway remodelling. Further studies are warranted to validate findings in primary epithelial cells.

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2. Wrench CL, Baker JR, Monkley S, Fenwick PS, Murray L, Donnelly LE, *Barnes PJ*. PMID: 38150543

## S121 EFFECT OF A NOVEL SENOTHERAPY, GERALDOL, ON CELLULAR SENESCENCE MARKERS IN EPITHELIAL CELLS

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10.1136/thorax-2025-BTSabstracts.127

**Background** Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous condition associated with accelerated lung ageing. Lifestyle factors such as smoking increase oxidative stress driving emphysema, chronic bronchitis and small airway disease. In COPD, small airway epithelial cells show increased cellular senescence with increased expression of cell-cycle inhibitors p21CIP1 and p16INK4A and downregulation of the anti-ageing protein sirtuin (SIRT).[1] Cellular senescence describes irreversible cell cycle arrest with a senescence-associated secretory phenotype (SASP) involving inflammatory markers such as CXCL8 and senescence-associated β-galactosidase (SA-β gal). Fisetin is a flavonoid with senolytic properties but has limited efficacy due to rapid metabolism and low bioavailability. Geraldol, a methylated metabolite of fisetin, displays improved in-vivo retention. This study compares the efficacy of fisetin and geraldol in a BEAS-2B airway epithelial model of cellular senescence.

**Methods** BEAS-2B cells were cultured with 50nM Doxorubicin on Day 0 and Day 2 to induce cellular senescence.[1] On Day 6, cells were treated 48 hours with 1–30 μM of fisetin, or geraldol. Cells were harvested on Day 8 for cell viability assays, SA-β-galactosidase, western blots and RT-qPCR measurements of p21CIP1, p16INK4A and SIRT1. CXCL8 secretion was quantified using ELISA.

**Results** 50nM Doxorubicin-induced senescence in BEAS-2B cells demonstrated by increased p21CIP1 expression ($p<0.05$) and SA-β gal activity ($n=7$). When these cells were cultured with 10μM geraldol, there was a reduction in p21CIP1, p16INK4A and SIRT1 mRNA ($p<0.05$) ($n=6$). At 30μM, geraldol reduced SA-β-galactosidase activity and CXCL8 release by ~56% and ~59% respectively ($p<0.01$) along with a concentration-dependant reduction in p16INK4A, p21CIP1 and SIRT1 proteins ($n=3$). In contrast, fisetin showed no significant effects on any senescence marker examined.

**Discussion** This study showed that geraldol was more effective than fisetin in reducing markers of doxorubicin-induced senescence in BEAS-2B cells.

**Conclusion** These data suggest that geraldol could be a novel senotherapy for the treatment of COPD or other accelerated ageing conditions. Future directions should explore anti-apoptotic pathways, optimise harvest timings and use primary airway epithelial cells from COPD patients.

### REFERENCE

1. Generating senescent airway epithelial cell populations using low-concentration doxorubicin or etoposide. *European Respiratory Journal* 2021;**58**(suppl 65): PA3687. 10.1183/13993003.congress-2021.PA3687

## S122 OESTROGEN AND ER-β: IMPLICATIONS FOR COPD

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10.1136/thorax-2025-BTSabstracts.128

**Background** Oestrogen plays a regulatory role in tissue homeostasis. Following menopause, oestrogen levels decline, which may reduce protective signalling through oestrogen receptors. In COPD, postmenopausal women experience greater symptom burden, while men display more emphysema (1). Fibroblasts are involved in both inflammation and remodelling. Oestrogen receptor beta (ER-β), encoded by *ESR2*, is expressed in the lung and may influence sex-specific airway pathology (2). We hypothesised that oestrogen modulates inflammatory and matrix-related responses via ER-β in a sex-specific manner.

**Methods** Small airway fibroblasts (SAFs) were isolated from COPD patients undergoing lung resection ($n = 17$; 9 women) and treated with oestrogen ($10^{-12}$ to $10^{-6}$ M), PPT (ER-α agonist), or DPN (ER-β agonist), ± IL-1β. CXCL8 release was measured by ELISA. Expression of oestrogen receptor alpha

(*ESR1*) and oestrogen receptor beta (*ESR2*) mRNA was quantified by RT-qPCR in SAFs. Lung tissue (n = 33; 13 women) was analysed for *ESR1*, *ESR2*, matrix metalloproteinases (*MMP9*, *MMP2*), and collagens (*COL1A1*, *COL3A1*, *COL4A1*). Among women donors, all were postmenopausal and not on hormone therapy; mean age was 71 years (range 59–77) and BMI 25.8 (18.5–29.6). Men had a mean age of 67 years (range 45–81) and BMI 26.4 (21.5–31.5).

**Results** In SAFs, oestrogen reduced IL-1β-induced CXCL8 release in a concentration-dependent manner (maximally at $10^{-9}$ M, p = 0.0077). DPN reduced CXCL8 by 46.9% (p = 0.00047), while PPT had no effect. SAFs expressed approximately twofold higher *ESR2* than *ESR1*. In lung tissue, *ESR2* expression was higher in women. A moderate correlation between *ESR2* and *MMP9* was observed in men (r = 0.42, p = 0.097), but not in women (r = 0.05, p = 0.86). Other matrix-related genes and collagens did not show significant sex-based variation.

**Conclusion** Our results suggest that ER-β is the predominant oestrogen receptor in airway fibroblasts and reduces CXCL8 release. These findings support ER-β as a context-dependent modulator of COPD. Further research is needed to clarify ER-β's regulatory mechanisms relevance to sex-specific airway inflammation and remodelling.

# Patient centred research in respiratory infection: from design to outcomes

## S123 RESEARCH PRIORITIES FOR COMMUNITY ACQUIRED PNEUMONIA IN THE UK: A JAMES LIND ALLIANCE PRIORITY SETTING PARTNERSHIP

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10.1136/thorax-2025-BTSabstracts.129

**Background** Community-acquired pneumonia (CAP) remains a significant global health burden, yet key uncertainties persist regarding its optimal diagnosis, management, and long-term outcomes. The James Lind Alliance Community-Acquired Pneumonia Priority Setting Partnership (CAP PSP) aims to address these critical evidence gaps by identifying and prioritising research questions from the perspectives of patients, carers, and healthcare professionals.

**Methods** The CAP PSP systematically gathered questions from a diverse range of stakeholders via an open survey. Respondents were sought from a wide variety of settings including patient support groups, charities, healthcare colleges, outpatient clinics, hospital inpatients and nursing home residents. Submissions were screened for scope and duplicates, see JLA website for more details on our protocol (https://jla.nihr.ac.uk/node/32856). Subsequently, questions were categorised by theme to identify future research question.

**Results** As of June 2025, a total of 695 research responses were submitted by stakeholders, from which 912 unique questions were identified. Following the robust methodology of the JLA, 856 questions were deemed to be within the scope of the partnership, while 56 were excluded. The respondent profile reflected broad engagement, with contributions predominantly from healthcare professionals (43.7%), patients/persons with CAP (38.1%), and carers/family/friends (14.4%).

Detailed thematic analysis is underway and reflects the diverse concerns of stakeholders. The highest priority themes, based on the volume of questions, include those around i) optimal follow-up, ii) preventing readmission/recurrence, iii) diagnostic methods, and iv) optimising antibiotic/adjunctive treatment strategies. Other prominent themes address comprehensive CAP management in vulnerable populations, recovery and rehabilitation, differentiation from other respiratory infections, and long-term outcomes.

**Next steps** The identified themes will be developed into specific, answerable research questions. These questions will then be presented to stakeholders for ranking, culminating in the identification of the top 10 research priorities for CAP. The top 10 priorities will be presented at the BTS Winter Meeting 2025 and will be widely disseminated to research funders and the broader scientific community to guide future research agendas and improve patient care.

Submitted on behalf of the CAP James Lind Alliance PSP Steering Group

## S124 DEVELOPMENT AND VALIDATION OF CHRONIC PULMONARY ASPERGILLOSIS HEALTH ASSESSMENT QUESTIONNAIRE (CPA-HAQ)

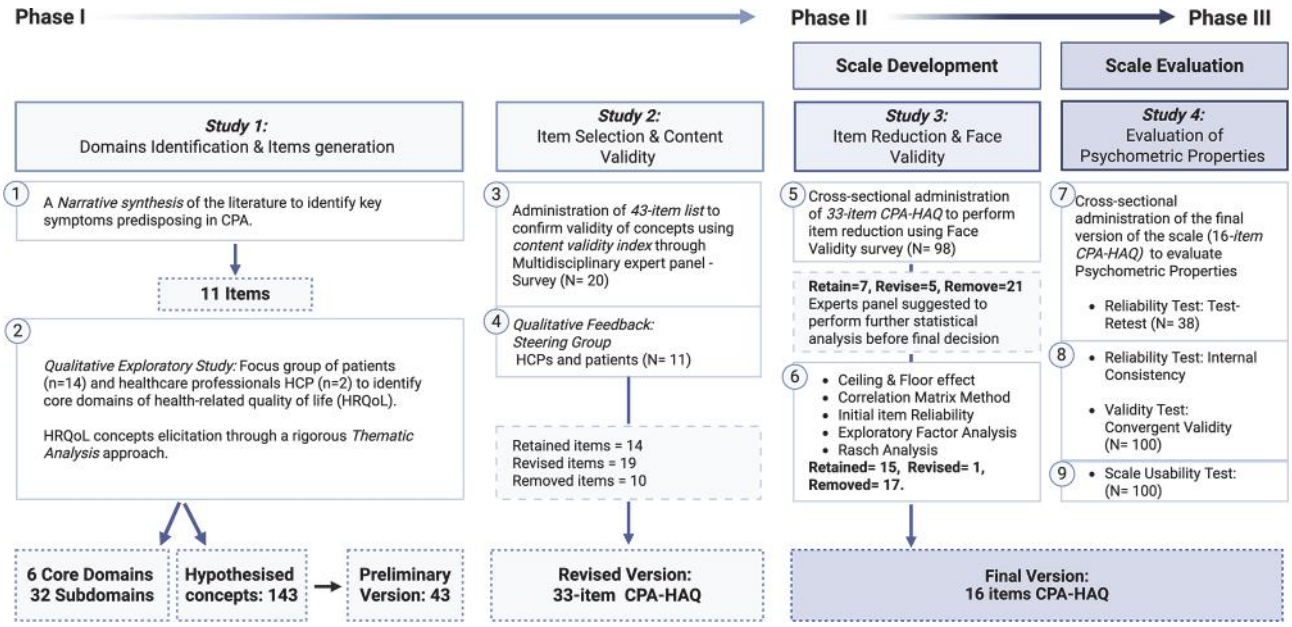
[1]D Marghlani, [2]A Alasfour, [2]E Bradshaw, [2]C Kosmidis, [3]MA McGouldrick, [3]C Deas, [3]D Connell, [4]A Johar, [5]P Lagergren, [5]T Soukup, [1]A Singanayagam, [5]M Zhifang Ni, [6]D Armstrong-James, [6]A Shah. *[1]Centre of Bacterial Resistance Biology, Department of Infectious Disease, Imperial College London, London, UK; [2]National Aspergillosis Centre, Wythenshawe Hospital, Manchester University NHS Foundation Trust, Manchester, UK; [3]Scottish Centre for Respiratory Research, Division of Molecular and Clinical Medicine, University of Dundee, Dundee, UK; [4]Surgical Care Science, Department of Molecular Medicine and Surgery, Karolinska Institute, Stockholm, Sweden; [5]Department of Surgery and Cancer, Imperial College London, London, UK; [6]Royal Brompton and Harefield Hospitals, Guy's and St. Thomas' NHS Foundation Trust, London, UK*

10.1136/thorax-2025-BTSabstracts.130

**Background** Health-Related Quality of Life (HRQoL) is a critical patient-centered outcome in chronic lung diseases, where symptoms are progressive and debilitating with limited treatment options. In Chronic Pulmonary Aspergillosis (CPA), HRQoL can be severely affected by symptom severity, frequent relapses, and side effects of treatments. However, a disease-specific HRQoL tool for CPA has not been developed, making it challenging to identify endpoints for therapeutic trials.

**Aim** To develop and validate the Chronic Pulmonary Aspergillosis Health Assessment Questionnaire (CPA-HAQ), a novel disease-specific HRQoL tool.

**Method** A multi-phase, mixed-method study was designed to develop a reliable HRQoL tool. **Phase 1** (domain identification and item generation) involved a thematic analysis of focus groups and expert consultations to generate 43 items. **Phase 2** (item reduction) involved 98 patients who assessed the relevance, importance, and clarity of these items over the past month using a 5-point scale. A face validity study (n=98 patients and n=20 experts) led to the refinement and elimination of irrelevant items. Exploratory Factor Analysis (EFA) and Rasch analysis were performed on the 33 items to further refine items on the CPA-HAQ.




**Abstract S124 Figure 1**

**Results** The scale-level face validity index (S-FVI/Ave) was 0.67, showing initial moderate validity of the scale, while the universal agreement index (S-FVI/UA) was relatively high at 0.91. EFA showed clean item loadings ($\geq$ 0.40), indicating distinct dimensions. Rasch analysis revealed good fit statistics (infit/outfit MNSQ = 0.70–1.30), confirming most items were well-fitting. Initial Scale reliability is ($\alpha$= 0.86), which indicates good internal consistency among the items in the scale. The final version of CPA-HAQ includes 16 items: sputum, haemoptysis, weight loss, dyspnoea, appetite, fever, chest infection, chest pain, chest wheezing, nocturnal awakening, exacerbation, fatigue, physical activity, psychological burden, and general health.

**Conclusion** The CPA-HAQ was developed with input from patients and experts, including the most relevant and frequent symptoms for CPA patients. A **Phase 3** clinical validation study is currently ongoing in three UK centers (n=100) to further assess its reliability, validity, and usability to extend its use in clinical practice and research settings.

## S125 NAVIGATING THE BOAT TRIAL DEVELOPMENT: A PPIE-LED JOURNEY TO IMPROVE PATIENT PARTICIPATION IN BRONCHIECTASIS RESEARCH

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10.1136/thorax-2025-BTSabstracts.131

**Background** Poor recruitment and retention of patients in clinical trials is an ongoing issue and innovative methods are required to address these issues. The National Institute for Health and Care Research (NIHR) mandates Patient and Public Involvement and Engagement (PPIE) through all stages of the grant application and trial delivery process. This was incorporated in the development of a Study Within a Trial (SWAT) for the BOAT (Bronchiectasis Optimizing Azithromycin Therapy) trial. This trial compares continuing vs stopping azithromycin therapy in patients with non-cystic fibrosis bronchiectasis. The SWAT was developed to reduce disparities in patient participation from 'seldom heard groups' using a recruitment video translated into four languages.

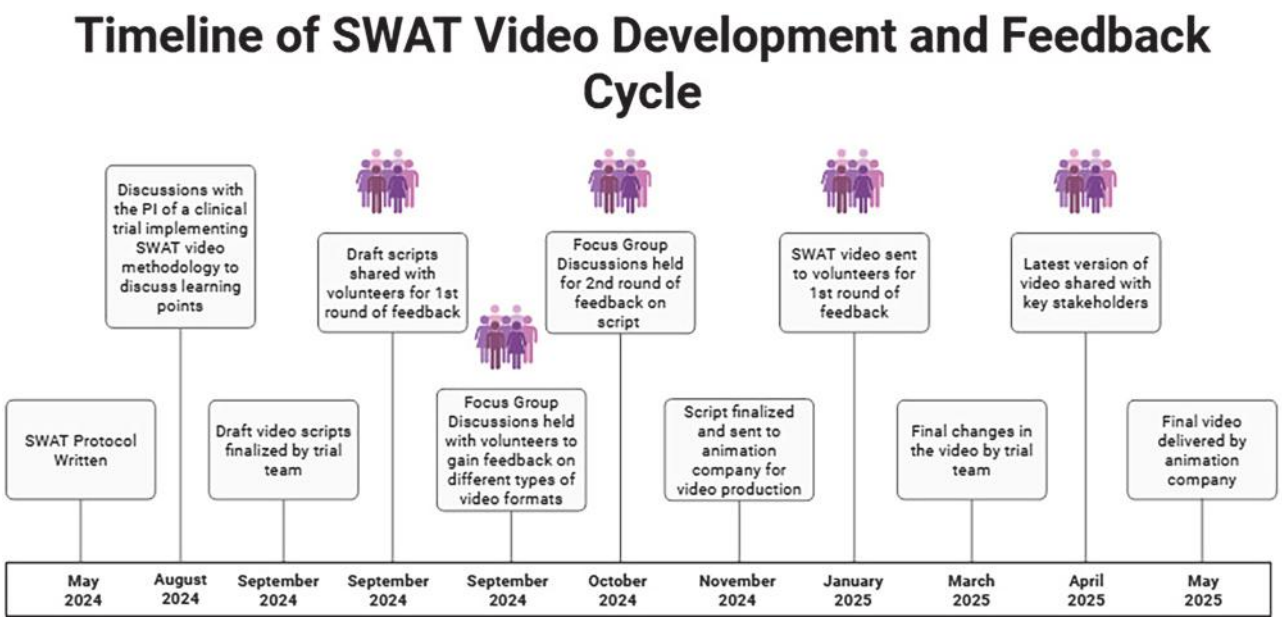
**Methods** A PPIE approach was used during the grant application and video development process. The discussions were led by 'Asthma and Lung UK' foundation with an aim to identify the visuals, style and narration preferred by patients to create an accessible and engaging video. In addition, we aimed to ensure the video will effectively explain the trial to potential participants and motivate them to take part in it. Volunteers received 3 video examples from other clinical trials, 4 different versions of the trial script and questions to prompt feedback/ideas.

**Results** 8 volunteers with bronchiectasis (age range 42- 80 years) of varying ethnicity (5 Caucasians, 2 Asian/Asian British and 1 British/Black/African/Caribbean) participated in the sessions. 3 volunteers had hearing impairment.

The majority of participants had taken azithromycin at some point for an infection, but the duration of their treatment did vary. Some patients had taken it as a long-term prophylactic while others for acute infection.

A focus group was conducted with 4 volunteers, followed by 2 individual interviews and 2 written feedback. The approach used during the PPIE process is shown in figure 1.

**Conclusion** Patients are centre to clinical trials and provide important feedback essential for effective trial development. The focus groups and use of a charity organisation allowed a




**Abstract S125 Figure 1**

broad consensus and permits robust critique of initial drafts. Future clinical trials should adopt this approach more frequency to address retention and recruitment issues.

## S126 INTRODUCING AND EVALUATING THE UK'S FIRST MEDICINE-FOCUSSED PATIENT INFORMATION RESOURCES FOR NON-TUBERCULOUS MYCOBACTERIAL (NTM) DISEASE: WHAT DO WE FIND?

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10.1136/thorax-2025-BTSabstracts.132

**Background** Non-tuberculous mycobacterial (NTM) disease is rare, and treatment is prolonged, often requiring complex drug regimens. The NTM Network UK aims to improve the quality of care for NTM patients through collaborative work across multidisciplinary healthcare professionals (HCPs). Recognising the lack of relevant treatment-support materials, a work stream was established to create two resources; *Treatment of NTM Tips and Information* and *Individual Antibiotic Medicine Information Leaflets*, published August 2023 via NTM Patient Care UK and NTM Network UK.[1] Our aim was to evaluate the access and use of these resources by patients and HCPs.

**Methods** We analysed download data from the NTM Patient Care UK website and reviewed printed orders requested between March 2024 and March 2025. During May 2025, NTM Network UK and NTM Patient Care UK members were invited to participate in anonymised feedback questionnaires via Microsoft Forms.

**Results** There was significant engagement with the resources; **2,319 views online** and **125 printed copies requested by HCPs. Survey responses were received from 23 patients and 45 HCPs (doctors, nurses, pharmacists, dietitians and physiotherapists).** All patients were currently/previously prescribed NTM treatment or actively discussing treatment options. Both HCP and patient groups found the information provided in the resources fulfilled their purpose, in particular the sections on managing medicines and side effects (table 1). 44/45 HCPs would recommend the resources to patients. Ease of access to the resources were reported by an average of 65% and 59% of HCPs and patients respectively. Identified barriers included difficulties in accessing online and clinic time constraints. The majority (65%) of patients independently identified the resources through the website.[1] Only 5 patients were directed by their HCPs. HCPs preferred printed copies 42% (19/45) vs 7% (2/23) of patients.

**Conclusion** These resources have had considerable use, with the feedback received overwhelmingly positive. They support the national NTM Standards of Care, and we plan to increase

**Abstract S126 Table 1** Breakdown of responses from HCPs and patients

| Survey Question Options | HCPs (45 responses) | Patients (23 responses) |
|---|---|---|
| What do you find useful about the *Treatment of NTM Tips and Information Booklet*? (multiple responses allowed) | | |
| Easy to access | 31 (68%) | 14 (61%) |
| General information provided about treatments | 38 (84%) | 17 (74%) |
| Top tips about managing NTM medicines | 34 (76%) | 14 (61%) |
| Top tips about managing the side effects | 34 (76%) | 16 (70%) |
| Further information inc. lifestyle, travel & costs | 14 (31%) | 6 (26%) |
| What do you find useful about the *Individual Antibiotic Medicine Information Leaflets*? (multiple responses allowed) | | |
| Easy to access | 28 (62%) | 13 (57%) |
| How to take the antibiotics | 33 (73%) | 9 (39%) |
| What patients need to look out for | 32 (71%) | 14 (61%) |
| Side effects section | 38 (84%) | 17 (74%) |
| Expected monitoring section | 27 (60%) | 11 (48%) |

their uptake amongst patients and HCPs, by improving their visibility and ease of access.

## REFERENCE

1. NTM Patient Care UK. Coping with the medicines. *NTM Patient Care* UK. 2023. https://www.ntmpatientcare.uk/coping-with-the-medicines

## S127 EXPLORING THE EFFECT OF DIFFERENT RESPIRATORY DISEASES ON PATIENTS' PERSPECTIVE OF ANTIMICROBIAL RESISTANCE: AN AMR-LUNG/ EUROPEAN LUNG FOUNDATION GLOBAL PATIENT SURVEY

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10.1136/thorax-2025-BTSabstracts.133

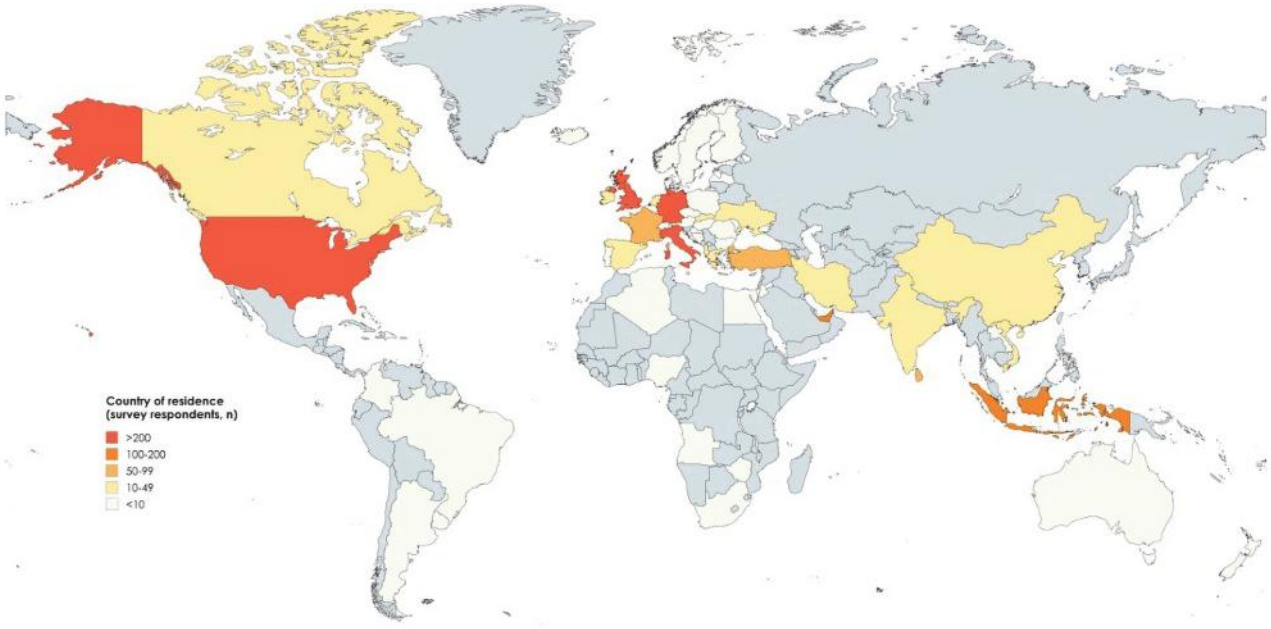
**Introduction** Antimicrobial resistance (AMR) is a global health threat for people with chronic respiratory disease (CRD). It is crucial to understand what such individuals understand by AMR and how they feel it affects them.

**Aim** To further understand the burden of AMR from the perspective of people with CRD and identify any differences dependent on primary respiratory diagnosis.

**Methods** An online, multilingual, anonymous patient survey was developed by the AMR-Lung ERS Clinical Research Collaboration and European Lung Foundation patient advisory groups. Multivariable logistic regression models were used to analyse disease-specific burden and factors affecting AMR perception across different respiratory diseases.

**Results** Among 1,801 respondents from 57 countries globally (figure 1), 31% recalled having an AMR-associated infection. Commonly self-reported diseases included bronchiectasis (40.8%, n=735), Cystic Fibrosis (CF) (27.2%, n=489), asthma (25.3%, n=455), and COPD (14%, n=252). Patients who reported diagnosis of longer history CRD (>5 years) were significantly more likely to report AMR-associated infection (OR=1.37 [95% CI 1.01–1.86, p=0.041], as well as those with genetic respiratory diseases such as CF or primary ciliary dyskinesia (OR=2.77 [95% CI 2.01–3.80, p<0.001). Patients with airways diseases were significantly less likely to report prior knowledge of the concept of AMR (OR=0.68 [95% CI 0.47–0.97, p=0.035). Patients with bronchiectasis were significantly more likely to report a perceived lack of antimicrobial effectiveness (OR=1.67 [95% CI 1.07–2.60], p=0.024), use of antimicrobials without medical prescription (OR=1.59 [95% CI 1.10–2.29], p=0.014), and perceiving AMR as a personal threat (OR=2.68 [95% CI 1.64–4.39], p<0.001).

**Conclusions** This global survey is the first to suggest a high AMR burden and threat from the perspective of people with CRD, with disparities across different diseases highlighting the need for targeted education strategies for different patient groups.




**Abstract S127 Figure 1** Respondents' country of residence with colours representing the number of respondents. Total number of valid responses=1799. Created with mapchart.net

## S128 THE ASSOCIATION BETWEEN PATIENT-REPORTED OUTCOMES AND PULMONARY EXACERBATION FREQUENCY IN PATIENTS WITH ALPHA-1 ANTITRYPSIN DEFICIENCY

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10.1136/thorax-2025-BTSabstracts.134

**Introduction** The detrimental impact of pulmonary exacerbations on lung function in Alpha-1 Antitrypsin Deficiency (AATD) is well established. However, limited research exists on the relationship between patient-reported outcomes (PROs) and exacerbation frequency. Exploring this link is important, as it integrates patient experience with clinical indicators, supports early intervention, and connects subjective symptom reporting with objective clinical deterioration in this rare chronic disease.

**Study Aim** To examine the association between exacerbation frequency and patient-reported outcomes in individuals with alpha-1 antitrypsin deficiency and assess the strength of these associations in relation to lung function (FEV1%).

**Method** Data for patients with PiZZ AATD was extracted from a cohort/registry held at Queen Elizabeth Hospital Birmingham. Patients were categorized, based on their self-reported exacerbation frequency over the previous 12 months, into frequent exacerbators FE (≥2), infrequent exacerbators IE (<2), and non-exacerbators (0). PROs were measured using the St. George's Respiratory Questionnaire (SGRQ), the COPD Assessment Test (CAT), and the Modified Medical Research Council (mMRC) Dyspnea Scale. Associations were analyzed using non-parametric tests, Spearman correlation, and multivariable linear regression to control for confounders such as smoking history, COPD diagnosis, and FEV1%.

**Results** A total of 234 PiZZ patients were included in this study. The FE group (n=69) reported significantly poorer PROs when compared to the IE group (n=165), even though FEV1% was not significantly different between the two groups (p = 0.055). When comparing exacerbators (both FE and IE) to non-exacerbators, all PROs and FEV1% were significantly different with poorer results seen in the exacerbators group. Spearman rank correlation supported that lower FEV1% and more exacerbations are significantly correlated with poorer PROs (p ≤ 0.001 in all correlations). Multiple regression analysis confirmed that both exacerbation frequency and FEV1% independently predicted PRO scores, with FEV1% showing stronger correlations with activity and dyspnea severity.

**Conclusion** Exacerbation frequency and impaired lung function are associated with poorer PROs in individuals with AATD. These findings support the clinical relevance of integrating PROs into routine assessment for AATD patients, especially those at risk of frequent exacerbations. Routine PRO monitoring may facilitate early intervention, improve patient-cantered care and quality-of-life outcomes.

**Abstract S128 Table 1** Demographic characteristics and clinical parameters of all patients with association between exacerbation frequency groups, demographic, and clinical characteristics

| Variables | Total n=234 | FE 69 (29.49) | IE 165 (70.51) | p-value[#] |
|---|---|---|---|---|
| **Age** | 53 (45–62) | 51 (42–60) | 55 (46–63) | 0.051[a] |
| **Gender** | | | | |
| Male | 129 (55.13) | 32 (43.68) | 97 (58.79) | 0.082[b] |
| Female | 105 (44.87) | 37 (53.62) | 68 (41.21) | |
| **Pack-year** | 8 (0–19) | 10 (0–19) | 8 (0–19.50) | 0.380[a] |
| **Smoking status** | | | | |
| Current smoker | 16 (6.84) | 4 (5.80) | 12 (7.27) | 0.386[b] |
| Never smoking | 78 (33.33) | 19 (27.54) | 59 (35.76) | |
| Former smoking | 140 (59.83) | 46 (66.67) | 94 (56.97) | |
| **Genotype ZZ** | 196 (83.76) | 57 (82.61) | 139 (84.24) | 0.757[b] |
| **Genotype SZ** | 38 (16.24) | 12 (17.39) | 26 (15.76) | |
| **COPD diagnosis** | | | | |
| Yes | 172 (73.50) | 52 (75.36) | 120 (72.73) | 0.747[b] |
| No | 62 (26.50) | 17 (24.64) | 45 (27.27) | |
| **FEV1%** | 58.50 (39.38–91) | 50.47 (31.93–83.00) | 60 (43.51–93.85) | 0.057[a] |
| **CAT** | 20 (2–26) | 22 (15.50–29.00) | 19 (11.00–25.00) | 0.010[a]* |
| **SGRQ** | 62.25 (45.30–78.50) | 70.30 (56.12–88.45) | 59.80 (36.50–73.70) | <0.001[a]* |
| **Symptoms** | | | | |
| **SGRQ Activity** | 66.19 (41.31–92.51) | 84.98 (47.69-92.51) | 60.35 (35.36–91.97) | 0.016[a]* |
| **SGRQ Impact** | 30.82 (17.39–52.47) | 45.04 (21.25–63.19) | 25.98 (13.38–47.66) | 0.001[a]* |
| **SGRQ Total** | 47.38 (29.42–67.26) | 59.64 (37.29–74.39) | 42.18 (25.13–63.57) | <0.001[a]* |
| **mMRC 0** | 70 (29.91) | 16 (23.19) | 54 (32.73) | 0.037[b]* |
| **mMRC 1** | 57 (24.36) | 15 (27.14) | 42 (25.45) | |
| **mMRC 2** | 50 (21.37) | 12 (17.39) | 38 (23.03) | |
| **mMRC 3** | 39 (16.67) | 19 (27.54) | 20 (12.12) | |
| **mMRC 4** | 18 (7.69) | 7 (10.14) | 11 (6.67) | |

Parameters are reports as median (IQR) or n (%) ; a: Mann Whitney U test; b: Chi-square or Fisher exact test; *-Significant p value < 0.05; #-comparison reported between FE and IE. IQR: Inter quartile range; FE: Frequent exacerbators; IE: infrequent exacerbators; SGRQ: St George's Respiratory Questionnaire; COPD: chronic obstructive pulmonary disease; FEV1%: forced expiratory volume in one second; CAT: COPD Assessment Test; mMRC: Modified Medical Research Council Dyspnea Scale

# Asthma – remission and beyond

## S129 LONG TERM CLINICAL REMISSION ON BIOLOGICS: AN ANALYSIS OF REAL-WORLD DATA FROM THE UK SEVERE ASTHMA REGISTRY

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10.1136/thorax-2025-BTSabstracts.135

**Introduction** Clinical remission on biologics is an achievable goal for patients with severe asthma (SA). Most reports present short term follow-up, include small cohort sizes or present data on patients included in clinical trials. This study evaluates clinical remission rates in a real-world cohort of patients over the course of up to 4 years on different biologics and include those who switched biologic during this period. We also assess barriers and predictors of remission.

**Methods** Retrospective study of 525 patients in the UK SA registry who were initiated on a biologic between January 2015 and May 2022. Clinical remission was assessed at two time points: first review (9–24 months) and long-term review (20–48 months) and defined as controlled asthma (ACQ6<1.5), no exacerbations in the preceding 12 months and no maintenance oral corticosteroid use (mOCS). Logistic regression and multivariate models were used to identify predictors of remission.

**Results** At baseline, patients had high symptom burden (ACQ6 score 3.0 [2.2, 3.8]), high exacerbation burden (median 5 exacerbations [IQR 4, 8] in previous year) and mOCS use (49.9% of patients). Clinical remission was achieved in 25.1% of patients at first review (13.3 [IQR 11.9, 17.1] months), increasing to 32.1% at long-term review (36.1 [IQR 33.9, 38.9] months). Higher asthma symptom burden and presence of anxiety/depression at baseline were negatively associated with achieving long-term remission. Older age at baseline and the presence of nasal polyps increased likelihood of long-term remission. Among those in remission at first review, 69.7% remained in remission at long-term review while 45.6% of those in long-term remission had not been in remission at first review (figure 1). Baseline characteristics predictive of this latter transition included elevated blood eosinophils, absence of obesity, lower symptom burden and non-reliance on mOCS. Of the 525 patients, 412 (78.5%) remained on the same biologic until long-term review. Among the 103 patients who switched biologic, 14 (13.6%) achieved remission.

**Conclusion** In this large real-world cohort of patients with SA, there is a progressive increase in clinical remission rates over time, which is influenced by the presence of comorbidities but is largely independent of biologic switching.

## S130 ASSOCIATION BETWEEN HRT USE AND ASTHMA ATTACKS: A UK POPULATION-BASED COHORT

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10.1136/thorax-2025-BTSabstracts.136

**Background** Asthma exhibits a marked sex-based disparity suggesting sex hormones may influence asthma outcomes. To investigate this, we explored the relationship between exogenous sex hormones and asthma exacerbations using population-level data. Previously, we found the progesterone-only oral contraceptive pill was associated with an increased risk of exacerbations, only in women <35 years old. Here we focus on hormone replacement therapy (HRT). Only one study has previously investigated HRT; they found only previous HRT use – not current - was associated with exacerbations. There was no clear biological explanation for this unexpected finding.

**Methods** We used UK electronic health records to draw a cohort of women with asthma aged 45–60, comparing HRT initiators to non-users. Women were censored at two years or end of follow-up; HRT users were also censored upon stopping HRT. We used inverse-probability of treatment weighting and Cox proportional hazards to estimate associations with exacerbations. Covariates included age, BMI, asthma severity (exacerbation history, inhaler use), comorbidities (e.g., atopy, depression), smoking, and socioeconomic status. Interaction analyses assessed effect modification by HRT type, BMI, and type 2 inflammation (blood eosinophils).

**Results** A total of 182,010 women were included, of whom 9,663 (5.1%) used HRT and 172,347 did not. Among HRT users, 51.5% were prescribed combined HRT, 46.3% oestrogen-only, and 2.2% progesterone-only. In the overall cohort, 15% only used reliever inhalers, 47% used inhaled corticosteroids, and 35% used combination inhalers. Additionally, 23% experienced an exacerbation in the year prior to inclusion.

HRT use was not associated with asthma exacerbations (weighted-HR=0.97, 95%CI 0.90–1.09, p=0.441). The association was not modified by HRT type, BMI or type 2 inflammation (all interaction p-values>0.05).

**Conclusion** We found no association between HRT and asthma exacerbations, regardless of HRT type, BMI or type-2 inflammation. These findings differ from the previous HRT study, which may have been affected by temporality bias. However, our findings appear to align with those from our oral contraceptive study. Taken together, we hypothesis that the addition of exogenous sex hormone – specifically progesterone – may only be associated with asthma exacerbations in younger women with higher endogenous progesterone levels. This warrants further investigation.

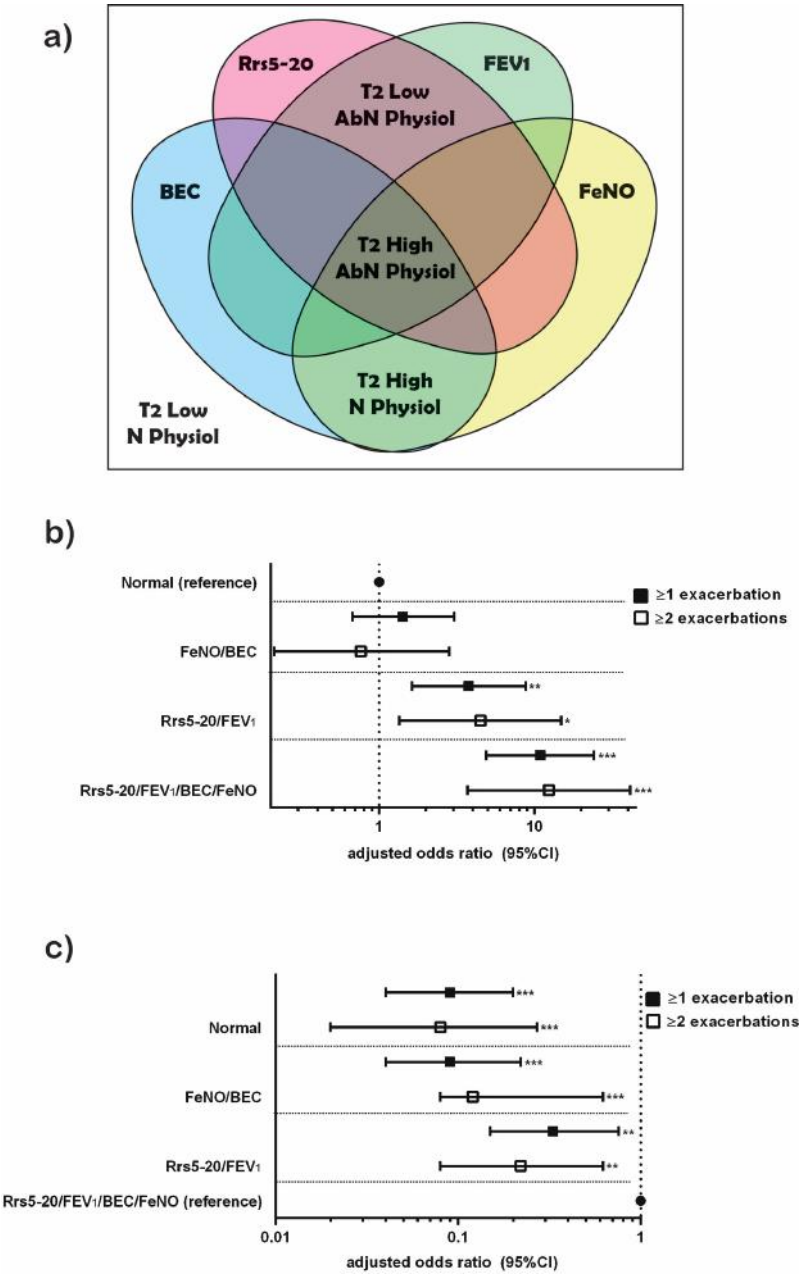
## S131 AIRWAY PHYSIOLOGY IS A STRONGER CONTRIBUTOR TO ASTHMA EXACERBATION RISK THAN TYPE 2 INFLAMMATION

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10.1136/thorax-2025-BTSabstracts.137

**Introduction** Recently data has demonstrated an association between blood eosinophil counts (BEC) and fractional exhaled nitric oxide (FeNO) levels and exacerbation risk, reinforcing the central role of T2 inflammation in asthma pathogenesis. Two point-of-care tests, airway oscillometry and spirometry, have been shown to synergistically increase the risk of poorer asthma control. Oscillometry can assess small airway resistance by the resistance heterogeneity between 5Hz and 20Hz (Rrs5–20).

**Methods** The Oscillometry Asthma Registry (OAR) included adults diagnosed with asthma according to GINA. Data were retrospectively collected from two clinical sites: Ninewells Teaching Hospital in Dundee, UK, and the Allergy and Pneumology Outpatient Clinic in Bergamo, Italy. Four representative phenotypic groups were compared: (a) Group 1 – normal phenotype where Rrs5–20<0.10 kPa/L/s, $FEV_1 \geq 80\%$, BEC<300 cells/μL and FeNO<25ppb; (b) Group 2 – T2 high only phenotype where Rrs5–20<0.10 kPa/L/s, $FEV_1 \geq 80\%$, BEC≥300 cells/μL and FeNO≥25ppb; (c) Group 3 – abnormal physiology only phenotype where Rrs5–20≥0.10 kPa/L/s, $FEV_1 < 80\%$, BEC<300 cells/μL and FeNO<25ppb; and (d) Group 4 – abnormal immuno-physiological phenotype where




**Abstract S131 Figure 1**

Rrs5–20≥0.10 kPa/L/s, $FEV_1$<80%, BEC≥300 cells/μL and FeNO≥25ppb (figure 1a).

**Results** 937 patients with mild to severe asthma were included. The adjusted odds ratio (aOR) [95% CI] for experiencing ≥1 and ≥2 exacerbations in Group 3 compared to Group 1 (reference group) was 3.77 (1.62,8.79) p<0.01, and 4.48 (1.35,14.85) p<0.05, respectively (figure 1b). For Group 4 (abnormal physiology, T2-high), the aORs were substantially higher: 10.89 (4.89,24.25) p<0.001 for ≥1 exacerbation, and 12.39 (3.70,41.43) p<0.001 for ≥2 exacerbations, relative to Group 1 (figure 1b). This analysis was repeated using Group 4 as the reference group (figure 1c).

**Conclusion** Future research should aim to prospectively incorporate small airways dysfunction alongside symptom burden, type 2 biomarkers and spirometry into a comprehensive longitudinal tool for predicting asthma exacerbation risk.

## S132 ACHIEVING ASTHMA REMISSION WITH EXTRAFINE BECLOMETHASONE DIPROPIONATE, FORMOTEROL FUMARATE, AND GLYCOPYRRONIUM BROMIDE (BDP/FF/GB) PMDI IN ADULTS WITH MODERATE-TO-SEVERE ASTHMA: A POST-HOC ANALYSIS OF THE TRIMARAN AND TRIGGER STUDIES

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10.1136/thorax-2025-BTSabstracts.138

**Rationale** Remission in asthma is generally agreed to be a conjugate of sustained symptom control, absent exacerbations, no

systemic corticosteroid use and stable lung function. The impact of triple inhaled therapy versus dual therapy for achieving asthma remission is unknown.

**Methods** We conducted a *post-hoc* analysis of the TRIMARAN (Triple versus medium strength of ICS/LABA) and TRIGGER (Triple versus high strength ICS/LABA) studies [Virchow JC et al, Lancet 2019] to assess the proportion of study participants who met the following criteria of remission on treatment: no severe asthma exacerbations and systemic corticosteroids over 52 weeks, ACQ-5 score < 1.5 at Week 26, 40 and 52, and change from baseline in trough $FEV_1 \geq 100$ mL at Week 52 *OR* a 2hr post-dose $FEV_1$/FVC ratio > 0.7 at Week 52. Odds ratios (OR; 95% CIs) were calculated from a logistic regression model.

**Results** In TRIMARAN, 174 (30.2%) and 147 (25.6%) patients met all remission criteria while on treatment with medium dose BDP/FF/GB (400/24/50 mcg/day) and BDP/FF (400/24 mcg/day), respectively (OR=1.268; 95% CI (0.976, 1.647), p=0.075). In TRIGGER, 165 (28.9%) and 107 (18.7%) patients met the criteria while on high-dose BDP/FF/GB (800/24/50 mcg/day) and BDP/FF (800/24 mcg/day), respectively (OR=1.776; 95% CI (1.344, 2.346), p<0.001).

**Conclusions** A greater proportion of patients achieved clinical remission while on treatment with BDP/FF/GB triple therapy than with BDP/FF. The difference was particularly noted in patients whose asthma was previously uncontrolled on high-dose ICS/LABA treatment who were stepped-up to high-dose BDP/FF/GB.

## S133 IMPROVEMENTS IN FEV1 AND ST GEORGE'S RESPIRATORY QUESTIONNAIRE (SGRQ) AT 4 AND 16 WEEKS ON BENRALIZUMAB ARE STRONGLY ASSOCIATED WITH CLINICAL REMISSION AT 1 YEAR

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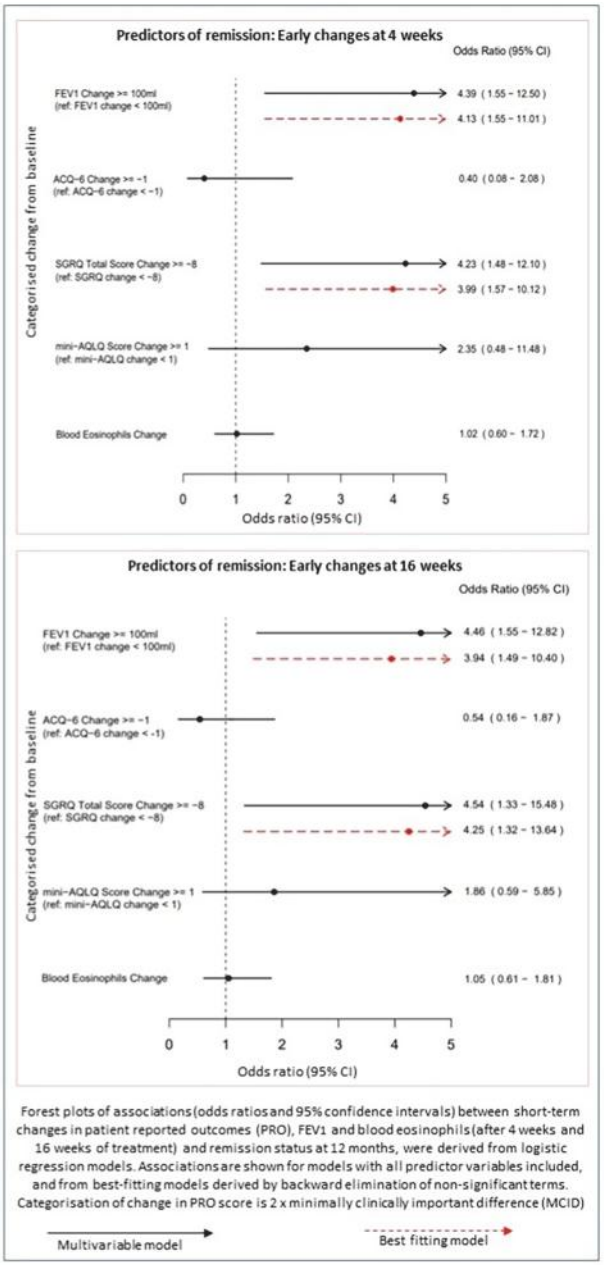
**Introduction** Remission is now an aspiration for asthma patients on biologics, although there is no agreed definition. Predicting the likelihood of remission at an early stage on treatment would improve precision therapy.

**Aims** To identify the characteristics at 4 and 16 weeks on treatment associated with remission at 1 year in a real-world asthma cohort on benralizumab.

**Methods** BenRex is a multicentre study of exacerbations on benralizumab for severe eosinophilic asthma. We defined remission at 1 year as meeting all of: no OCS for asthma (for maintenance/exacerbation); ACQ <1.5 and $FEV_1 \geq 80\%$ predicted and/or $\geq 10\%$ higher than baseline $FEV_1$% predicted. Forest plots of associations between early changes at 4 and 16 weeks and remission at 1 year were derived from logistic regression models.

**Results** 36 of 136 subjects (26.5%) met remission criteria. Subjects achieving remission were older (p=0.04). Male and female participants achieved remission at similar rates (p=0.84). Figure 1 shows predictors of remission using changes from baseline at 4 and 16 weeks. $FEV_1$ and SGRQ improvement are strongly associated with subsequent remission.

**Conclusion** An improvement in $FEV_1$ of ≥100 ml and SGRQ change of double the MCID at 4 and 16 weeks on benralizumab are strongly associated with clinical remission at one year.




**Abstract S133 Figure 1** Early change predictors of clinical remission on benralizumab

## S134 DOMINANT CLINICAL IMPROVEMENTS AND DURABILITY OF BIOLOGIC RESPONSE IN SEVERE ASTHMA

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10.1136/thorax-2025-BTSabstracts.140

**Background** Biologic therapies have transformed severe asthma care, but the real-world evolution of multidimensional clinical outcomes—including symptom control, exacerbations, steroid use, and hospital admissions—remains incompletely characterised, especially in relation to biomarker profiles and broad clinical response patterns.

**Methods** We conducted a comprehensive longitudinal cohort study of 215 adults with severe asthma treated with biologics in the Portsmouth Severe Asthma Service (2015–2024). Patients were assessed annually for symptom control (Asthma Control Questionnaire, ACQ), exacerbation frequency (oral corticosteroid-requiring events), lung function ($FEV_1$), maintenance oral steroid use (mOCS), and hospital admissions through Year 3. Clinically meaningful responses were defined as: ACQ improvement $\geq$0.5, OCS burst reduction $\geq$50%, $FEV_1$ increase $\geq$200 mL, hospital admission reduction by $\geq$1, mOCS cessation, and $\geq$50% mOCS dose reduction. Cumulative and incremental responder rates were quantified using Kaplan-Meier-style plots, including multi-domain (quadruple- and quintuple-domain) response and time-to-benefit. Baseline demographics, biomarkers (eosinophils, FeNO), and treatment factors were compared between quadruple-domain responders and non-responders.

**Results** By Year 3, cumulative responder rates were: OCS burst reduction 94.0%, mOCS cessation 76.1% (n=67), ACQ improvement 68.8%, $FEV_1$ improvement 55.8%, hospital admission reduction 60.9% (in those at risk), and $\geq$50% mOCS dose reduction (not weaned) 11.9%. Most benefit accrued in Year 1, but a substantial minority achieved late multi-domain response by Years 2 and 3. Quadruple-domain response (ACQ+OCS+$FEV_1$+admission reduction) increased from 18.0% to 38.7% for benralizumab, 27.3% to 36.4% for dupilumab, 24.2% to 40.9% for mepolizumab, and 11.1% to 33.3% for omalizumab from Year 1 to Year 3. Baseline comparisons revealed no significant differences in eosinophils, FeNO, ICS dose, lung function, adherence, or smoking between quadruple-domain responders (n=87) and quadruple non-responders (n=132); only maintenance oral corticosteroid use was less common in quadruple-domain responders (p=0.005). The figure illustrates sequential accrual of benefit across all domains and therapies.

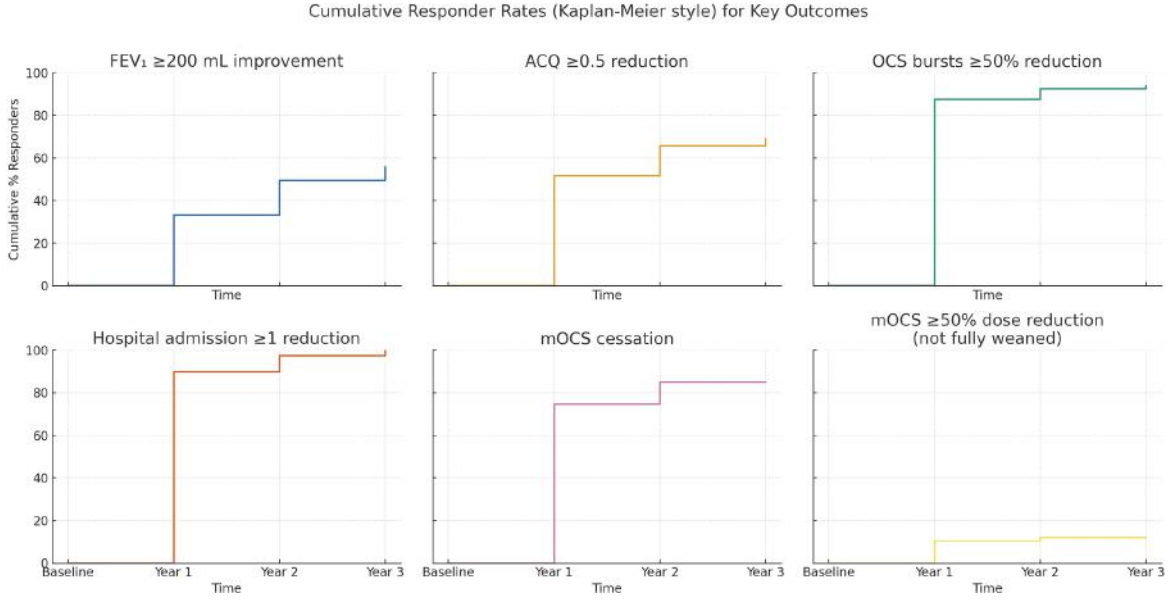
**Conclusion** In real-world practice, biologics provide robust, durable benefits across all asthma domains, with ongoing accrual of multi-domain (quadruple) response over three years. Quadruple-domain response was observed irrespective of biomarker profile but was less frequent among those on maintenance steroids at baseline. Tracking cumulative, multidomain outcomes is essential for optimal biologic selection and asthma management.

## S135 TWICE-YEARLY DEPEMOKIMAB DEMONSTRATES EFFICACY IN PATIENTS WITH ASTHMA ACROSS BASELINE MEDIUM- AND HIGH-DOSE ICS SUBGROUPS: PHASE III SWIFT-1/2 STUDIES

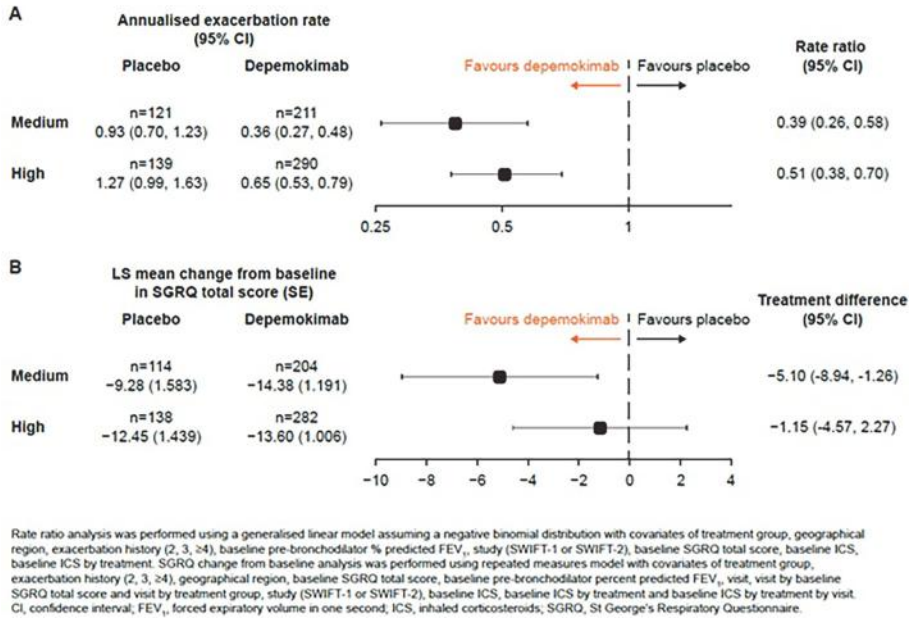
[1]ID Pavord, [2]M Bonini, [3]D Jarreta, [4]N Bird, [5]A Vichiendilokkul, [6]P Chanez. *[1]Oxford Respiratory NIHR Biomedical Research Centre, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK; [2]Department of Public Health and Infectious Disease, Sapienza University of Rome, Rome, Italy; [3]Clinical Sciences, GSK, Madrid, Spain; [4]Biostatistics, GSK, Stevenage, UK; [5]US Medical Affairs, GSK, Collegeville, PA, USA; [6]Aix-Marseille University, C2VN INSERM, Marseille, France*

10.1136/thorax-2025-BTSabstracts.141

**Introduction and Objectives** Depemokimab is the first ultra-long-acting biologic with enhanced IL-5 binding affinity, high potency, and extended half-life, enabling twice-yearly dosing in patients with asthma. Previously approved biologics are recommended as add-on treatment to high-dose ICS. In SWIFT-1/2, depemokimab significantly reduced exacerbations, with




**Abstract S134 Figure 1** Sequential cumulative responder rates for $FEV_1$, ACQ, OCS burst reduction, hospital admission, mOCS cessation, and mOCS dose reduction (Kaplan-Meier-style curves by year)




Rate ratio analysis was performed using a generalised linear model assuming a negative binomial distribution with covariates of treatment group, geographical region, exacerbation history (2, 3, ≥4), baseline pre-bronchodilator % predicted $FEV_1$, study (SWIFT-1 or SWIFT-2), baseline SGRQ total score, baseline ICS, baseline ICS by treatment. SGRQ change from baseline analysis was performed using repeated measures model with covariates of treatment group, exacerbation history (2, 3, ≥4), geographical region, baseline SGRQ total score, baseline pre-bronchodilator percent predicted $FEV_1$, visit, visit by baseline SGRQ total score and visit by treatment group, study (SWIFT-1 or SWIFT-2), baseline ICS, baseline ICS by treatment and baseline ICS by treatment by visit. CI, confidence interval; $FEV_1$, forced expiratory volume in one second; ICS, inhaled corticosteroids; SGRQ, St George's Respiratory Questionnaire.

**Abstract S135 Figure 1** **(A)** Annualised exacerbation rate and **(B)** LS mean change from baseline to Week 52 in SGRQ total score in patients receiving depemokimab or placebo stratified by baseline ICS dose (medium/high)

sustained suppression of inflammation as assessed by blood eosinophil count in patients with type 2 asthma. We aimed to assess depemokimab efficacy in SWIFT-1/2 patients by medium- and high-dose ICS subgroup.

**Methods** Patients randomised 2:1 (stratified by medium-/high-dose ICS) with any ACQ-5 score were studied for 52 weeks and received depemokimab 100 mg subcutaneous or placebo at Week 0 and 26, plus standard of care. Pre-specified analyses examined exacerbation rate and change from baseline in St George's Respiratory Questionnaire (SGRQ) score by baseline ICS dose.

**Results** Depemokimab reduced exacerbations vs placebo by 61% and 49% in patients on medium- and high-dose ICS, respectively (figure 1). There was a greater SGRQ improvement for depemokimab vs placebo in patients on medium- vs high-dose ICS (figure 1).

**Conclusions** Depemokimab reduces exacerbations in patfients with asthma across baseline ICS dose subgroups, supporting potential initiation prior to ICS escalation.

**Funding** GSK (206713/213744; NCT04719832/NCT04718103).

# Pulmonary rehabilitation: what works and why

## S136 IMPLEMENTATION OF THE BTS CLINICAL STATEMENT ON PULMONARY REHABILITATION 2023: EVIDENCE FROM A COMMUNITY COPD SERVICE

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10.1136/thorax-2025-BTSabstracts.142

Pulmonary rehabilitation (PR) in chronic obstructive pulmonary disease (COPD) is associated with improved symptoms, exercise capacity, health- related quality of life and a reduction in exacerbation frequency. The British Thoracic Society (BTS) clinical statement (2023) recommends both aerobic and resistance exercise training as part of PR programmes, which underpins standards to achieve accreditation from the PR Service Accreditation Scheme (PRSAS). We aimed to evaluate the impact of a new PR programme ($PR^{new}$), adapted to meet PRSAS standards, on outcomes in patients from a COPD community service (CCS), compared to the former PR programme ($PR^{old}$).

PR programmes were community-based, comprising of 90-minute supervised sessions performed twice weekly; $PR^{old}$ consisted of aerobic components only, whilst $PR^{new}$ included the addition of resistance training, in lieu of 3 aerobic components. Clinical outcomes following the completion of the programmes were compared between programmes, including: COPD assessment test (CAT) score, hand grip strength (HGS) and incremental shuttle walk test (ISWT), in a retrospective analysis of patients under the care of a CCS, Manchester University NHS Foundation Trust, between 2023–2025.

COPD patients (n=89) were aged 67.6 (8.7) years, well balanced for biological sex (53.9% female) and 28.1% were current smokers, with a pack year history of 39.0 (21.9). Mean $FEV_1$ was 69.0% (19.4%). $PR^{new}$ and $PR^{old}$ comprised n=43 vs n=46 COPD patients; there were no differences in baseline characteristics between groups (table 1).

Significant improvements in CAT, HGS and ISWT were observed following completion of $PR^{new}$ (CAT: 20.1 vs 15.9, $p<0.01$; ISWT: 234.7 vs 327.0m, $p<0.01$; HGS: 26.4 vs 29.4 kg, $p=0.01$) and $PR^{old}$ (CAT: 22.6 vs 16.8, $p<0.01$; ISWT: 215.9 vs 295.0m, $p<0.01$; HGS: 30.6 vs 32.1 kg, $p=0.02$). No differences in the magnitude of improvement were observed between programmes for any clinical outcome when assessed as a continuous variable or as the proportion of patients meeting the MCID (table 1).

**Abstract S136 Table 1** Clinical characteristics and outcomes following completion of the pulmonary rehabilitation programme

| Characteristic | All (n=89) | $PR^{new}$ (n=43) | $PR^{old}$ (n=46) | p-value |
|---|---|---|---|---|
| Age (years) | 67.6 (8.7) | 68.7 (7.7) | 66.7 (9.47) | 0.29 |
| Sex (F:M) | 48 : 41 | 26 : 17 | 22 : 24 | 0.29 |
| Smoking status (% current) | 28.1 | 27.9 | 28.3 | >0.99 |
| Pack years | 39.0 (21.9) | 37.1 (22.8) | 41.1 (20.9) | 0.29 |
| $FEV_1$% predicted | 69.0 (19.4) | 65.3 (18.1) | 72.6 (20.3) | 0.08 |
| BMI ($kg/m^2$) | 30.4 (81.1) | 29.4 (7.8) | 31.3 (8.5) | 0.19 |
| CAT before | 21.3 (6.9) | 20.1 (6.6) | 22.6 (7.0) | 0.66 |
| CAT after | 16.4 (6.5) | 15.9 (6.5) | 16.8 (6.5) | 0.98 |
| Δ CAT | -5.0 (6.2) | -4.2 (6.5) | -5.8 (5.8) | 0.28 |
| Achieved MCID (%) | 65.2 | 62.8 | 67.4 | 0.33 |
| ISWT before (m) | 225.0 (125.0) | 235.0 (122.0) | 216.0 (129.0) | 0.39 |
| ISWT after (m) | 311.0 (145.0) | 327.0 (144.0) | 295.0 (147.0) | 0.21 |
| Δ ISWT (m) | 80.5 (78.1) | 89.1 (70.9) | 71.7 (84.9) | 0.07 |
| Achieved MCID (%) | 61.8 | 74.4 | 50.0 | 0.07 |
| HGS before (kg) | 28.5 (11.9) | 26.4 (10.3) | 30.6 (13.0) | 0.21 |
| HGS after (kg) | 30.9 (11.1) | 29.4 (10.5) | 32.1 (11.6) | 0.51 |
| Δ HGS (kg) | 1.6 (6.9) | 1.6 (7.7) | 1.6 (6.1) | 0.66 |
| Achieved MCID (%) | 14.6 | 11.6 | 17.4 | 0.55 |

P-value for comparison between $PR^{new}$ and $PR^{old}$ is reported. MCID defined as 2 (CAT), 47.5m (ISWT) and 5 kg (HGS).

Improvements in outcome measures were observed for all patients undertaking PR, regardless of PR programme structure. Despite the addition of resistance training, we observed no difference in outcomes between the programmes, including HGS, a surrogate marker of muscle strength.

## S137 PREDICTORS OF MEETING THE MINIMAL CLINICALLY IMPORTANT DIFFERENCE IN PULMONARY REHABILITATION OUTCOMES USING A UK NATIONAL DATASET

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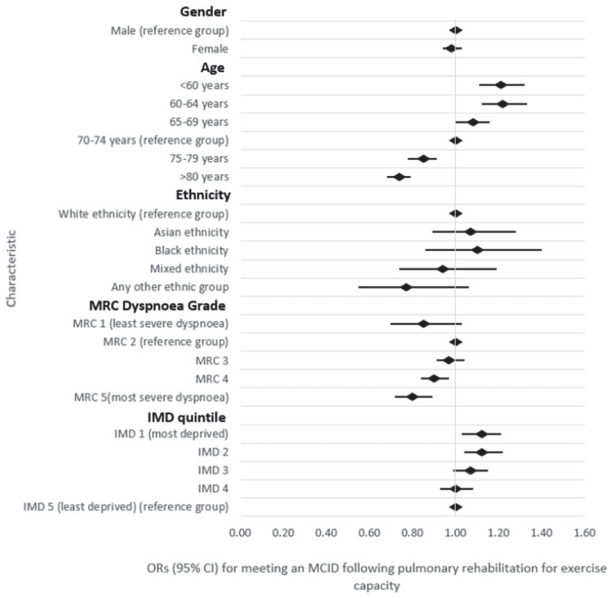
**Background** Pulmonary rehabilitation (PR) is an internationally recommended intervention for individuals living with Chronic Obstructive Pulmonary Disease (COPD). An individual's characteristics are reported to influence PR outcomes.

**Aim** To identify predictors of achieving the minimal clinically important difference (MCID) in PR outcomes using a National Respiratory Audit Programme (NRAP) dataset for England and Wales.

**Methods** 52,669 individuals with COPD entered into the NRAP were assessed for PR from March 2019-September 2023 completed a PR programme. Generalised linear mixed models with a logit link function were used to explore the characteristics associated with achieving MCIDs in PR outcomes. Covariates included age, gender, ethnicity, Medical Research Council dyspnoea grade and index of multiple deprivation (IMD) quintile. Models included exercise capacity (incremental shuttle walk test and six-minute walk test combined, with MCIDs of ≥35m and ≥30m respectively), endurance (endurance shuttle walk test, MCID ≥174 seconds) and health related quality of life (HRQoL) (COPD assessment test and Chronic Respiratory Questionnaire combined, with MCIDs of reduction of ≥2 points and increase of ≥2 points respectively). Meeting the MCID was binary (yes/no).

**Results** Overall, 60.43% (23,730/39,267) met the MCID for exercise capacity, 47.66% (1,700/3,567) for endurance and 61.56% (29,485/47,898) for HRQoL. Age ≥75 years or MRC grades 4 and 5 had significantly reduced odds of meeting the MCID for exercise capacity ($p<0.01$), while age <65 years or IMD 1 and 2 groups (most deprived) had greater odds ($p<0.01$) (figure 1). Similarly for endurance, MRC grade 3, 4 and 5 groups had reduced odds of meeting the MCID (MRC 3: OR 0.72, 0.59–0.88, $p<0.01$; MRC 4: OR 0.53, 0.45–0.66, $p<0.01$; MRC 5: OR 0.38, 0.28–0.52, $p<0.01$). For HRQoL, individuals aged >80 years had reduced odds of achieving the MCID (OR 0.84, 0.78–0.90, $p<0.01$) and MRC grade 4 or 5 groups had increased odds (MRC 4: OR 1.19, 1.12–1.26, $p<0.01$; MRC 5: OR 1.28, 1.17–1.40, $p<0.01$).

**Conclusions** The odds of meeting the MCID in PR outcomes varied across characteristics, with higher MRC grades associated with reduced odds of achieving the MCID in exercise capacity and endurance outcomes, but increased odds of meeting the MCID in HRQoL outcomes.




**Abstract S137 Figure 1** Plot showing the odds ratios for meeting the minimal clinically important difference in exercise capacity outcome measures across individual characteristics (OR: odds ratios; CI: confidence intervals; MCID: minimal clinically important difference; MRC: Medical Research Council; IMD: Index of Multiple Deprivation)

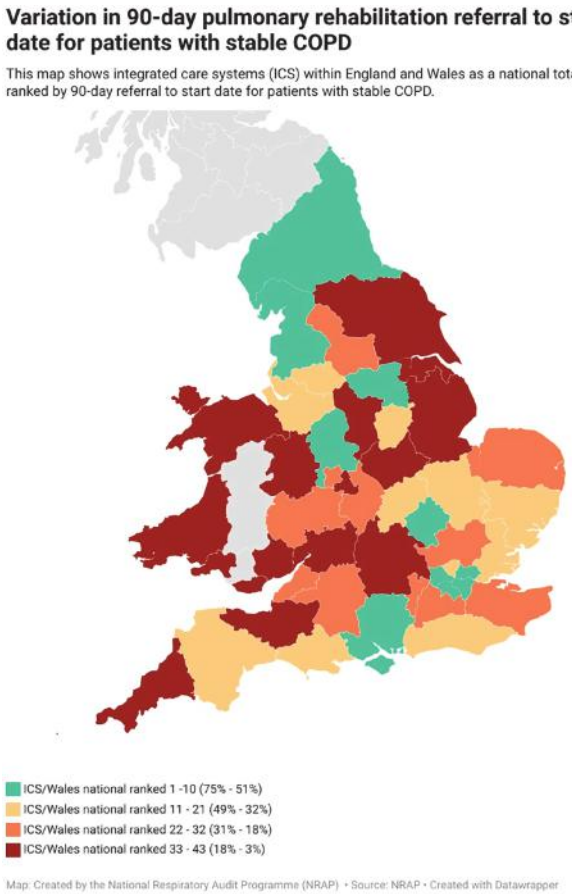
## S138 TIMELY ACCESS TO PULMONARY REHABILITATION IN STABLE COPD AND FOLLOWING AN ACUTE EXACERBATION OF COPD: INSIGHTS FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME

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10.1136/thorax-2025-BTSabstracts.144

**Introduction and Objectives** Pulmonary rehabilitation (PR) is a cost-effective intervention proven to improve quality of life and survival. Individuals with stable chronic obstructive pulmonary disease (COPD) (if suitable) should start PR within 90-days of receipt of referral and individuals referred to PR following hospitalisation for an acute exacerbation of COPD (AECOPD) should be enrolled for PR within 30-days of leaving hospital.[1] It is important that all individuals eligible for PR are able to access the intervention in a timely way to prevent differences in access to care across geographical regions, which may lead to health inequalities.

We aimed to assess if the time to access PR in individuals with stable COPD and individuals post AECOPD is meeting national guidelines.




**Abstract S138 Figure 1** Variation in 90-day pulmonary rehabilitation referral to start date for patients with stable COPD

**Methods** The National Respiratory Audit Programme (NRAP) is a continuous national audit across England and Wales that collects clinical data from PR services, including time to access. This study used data on patients with COPD from the 2023–2024 PR clinical and organisational audit.

**Main results** 27,507 individuals with COPD attended an assessment across 183 PR services. 40% (n=9164) of individuals with stable COPD referred to PR who enrolled in a programme started within 90 days of referral, with considerable variation in access across integrated care systems (3%, 14/451 - 75%, 624/828) (figure 1). Only 4% (n=992) of individuals included in the PR audit were referred to PR after an AECOPD managed in secondary care, of which 69% (n=685) were enrolled. Of these patients, only 16% (n=109) started the programme within 30 days of referral.

**Conclusions** The minority of individuals with COPD access PR in a timely way, with inequities across geographical regions. The small proportion of individuals referred to PR after an AECOPD indicates a missed opportunity for the majority of individuals. Ensuring that services are funded with suitable staffing levels may support timely access to PR.

### REFERENCE

1. BTS Quality Standards for Pulmonary Rehabilitation in Adults. 2014.

## S139 COMPARING THE EFFECTIVENESS OF CENTRE-BASED AND HOME-BASED PULMONARY REHABILITATION USING A UK NATIONAL DATASET

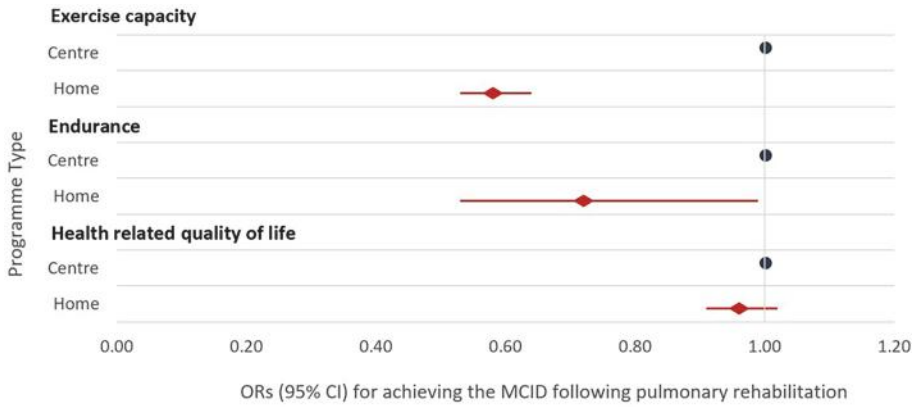
[1,2]H Drover, [1,2]L Wang, [1,2]M Richardson, [3]JK Quint, [1,2]R Evans, [1,2]SJ Singh, [1,2]MW Orme, [1,2]E Daynes. *[1]Department of Respiratory Sciences, University of Leicester, Leicester, UK; [2]Centre for Exercise and Rehabilitation Science, NIHR Leicester Biomedical Research Centre – Respiratory, University Hospitals of Leicester NHS Trust, Leicester, UK; [3]School of Public Health, Imperial College London, London, UK*

10.1136/thorax-2025-BTSabstracts.145

**Background** Pulmonary rehabilitation (PR) is a highly evidenced intervention that improves exercise capacity and health-related quality of life. The BTS Clinical Statement recommends that all eligible individuals are preferentially offered a centre-based PR programme, with home-based programmes offered as an alternative for those unable or unwilling to attend a centre-based programme.

**Aim** To compare the effectiveness of PR between centre-based and home-based programmes using a National Respiratory Audit Programme (NRAP) dataset.

**Methods** 52,669 individuals with COPD entered into the NRAP were assessed for PR from March 2019–September 2023 and completed a programme. Generalised linear mixed models with a logit link function were used to explore the programme types associated with PR effectiveness. PR effectiveness was determined by meeting an established minimal clinically important difference (MCID). Models were adjusted for age, gender, ethnicity, Medical Research Council dyspnoea grade and index of multiple deprivation quintile. Outcomes of effectiveness were exercise capacity (incremental shuttle walk test and six-minute walk test combined, with MCIDs of $\geq$35m and $\geq$30m respectively), endurance (endurance shuttle walk test, MCID $\geq$174 seconds) and health related quality of life (HRQoL) (COPD assessment test and Chronic Respiratory Questionnaire combined, with MCIDs of reduction of $\geq$2 points and increase of $\geq$2 points respectively). Meeting the




**Abstract S139 Figure 1** Plot showing the odds ratios for meeting the minimal clinically important difference in pulmonary rehabilitation outcome measures across centre-based and home-based programmes (OR: odds ratios; CI: confidence intervals; MCID: minimal clinically important difference).

MCID after completing a centre-based or home-based PR programme was binary (yes/no).

**Results** The MCID was achieved more frequently in centre-based programmes than home-based programmes for exercise capacity (centre: 61.37% (n=21,823/35,560); home: 48.36% (n=1,178/2,436) p<0.01), endurance (centre: 48.37% (n=1,588/3,283); home: 38.55% (n=101/262) p<0.01), and HRQoL (centre: 61.90% (n=22,313/36,047); home: 59.89% (n=6,042/10,088) p<0.01). Individuals were significantly less likely to achieve the MCID in exercise capacity or endurance following a home-based programme than a centre-based programme (exercise capacity: OR 0.58, 0.53–0.64, p<0.01; endurance: OR 0.72, 0.53–0.99, p<0.05) (figure 1). There was no significant difference for HRQoL (OR 0.96, 0.91–1.02, p=0.15).

**Conclusions** Effectiveness of PR varied across programme type, with home-programmes less likely to achieve the MCID in exercise capacity or endurance outcome measures. This supports the BTS Clinical Statement that individuals eligible for PR should only be offered a home-based programme if a centre-based programme is declined.

## S140 THE IMPACT OF MULTIPLE LONG-TERM CONDITION PROFILES ON EXERCISE CAPACITY IN THOSE ENROLLED IN PULMONARY REHABILITATION: A LATENT CLASS AND REGRESSION ANALYSIS

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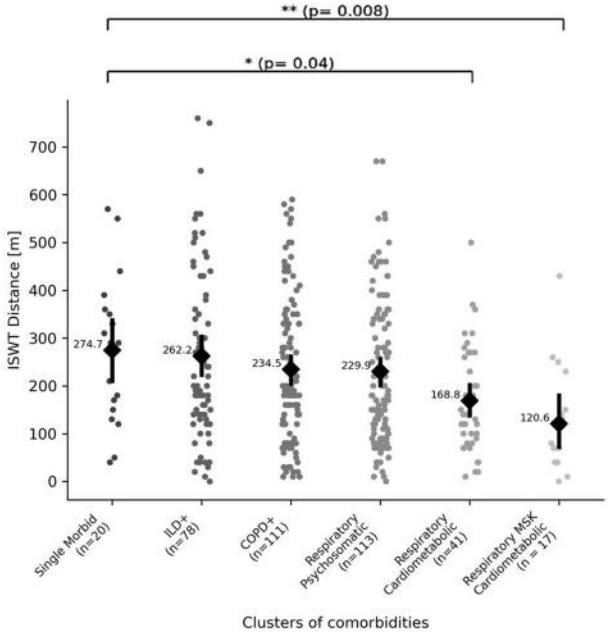
10.1136/thorax-2025-BTSabstracts.146

**Introduction and objectives** Individuals with respiratory disease often display reduced exercise capacity, which is also observed in other long-term conditions (LTCs), including coronary heart disease, heart failure, and chronic kidney disease. In patients with multiple long-term conditions (MLTC)—defined as the presence of two or more LTCs—the interaction of these conditions may compound functional impairment. This study aims to investigate the relationship between MLTC profiles and exercise tolerance, measured by the Incremental Shuttle Walk Test (ISWT), in patients enrolled in Pulmonary Rehabilitation (PR).

**Methods** Data was collected from the PR service at Glenfield Hospital, Leicester (Jan-Nov 2024). Two statistical models were employed. First, a linear regression model assessed the association between the number of LTCs and baseline maximal ISWT distance, adjusting for age. Second, a Gaussian Mixture Model (Latent class analysis) was used to identify distinct MLTC profiles based on posterior probabilities of conditions included in the classes. These class memberships were included in a follow-up regression model predicting ISWT distance.

**Results** 379 patients (67.1±10.8 years old, 51.5% female, 4.7 ±2.25 LTCs) were included in the models. Linear regression indicated that each additional LTC was associated with a significant reduction in ISWT distance of-16 metres (p < 0.001), alongside a significant age-related decline of -3.21 metres per year (p < 0.001).

A five-class model was identified from latent class analysis, yielding clusters labelled as: 'COPD+', 'ILD+' (Interstitial lung disease), Respiratory Psychosomatic (Anxiety & Depression), Respiratory Cardiometabolic, and Respiratory Musculoskeletal Cardiometabolic. Compared to the single morbid




**Abstract S140 Figure 1**

reference group, the Respiratory Cardiometabolic (-84 meters; p = 0.04) and Respiratory Musculo Skeletal Cardiometabolic (-133 meters; p = 0.008) classes showed significantly lower ISWT distance, alongside a significant age-related decline of -3.54 metres per year (p < 0.001).

**Conclusions** These findings support the hypothesis that both the number and specific combinations of MLTCs significantly influence exercise capacity in this cohort of patients enrolled in PR, with potential implications for personalised management and rehabilitation planning.

## S141 AN INTERIM ANALYSIS OF THE LONG-TERM OUTCOMES OF AN EXERCISE-BASED REHABILITATION PROGRAMME IN PREVIOUSLY HOSPITALISED COVID-19 SURVIVORS

[1]S Chakraverty, [1]E Hume, [1]D Megaritis, [1]G Cucato, [1]E Neto, [2]E Daynes, [2]R Evans, [2]S Singh, [3]C Echevarria, [1]I Vogiatzis. *[1]Department of Sports, Exercise and Rehabilitation, Northumbria University, Newcastle Upon Tyne, UK; [2]Department of Respiratory Sciences, University of Leicester, Leicester, UK; [3]The Newcastle upon Tyne Hospitals NHS Foundation Trust, Translational and Clinical Research Institute, Newcastle University, Newcastle Upon Tyne, UK*

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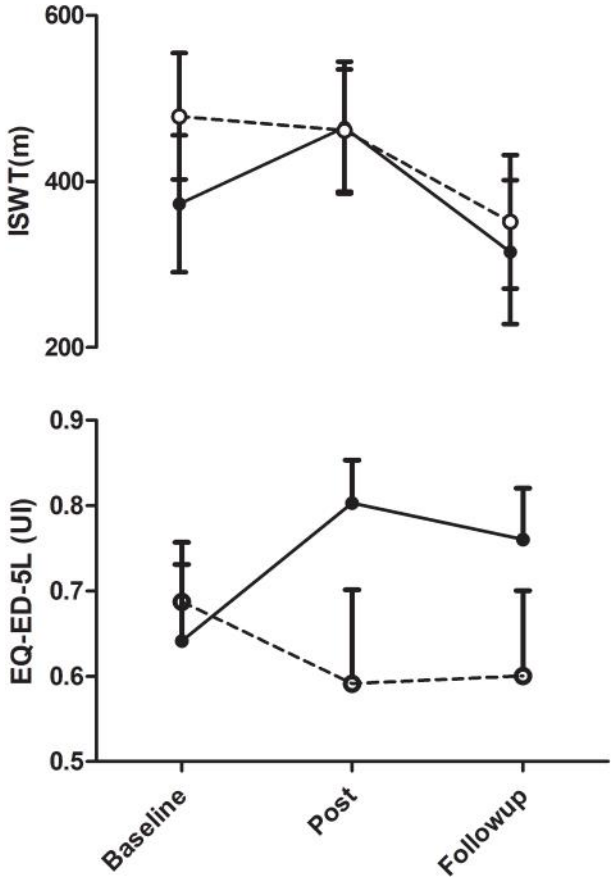
**Background** While exercise-based rehabilitation has shown to improve short-term outcomes in post-COVID syndrome following an acute hospitalisation compared to usual care (UC) (Daynes et al; Eur Respir J 2025; 10.1183/13993003.02152–2024), its long-term effects remain unclear.

**Aim** To determine the long-term effects of a face-to-face (F2F) exercise-based rehabilitation programme compared to UC over a period of 26±3 months post-intervention in previously hospitalised COVID-19 survivors.

**Methods** Participants who took part in the PHOSP-Rehabilitation RCT (ISRCTN10980107/ISRCTN13293865) comparing an 8-week supervised rehabilitation programme (consisting of individualised prescribed exercise and education) to usual care at Newcastle Upon Tyne Hospitals NHS Trust, were invited for a follow-up assessment. The outcomes included change in performance on the Incremental Shuttle Walking Test (ISWT), health-related quality of life (EQ-5D-5L), anxiety and depression scores (GAD-7 and PHQ-9), Quadriceps muscle force (QMVC) and handgrip strength, dyspnoea (Dyspnea-12), fatigue (FACIT-Fatigue Scale), and the short physical performance battery (SPPB) test.

**Results** A total of 23 participants have completed the follow-up visit to date: 12 in the face-to-face (F2F) group (mean age: 70 ± 6 years) and 11 in the usual care (UC) group (mean age: 68 ± 10 years). A two-way repeated measures ANOVA revealed a significant group-by-time interaction only for the ISWT (p=0.02) and the EQ-5D-5L Utility Index (UI) (p=0.05) (figure 1). Post hoc within group analysis (Bonferroni-adjusted) revealed a significant decrease (p=0.001) in the ISWT between baseline and follow up in the UC group (by 127.14 m; 95% CI: -196.09 to -58.19), whereas the decrease in the F2F group (by -58.33 m; CI: -132.8 to 16.14) was not significant (p=0.15). No significant within-group changes were found for EQ-5D-5L UI scores. Compared to baseline, clinically meaningful improvements were observed at follow-up in QMVC in the F2F group compared to the UC group (mean difference: 5.47 kg; 95% CI: 0.68 to 10.25), and in Dyspnea-12 scores (mean difference: 4.91; 95% CI: 0.45 to 9.21).

**Conclusion** The long-term outcomes of an exercise-based rehabilitation programme are superior to usual care in terms of exercise capacity, quality of life, leg strength and dyspnoea symptoms.




**Abstract S141 Figure 1** Data points (mean±SEM) are shown at baseline, post-intervention discharge and follow up in the F2F group (closed circles) and the UC group (open circles) for the ISWT and the EQ-5D-5L UI

# Translational pulmonary vascular science

## S142 PROTEOMIC SIGNATURES FOR GCN2 AND BMPR2-MUTATION DRIVEN PULMONARY VASCULAR DISEASE

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**Introduction** Pulmonary arterial hypertension (PAH) covers a range of diseases characterised by increased pulmonary vessel pressures, which result in right heart failure and death if left untreated. Mutations in genes such as eukaryotic translation initiation factor 2 α kinase 4 (which encodes general control nonderepressible 2, GCN2) and bone morphogenetic protein receptor type 2 (BMPR2) cause heritable pulmonary veno-occlusive disease (PVOD) and heritable PAH respectively, which have particularly poor outcomes. The majority of PVOD patients die within 1–2 years unless transplanted. We sought to find novel plasma biomarkers that would enable us

to accurately and swiftly identify whether patients have genetic mutations.

**Methods** Plasma samples from patients with GCN2-associated PVOD (n=8), BMPR2-associated PAH (n=14), and age- and sex-matched healthy volunteers (n=12, who were confirmed to have no history of PAH, thromboembolic disease and not on anticoagulants) were submitted for plasma proteomic analysis. Demographic data and details of the diagnostic right heart catheterisation (for patients) were also collected.

**Results and discussion** We identified two plasma proteins, Caveolae Associated Protein 2 (Cavin-2) and caldesmon which were elevated in the GCN2-associated PVOD cohort alone. Both cavin-2 and caldesmon levels were capable of distinguishing between GCN2-associated PVOD and healthy volunteers (AUC of 0.90, P=0.0045 for cavin-2 and AUC of 0.85, *P*=0.013 for caldesmon). Intriguingly, they were also both capable of distinguishing between GCN2-associated PVOD and BMPR2-associated PAH (AUC of 0.82, *P*=0.014 for cavin-2 and AUC of 0.79, *P*=0.03 for caldesmon). Both proteins are biologically plausible as biomarkers. Cavin-2-deficient mice are more susceptible to hypoxia-induced pulmonary vasoconstriction. Cavin-2 forms complexes with caveolin-1 and mutations in caveolin-1 can cause heritable PAH. Blockade of caldesmon reduces endothelin-1-induced contraction in pulmonary artery smooth muscle cells. Interestingly there is also an inflammatory signal common to both GCN2-associated PVOD and BMPR2-associated PAH, with elevated levels of serum amyloid A, heparanase and myeloblastin.

**Conclusions** It may be possible to identify a metabolic signature unique to different types of genetically-driven pulmonary vascular disease. This will be validated in an independent Spanish cohort. This approach may be advantageous as it is potentially faster and cheaper than conventional genetic testing.

## S143 STRATOSPHERE 1 STUDY – A NOVEL BMP TARGET ENGAGEMENT BIOMARKER PANEL FOR USE IN CLINICAL TRIALS DEMONSTRATES SOTATERCEPT ALTERS GENE EXPRESSION IN PAH

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10.1136/thorax-2025-BTSabstracts.149

Pulmonary arterial hypertension (PAH) is a rare, life-limiting disease where imbalances in the TGFb superfamily pathways have causal roles in hereditary and idiopathic forms. These pathways are emerging attractive candidates for therapeutic intervention but there is an unmet need for clinically relevant and practical biomarkers that can measure target engagement. A major challenge has been the inaccessibility of lung tissue in disease for molecular profiling. Here we explore the surrogate capacity of peripheral blood BMP pathway-specific markers. Plasma proteomic analysis demonstrates widespread pleiotropic alterations of TGFß/BMP modulators. Downstream BMPR-II canonical and non-canonical signaling is altered and measurable in whole blood, and transcriptomic signatures cluster by discrete BMPR-II gene modules. We present discovery and international replication cohorts for the transcriptomic BMPR-II signaling signatures and derive a composite transcriptomic biomarker panel that is repeatable, reproducible, longitudinally stable and expressed in correlated gene modules in PAH which associate with clinical outcomes, most notably mortality. The assay performance characteristics of the biomarker panel make it feasible for early phase, target engagement clinical trials and we have utilised it in a pilot study of sotatercept-treated patients that suggests the therapy does not mechanistically rebalance/increase BMPR-II pathway signaling, but rather shows a reduction, likely due to depletion of circulating BMP9 and BMP10.

## S144 INVESTIGATION OF WHOLE BODY AND TISSUE SPECIFIC METABOLIC PROFILES DURING THE DEVELOPMENT OF PULMONARY ARTERIAL HYPERTENSION IN THE MONOCROTALINE RAT MODEL

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**Introduction and Objectives** Pulmonary arterial hypertension (PAH) is a fatal disease characterised by progressive vascular remodelling and metabolic dysregulation. Key hallmarks of PAH metabolism include enhanced glycolysis, increased glutamine utilization, decreased fatty acid oxidation and impaired TCA cycle. However, the detailed profile of these metabolic shifts remains incompletely characterised during PAH development. Here we explored the whole body and tissue specific metabolic changes in the monocrotaline (MCT)-induced PAH rat model.

**Methods** MCT (60 mg/kg, 4 weeks)-induced PAH rat model was used to investigate the whole body metabolic profile for healthy baseline and 1–4 weeks after MCT injection using the comprehensive lab animal monitoring system (CLAMS). Tissue specific metabolism, including liver and right ventricular (RV), was assessed using high-resolution respirometry (Oroboros) at weeks 3 and 4. PAH phenotypes were evaluated by echocardiography and Fulton's index.

**Results** As MCT-induced PAH developed, both male and female rats demonstrated decreased oxygen consumption (VO2) and carbon dioxide production (VCO2) in light and dark cycles. From week 1 to week 3, both male and female showed an increased respiratory exchange ratio (RER), suggesting a metabolic shift preference for carbohydrate metabolism versus fat. Female maintained elevated RER levels through week 4. Male RER increased through week1–3 with an initial drop from baseline but exhibited a significant reduction at week 4. Oroboros data indicated that MCT significantly reduced RV mitochondrial function, indicated by decreased mitochondrial oxidative phosphorylation, complex I, II activity and electron-transfer-pathway (ET) capacity at weeks 3 and 4. Liver mitochondrial respiration, including oxidative phosphorylation, complex I, II and ET capacity, was significantly increased at the same period. At week 4, female exhibited higher RV mitochondrial respiration, consumed more food and produced more heat than males at week 4. ECHO and RV hypertrophy indicated more severe PAH phenotypes in males than females at weeks 3 and 4.

**Conclusion** This study described the rat's whole body and tissue specific metabolic profile during the MCT-induced PAH development for the first time. Our results support the metabolic difference between the male and female in PAH. This pattern may underlie observed differences in disease severity and therapeutic response in different sexes.

## S145 INTERROGATION OF THE GLYCOLYTIC AXIS IN PULMONARY ARTERIAL HYPERTENSION MODELS AND HOW MODIFYING THESE CAN LEAD TO POTENTIAL THERAPEUTIC OPTIONS

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10.1136/thorax-2025-BTSabstracts.151

**Introduction** Pulmonary arterial hypertension (PAH) is an umbrella term describing a group of diseases characterised by increased pulmonary vessel pressures that lead to right ventricular failure and death if left untreated. Dysregulated cellular metabolic function has been reported in endothelial cells (ECs) from PAH patients. Normal ECs generate more than 80% of their ATP via glycolysis, which enables rapid ATP production compared to oxidative phosphorylation. Thus, ECs can quickly adapt to pro-angiogenic stimuli and shift rapidly from quiescence to cellular functions such as proliferation, migration and apoptosis. Pulmonary artery ECs (PAECs) isolated from PAH patients are even more glycolytic, which is accompanied by a reduction in oxidative phosphorylation. This phenomenon is known as the Warburg effect and was initially reported in cancer. We have investigated the roles of both the splicing factor polypyrimidine-tract-binding protein (PTBP1) and its target, pyruvate kinase M2 (PKM2), known instigators of glycolysis, paying particular attention to the regulation of proliferation, migration, and apoptosis in PAH patient-derived endothelial cells.

**Methods** We used blood outgrowth endothelial cells (BOECs) derived from healthy volunteers and PAH patients with *BMPR2* mutations. The effects of apigenin, a trihydroxyflavone suppressor of the Warburg effect, and a PKM2 activator, TEPP-46, were used to manipulate the PTPB1-PKM2 axis both in this cell model and then in the PAH Sugen-hypoxia rat model.

**Results and discussion** Drug specific effects were observed in both control and *BMPR2* mutant BOECs, as TEPP-46 stabilised the tetrameric form of PKM2 while reducing the dimer; whereas apigenin reduced the tetrameric PKM2 and inhibited expression of PTBP1. Both drugs decreased levels of PTPB1 and reduced endothelial cell proliferation and apoptosis. Only apigenin decreased lactate production in both control and mutant BOECs. In the Sugen-hypoxia rat model, administration of TEPP-46 but not apigenin significantly ameliorated the elevated right ventricular systolic pressure and loss of body weight associated with Sugen-hypoxia.

**Conclusions** We have shown that TEPP-46 treatment suppresses EC glycolysis, by repressing PKM2-driven glycolytic gene expression, with the functional effect of ameliorating the Sugen-hypoxia phenotype. This suggests that correcting endothelial cell dysfunction may improve the overall PAH phenotype.

## S146 INFLAMMATION AND OBESITY CORRELATE IN PULMONARY HYPERTENSION BUT ASSOCIATE WITH DIVERGING OUTCOMES

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10.1136/thorax-2025-BTSabstracts.152

**Rationale and Objectives** Inflammation is associated with all types of Pulmonary Hypertension (PH) both as a known cause and/or a putative confounder. The most common marker of inflammation, C-reactive protein (CRP), has not been widely studied in PH. This study set out to clarify if CRP informed clinical endotyping and outcomes.

**Methods** Time-series clustering of longitudinal CRP levels was employed. Clinical differences between clusters were validated in three independent UK/international cohorts using clinical cut off values (n=10,301; UK-cohort, ASPIRE and FDA cohort). Associations were analysed with functional and mortality outcomes by linear and Cox regression models including all-causes of PH (groups 1–5). To add mechanistic insight, multi-omics were interrogated from associated previously published arrays.

**Results** Patients segregated into two stable CRP clusters (median CRP 2 versus 6.5 mg/l), with the high cluster exhibiting significantly higher BMI (difference between medians DBM=5.4 kg/m2), higher RAP (DBM=2 mmHg) and reduced 6-minute walk distance (6MWD (DBM=55m)).

Inflammation was associated with worse survival, comorbidities, higher pulmonary vascular resistance (PVR), and smoking status. CRP and BMI were associated with differing inflammatory profiles in proteomic and transcriptomic analyses. Despite the relationship with CRP, higher BMI associated with improved survival, lower PVR and did not negatively affect 6MWD treatment-related functional responses.

**Conclusions** We establish a relationship between CRP and BMI across all-cause PH, though CRP and BMI associate with diverging clinical outcomes. Inflammation and obesity are relevant phenotypes for consideration in clinical trial design. Understanding their impacts on outcomes is important for clinical practice.

# Pleural disease – basic science, new trials

## S147 AN INTEGRATED UNBIASED MULTI-OMIC APPROACH TO UNDERSTAND THE IMMUNE RESPONSE TO PLEURAL INFECTION IN A DISEASE CONTEXT

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10.1136/thorax-2025-BTSabstracts.153

**Background** Pleural infection is a severe and complex disease with increasing incidence worldwide. The profile of neutrophil subsets and neutrophil extracellular traps (NETs) in pleural infection remains unclear.

**Aim** This study aims to investigate the neutrophil repertoire and its association with clinical outcomes.

**Methods** Single-cell RNA sequencing (scRNA-seq) and cell-surface protein profiling were performed on paired whole blood and pleural fluid samples collected from pleural infection patients (n=4). An independent cohort (pleural infection patients, n=13; non-infective effusion controls, n=4) was included for bulk RNA sequencing validation.

**Results** Neutrophils underwent the strongest transcriptional and compositional change in pleural fluid in contrast to blood. The heterogeneity of the neutrophils revealed an immature subpopulation exclusively detected in the blood, an activated subpopulation mostly appeared in the pleural fluid, and a mature subpopulation present in both. Interestingly, we found a group of hypoxia related neutrophils that highly express HIF1A and the glycolysis pathway. This subpopulation was exclusively present in the pleural fluid and was related to anaerobic infection by paired metagenomics data. Bulk RNA sequencing deconvolution validated the neutrophil preferences in pleural fluid and blood. Hypoxia related genes were also highly up regulated in the pleural fluid.

**Conclusion** This study identified neutrophil heterogeneity in pleural fluid and indicated a special hypoxia related neutrophil subpopulation in the pleural fluid.

## S148 CITRULLINATION OF FIBRIN BY PAD4 IS AN IMPORTANT CONTRIBUTOR TO FIBRINOLYTIC DEFICIENCY IN PLEURAL INFECTION

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10.1136/thorax-2025-BTSabstracts.154

**Background** Pleural infection is a complex disease with significant morbidity and mortality. Pleural fluid drainage and administration of antibiotics are the first line treatment. Intrapleural fibrinolytic deficiency leads to accumulation of fibrin deposits which segregate the pleural cavity and hamper fluid drainage. Patients with septations receive intrapleural fibrinolytic treatment (IFT, tissue plasminogen activator and DNase) to improve drainage. Citrullination of fibrin by Peptidylarginine deiminase 4 (PAD4), a key enzyme that initiates NETosis by citrullinating histones, has been suggested to lead to formation of denser fibrin clot, resulting in increased resistance to lysis.

**Aim** To assess the level of intrapleural citrullinated fibrin and its effect on the fibrinolytic potential ex vivo with clinical samples obtained from pleural infection patients prospectively at baseline and post-IFT.

**Results** We found an increase ($p<0.0001$) in soluble fibrin after IET, confirming that the treatment acts to increase the cleaved fibrous tissue. Significant decrease in the percentage of citrullinated fibrin was observed in the pleural cavity of pleural infection patients after IFT ($p<0.0001$), suggesting citrullinated fibrin contributes to fibrinolytic deficiency. A tendency of negative correlation ($r=$ -0.5351, $p=$ 0.073) was found between citrullinated fibrin and D-dimers, suggesting citrullinated fibrin could lead to fibrinolytic deficiency. Subsequent ex vivo experiments showed an increase in the level of D-dimers in the presence of the PAD4 inhibitor GSK484, tPA and Dnase ($p=$ 0.0003), confirming the contribution of citrullinated fibrin to fibrinolytic deficiency. Patients with higher intrapleural level of citrullinated fibrin were also found to be more likely to develop septations ($p=$ 0.0078). Moreover, the baseline intrapleural level of soluble fibrin was found to be potentially higher in patients who responded well to first line antimicrobial treatment in comparison to patients who proceeded to acquire IFT ($p=$ 0.0965).

**Conclusion** Our data suggests that the citrullination of fibrin contributes to fibrinolytic deficiency and hence the formation of fibrous septations. Our data also suggests that PAD4 inhibitor could potentially improve pleural fluid drainage.

## S149 BACTERIA RESPONSIBLE FOR INDWELLING PLEURAL CATHETER COLONISATION (BRICC)

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10.1136/thorax-2025-BTSabstracts.155

**Background** Indwelling pleural catheters (IPCs) offer symptom relief from pleural effusions. Usage is complicated by infection in ~5% of cases.[1] IPCs can be colonised when bacteria grow from pleural fluid without infective features.[2] The causes of colonisation are undescribed and its relationship with infection unexplored.

**Aims**

1. Describe the incidence and microbiology of IPC colonisation.
2. Identify clinical features associated with IPC colonisation.
3. Compare asymptomatically colonising bacteria with those from infection.

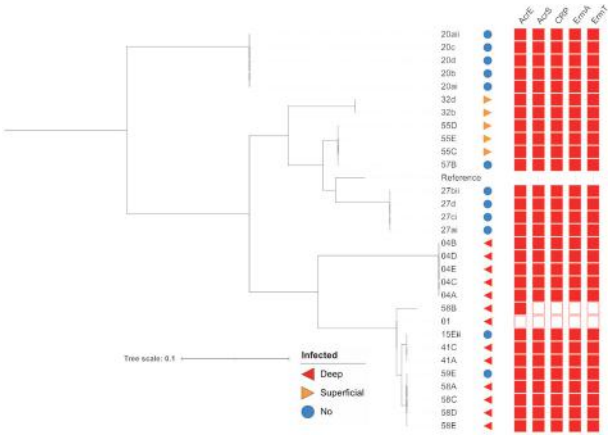
**Methods** All infected and non-infected IPCs were collected from four sites, along with associated metadata. Infected IPCs were identified clinically and non-infected IPCs categorised as 'colonised' if bacteria was cultured from them, and 'clean' if

not. The genomes of all cultured organisms were sequenced, assembled and taxonomically classified. For *Staphylococcus aureus* isolates, a maximum likelihood phylogenetic tree was constructed from Snippy-core alignment using IQ-Tree, with *S. aureus* NCTC8325 as reference.

**Results** Of 108 IPCs, 54 were 'clean', 34 colonised and 20 infected. Infected and colonised IPCs had longer mean insertion durations (197.6 and 181.3 days) compared with 'clean' ones (98 days).

Of 34 colonised IPCs, 17 were colonised by multiple species. The most common causes were Coagulase-negative *Staphylococci* (CoNS n=18), Enterobacteriaceae (n=18), Corynebacteria spp (n=4) & *S. aureus* (n=4).

Two of 20 infected IPCs were culture negative. We retrieved the same organism causing infection from 14/18 IPCs. The most common pathogens were *S. aureus* (n=8), *Pseudomonas aeruginosa* (n=3), Enterococcus spp (n=2) and *Enterobacter cloacae* (n=2). Figure 1 demonstrates clustering of *S. aureus* isolates from deep and superficial infection.




**Abstract S149 Figure 1** Phylogenetic tree of *Staphylococcus aureus* isolates from colonised and infected IPCs. *S. aureus* NCTC8325 is the reference strain

**Conclusion** A significant proportion of IPCs are colonised. Colonisation and infection are associated with longer insertion duration. Enterobacteriaceae and CoNS are the most common causes of colonisation, whereas *S. aureus* and *Pseudomonas aeruginosa* commonly cause infection.

*S. aureus* isolates causing deep infection show higher genetic similarity versus colonising strains. This, and our phylogenetic analysis, suggests colonisation does not cause infection, but rather there is a virulent subset of isolates causing infection. Ongoing work will identify genes and markers associated with infection.

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## S150 REDUCE 2: A RANDOMISED FEASIBILITY STUDY COMPARING RECURRENT THERAPEUTIC THORACENTESIS TO INDWELLING PLEURAL CATHETER INSERTION, DAILY DRAINAGE AND TALC INSTILLATION FOR THE MANAGEMENT OF MEDICALLY-RESISTANT CARDIOGENIC PLEURAL EFFUSIONS

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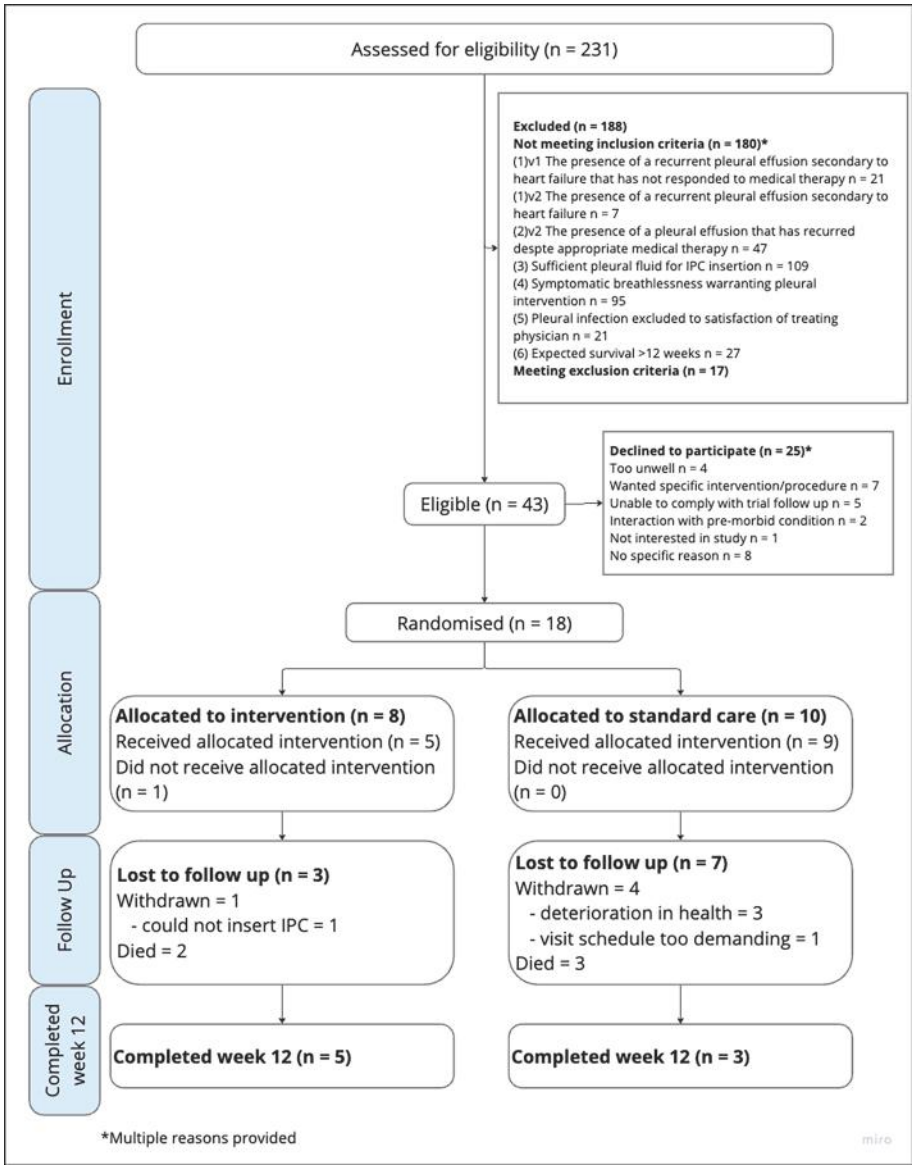
**Background** Heart failure (HF) is the commonest cause of pleural effusions, and up to 10% are resistant to medical therapy. The current standard care for resistant effusions is recurrent therapeutic thoracentesis (TT). Indwelling pleural catheters (IPCs) plus talc improve pleurodesis rates in malignant pleural effusions. This is the first randomised study to examine daily drainage and talc instillation via IPC in cardiogenic pleural effusions.

**Aims** To assess the feasibility of a full-scale trial of recurrent TT vs IPC insertion, daily drainage and talc instillation across 12 UK centres. The primary outcome was feasibility, defined as recruitment of 80% of the target of 40 patients and ≥50% eligible patients.

**Methods** Adult patients with a resistant HF-related pleural effusion were eligible for randomisation. Participants were randomised 1:1 in a non-blinded fashion to receive recurrent TT or IPC, daily drainage, and talc. The follow-up period was 12 weeks.

**Results** Of the 231 patients screened, 43 met eligibility criteria. Furosemide up titration and SGLT2i addition were the most common changes in the 47 patients deemed ineligible due to medical optimisation. 18 (42%) patients were randomised, with a median age of 79 (IQR 70–83) and a median Clinical Frailty Score of 6 (IQR 5–7). Eight patients completed follow-up, with five deaths and five withdrawals. Pleurodesis was reported in 3/8 (38%) in the IPC group and 0/10 in the TT group. Total pleural procedure numbers were lower in the IPC group (1.0:2.2, p<0.001). Median reduction of dyspnoea at 4 weeks (on 100 mm scale) was 58 mm in the IPC group; 40 mm in TT(p=0.247). A higher complication rate was observed in the IPC arm.

**Conclusions** The study did not meet the feasibility threshold. Patients with resistant HF effusions are elderly and frail, with limited life expectancy. Advances in medical HF management may have reduced the incidence of symptomatic HF effusions




**Abstract S150 Figure 1**

requiring recurrent drainage - a notable proportion of screened patients in this study were managed successfully with medical approaches. In persistently symptomatic patients, IPC/talc was a potentially beneficial management strategy; however, medical management with TT remains standard care.

## S151 WHEN TO TEST FOR BIRT-HOGG-DUBÉ SYNDROME? UK NATIONAL DATA

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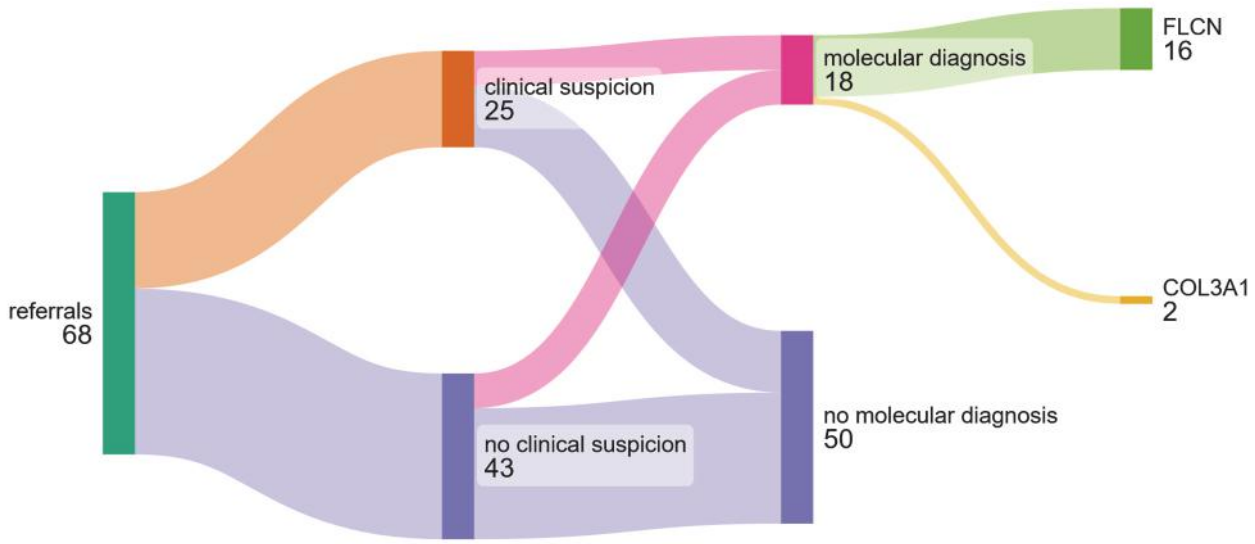
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**Background** Birt-Hogg-Dubé syndrome (BHD) is the commonest monogenic cause of pneumothorax. Caused by pathogenic variants within *FLCN* it can manifest as pneumothorax, pulmonary cysts, and skin features including fibrofolliculomas. Affected families are at increased risk of renal malignancy and diagnosis permits lifesaving renal surveillance. Our gene panel developed in the 100,000 Genomes Project became the UK genetic test for Familial Pneumothorax: the R190 panel. Testing criteria were initially: a history of spontaneous pneumothorax and having a first-degree relative with pneumothorax. However, not all suspected cases meet these criteria.

**Methods** We analysed national and regional data to assess the predictive value of these criteria for diagnosis of BHD using data from (i) the Rare Disease Collaborative Network (RDCN) for Familial Pneumothorax (n=77 patients); (ii) North East and Yorkshire Genomic Laboratory Hub (GLH, n=68 patients); (iii) Cambridge University Hospitals (n=53 patients).

**Results**

- GLH referral for testing had a 24% (16/68) FLCN positivity; fraction positivity increased to 36% (9/25) with a clinical suspicion of 'syndromic diagnosis'; figure 1
- RDCN MDT recommendation for testing had a 38% (8/21) FLCN-positivity




**Abstract S151 Figure 1**

- CUH cohort: 20% (10/51) confirmed FLCN positivity, with higher positivity (33%, 10/30) in those with pulmonary cysts; 20% of those with FLCN pathogenic variants had bibasal pulmonary cysts but no history of pneumothorax

**Conclusion** Our data support extending FLCN gene testing to individuals with characteristic lung cysts alone. Expanding FLCN gene testing will enable earlier diagnosis, facilitating family surveillance for renal malignancies and personalised care.

## S152 PRAGMATIC NON-INFERIORITY RANDOMISED TRIAL INVESTIGATING NEEDLE ASPIRATION VERSUS CHEST DRAIN FOR SECONDARY SPONTANEOUS PNEUMOTHORAX. THE PRINCE-SSP STUDY

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10.1136/thorax-2025-BTSabstracts.158

**Introduction** Secondary spontaneous pneumothorax (SSP) is a medical emergency where the lung collapses in the presence of underlying chronic lung disease. Current international clinical guidelines recommend the insertion of an intercostal chest drain (ICD) for SSP. However, needle aspiration (NA) is a simpler and quicker procedure, is less painful and associated with fewer complications than ICD. The primary objective of PRINCE-SSP was to test whether NA is non-inferior to ICD for the initial length of hospital stay (LOHS) during 30 days from randomisation.

**Methods** PRINCE-SSP was an efficient, pragmatic, multicentre, open-label, parallel, two-group, randomised, non-inferiority trial conducted in 20 UK hospitals over a 2-year period. Eligible patients had to have an SSP. Patients could be enrolled either by giving informed consent if they had capacity or via a deferred consent pathway, whereby consent was sought if the patient regained capacity within 72 hours of randomisation or else from a personal or nominated consultee. Participants were randomly allocated to the intervention (NA) or usual care (ICD) and followed up for at least 30 days. The primary outcome is initial LOHS up to day 30 post-randomisation, with pre-specified non-inferiority margin of +1 day.

**Results** Between August 18, 2023 and April 30, 2025, 303 patients were screened. 72 eligible patients were enrolled and randomised: 37 to NA and 35 to ICD. One patient (randomised to ICD) was subsequently found not to have a pneumothorax, prior to receiving any trial treatment, and was excluded from any further trial activities.

The mean (SD) age of the remaining 71 participants was 65.6 (13.5) years, with range 37 to 91; 55 (77.5%) were men; 88.7% were white; 64.8% had COPD and 54.9% had recurrent pneumothorax. The median pneumothorax size at the hilum was 3.55 cm (range 0 to 13.1). Baseline characteristics were generally well-balanced between allocated groups. Three deaths were reported: two during the initial hospital stay (one in each allocated group) and one death (ICD) reported after the initial hospital stay.

**Conclusion** Final follow-ups were in June 2025 and analyses are now underway. Results on clinical effectiveness and implications for practice will be presented and discussed.

# The importance of early diagnosis in ILD: novel approaches

## S153 ILA PHENOTYPE AND PROGRESSION IN ILD PATIENTS IN A TERTIARY ILD CENTRE

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10.1136/thorax-2025-BTSabstracts.159

**Introduction** Interstitial Lung Abnormalities (ILAs) are subtle lung parenchymal changes on CT scans, characterised by reticular patterns, ground-glass opacifications (GGO), and traction bronchiectasis. They represent early lung changes and may

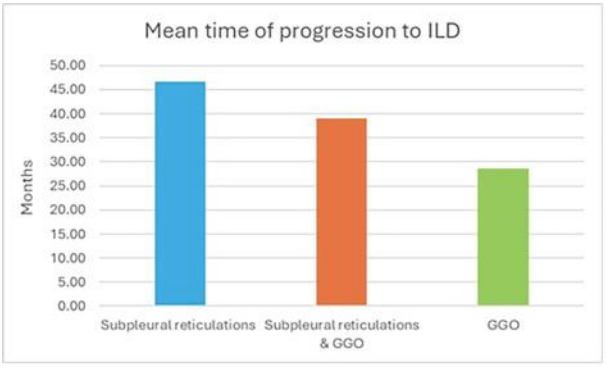
precede the development of Interstitial lung Disease (ILD). The timeline for progression remains unclear.

**Rationale** Our study aims to retrospectively evaluate initial lung changes in patients with confirmed ILD, to determine the average time for progression to ILD, and assess how factors such as age, gender, and smoking status influence progression rate and disease severity.

**Method** Retrospective cohort study of patients reviewed in the ILD MDT at a tertiary academic ILD unit between April 2024 – March 2025. 498 patients were discussed at MDT, of whom 223 were formally diagnosed with ILD. Inclusion criteria required patients to have undergone at least 1 Chest CT within the 10 years preceding their MDT discussion. Patients with Smoking related-ILD and CTD-ILD were excluded, as their progression is better understood. For the remaining patients, the earliest scan showing ILA in the 10 years prior to diagnosis was identified. Scans showing established fibrosis at this earliest time point were excluded from further analysis, leaving a cohort of 39 patients.

**Results** In this cohort, 36% were female and 64% male. Most (62%) were ex-smokers, with pack-years ranging from 5 to 92; 20.5% were non-smokers. The most common early CT finding was subpleural reticulations (64%), followed by combined reticulations and GGOs (23%) and isolated GGOs (13%). Progression time from ILA to ILD varied by initial CT findings: GGOs progressed in 29 months, a combination of subpleural reticulations and GGOs progressed in 39 months and subpleural reticulations alone progressed to ILD in 47 months. The average time to progression was 42.6 months.

**Conclusion** No current established guidelines exist for monitoring ILAs. Patients are often discharged back to primary care and re-evaluated only after respiratory symptoms develop, by which time fibrosis is often advanced. This study highlights the poorly characterised progression timeline of ILAs to ILD and suggests that regular surveillance, including annual high-resolution CT scans, may enable earlier detection and timely initiation of antifibrotic treatment to improve outcomes.



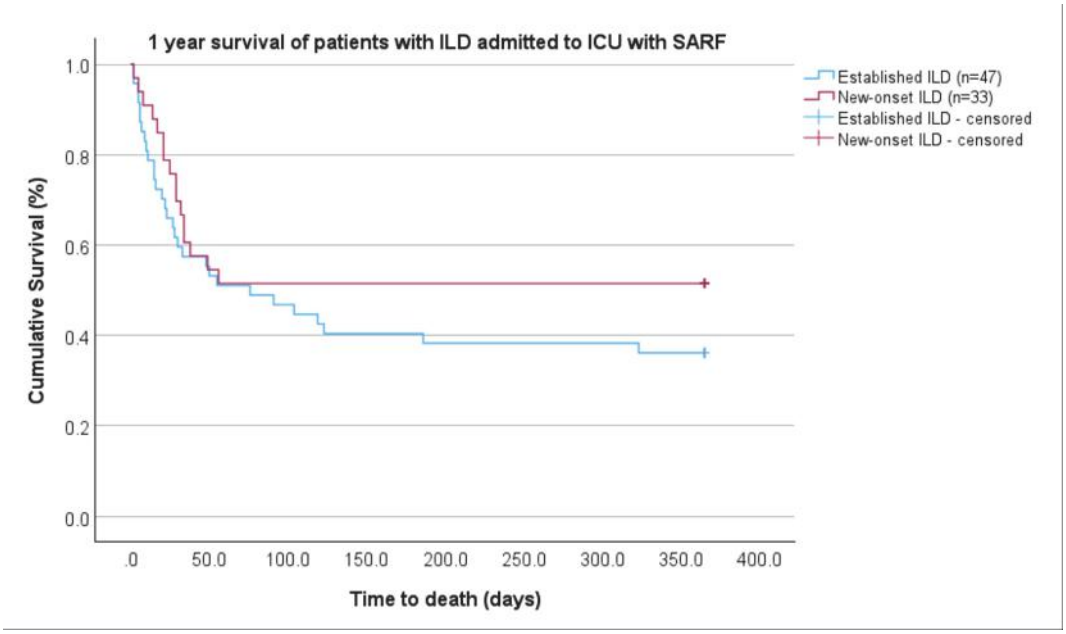

**Abstract S153 Figure 1**

## S154 PATIENTS WITH NEW-ONSET INTERSTITIAL LUNG DISEASE (ILD) DO NOT HAVE HIGHER MORTALITY THAN THOSE WITH ESTABLISHED ILD WHEN ADMITTED TO INTENSIVE CARE WITH SEVERE ACUTE RESPIRATORY FAILURE (SARF)

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10.1136/thorax-2025-BTSabstracts.160

**Introduction** Admission to the intensive care unit (ICU) is deemed unfavourable for most patients with interstitial lung disease (ILD) who develop severe acute respiratory failure (SARF). Owing to the fulminant trajectory of their disease, individuals with new-onset ILD may have a particularly poor prognosis. This study compares ICU and post-discharge outcomes between patients with new-onset ILD and established ILD following admission for SARF.




**Abstract S154 Figure 1**

**Methods** Patients with ILD-related SARF and consecutively admitted to the adult ICU between 2009–2023 were included in this retrospective observational cohort study. New-onset ILD was defined as onset of ILD symptoms less than 6 months before ICU admission. Established ILD was thus defined as onset of ILD symptoms more than 6 months before ICU admission. Kaplan-Meier time-to-event analysis was censored at 12 months from ICU admission.

**Results** Of 80 patients with ILD-SARF (55% male, median age 51 IQR 43–60), overall ICU mortality was 50%. Connective-Tissue Disease ILD (CTD-ILD), Idiopathic Interstitial Pneumonias (IIPs) and Exposure-Related ILD formed 40%, 28% and 10% of ILD subtypes diagnosed, respectively. 43 patients were transferred from secondary care. 50 were already intubated at admission. During admission, 64 patients received invasive mechanical ventilation (IMV), 23 received extracorporeal membrane oxygenation therapy (ECMO), and 30 received 2 or more immunosuppressive medications. Non-survivors in ICU had higher admission APACHE (p=0.026) and SOFA (p=0.018) scores, older age (OR 1.09, 95% CI 1.01–1.16), lower BMI (OR 0.84, 95% CI 0.72–0.98), and were more likely to receive IMV during admission (p=0.024). ICU mortality amongst new-onset ILD patients (n=33) was comparable to those with established ILD (n=47) (48% vs 51%, p= 0.82). New-onset ILD patients had a longer median length of ICU stay (25 vs 13 days, p=0.128) and time on IMV (29 vs 16 days, p=0.077). 1-year survival was higher in new-onset ILD patients (51% vs 36%, p=0.172).

**Conclusion** Half the patients with ILD-associated SARF survived ICU admission at this specialist SARF/ILD centre. New-onset ILD patients had similar ICU survival to established ILD, with possibly better 1-year survival. Further data is being analysed for predicting ICU and post-discharge survival of patients with ILD-SARF treated at a SARF centre.

## S155 POTENTIAL OF AI TO DETECT ILD IN PRIMARY CARE SETTINGS: A REAL-WORLD EVALUATION

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**Introduction** Interstitial Lung Disease (ILD) prevalence is rising, yet ILD often remains misdiagnosed or diagnosed late, particularly due to referral delays (3.8±2.1 years between first spirometry and diagnosis).[1] In primary care, spirometry is the predominant test for assessing respiratory symptoms, but ILD is often overlooked. ArtiQ.Spiro, an AI-based support tool for spirometry interpretation, shows strong potential to assist clinicians in identifying possible ILD. We examine the prevalence of AI-flagged ILD cases in a real-world primary care setting to evaluate AI's potential to prompt earlier specialist referrals.

**Methods** The Hillingdon Confederation runs a central spirometry hub for four GP practices and uses ArtiQ.Spiro to support spirometry execution and interpretation. We applied the AI model to demographic and clinical spirometry data from 2,794 consecutive individuals undergoing spirometry between October 2022 and May 2025. Interpretation followed the ERS/ATS 2021 standard. The AI-predicted ILD cases in primary care were assessed and contextualised based on spirometry patterns. Prediction reliability was supported by prior external validation[2] demonstrating strong ILD screening performance (AUC=0.9, NPV=92.3%, PPV=60.5%).

**Results** The model predicted ILD in 7.8% of cases (n=219; see the table for characteristics). These predictions are associated with higher age compared to the total cohort, and reduced FVC z-scores with normal to elevated FEV1/FVC z-scores (0.41±0.93), as expected in ILD patients when compared to a healthy population. Among the ILD predicted cases, 55.7% exhibited a possible restrictive pattern, 43.4% a normal pattern, and 0.9% a possible mixed disorder. Predicted ILD cases include 24.6% of the patients with obesity and restrictive pattern, indicating the model's predictions are not driven by obesity-related restriction. While the previously validated PPV suggests some overprediction, high NPV provides confidence that most true ILD cases are being identified.

**Conclusions** ArtiQ. Spiro can support ILD diagnosis by flagging at-risk patients across both restrictive and normal spirometry patterns that might otherwise be overlooked. Further work is needed to evaluate how integrating ArtiQ.Spiro into diagnostic pathways could impact secondary care by weighing the benefits of earlier ILD detection against the risks of unnecessary specialist referrals.

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## S156 TARGETED CLINICAL MANAGEMENT OF PROBANDS AND AT-RISK RELATIVES IN FAMILIAL PULMONARY FIBROSIS: A REGIONAL MODEL WITH NATIONAL RELEVANCE

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10.1136/thorax-2025-BTSabstracts.162

**Background** Familial Pulmonary Fibrosis (FPF) manifests when ≥2 family members have idiopathic pulmonary fibrosis or another interstitial lung disease (ILD). With advancements in precision and genomic medicine, there is wider scope to support the care of probands and their first-degree relatives.

**Aim** To assess the role of a dedicated FPF clinic and Multidisciplinary Team (MDT) discussion, involving national and international expertise, to optimise management of probands with suspected genetic predisposition and identifying first-degree relatives at risk of developing ILD.

**Methods** Prospective data were collected from December 2024 to June 2025 for patients attending our FPF service in the UK. We analysed baseline demographics, clinical and functional characteristics, FPF genetic results (table 1) and MDT outcomes.

**Results** A total of 51 patients were seen through our FPF service, with 50 patients (98%) consenting for the R421-FPF gene panel (nhsgms-panelapp.genomicsengland.co.uk/panels/1174/v1.3). Forty probands (78%) had family history of ILD, with 49 (96%) having living first-degree relatives ≥40 y/o without ILD. Per proband, the median of first-degree relatives was 2 (range 0 – 8). Of the total cohort, 31% of the total cohort (16/51) had first-degree relatives with clinical features suggestive of telomere biology disorders.

**Abstract S156 Table 1** Demographic, clinical, functional, and genetic characteristics of patients with ILD and suspected genetic predisposition evaluated in our Regional Familial Pulmonary Fibrosis Service

| Characteristics | N=51 |
|---|---|
| **Demographic** | |
| **Age in years, mean (±SD)** | 65.7 (12.5) |
| **Male, n (%)** | 25 (49.1) |
| **Female, n (%)** | 26 (50.9) |
| **Referring Centre in the United Kingdom:** | 40 (78.4) |
| • South West Peninsula, England, n (%) | 11 (21.6) |
| • Other regions, n (%) | |
| **Family history of ILD** | 40 (78.4) |
| **Clinical** | |
| **ILD diagnosis:** | |
| • Idiopathic Pulmonary Fibrosis, n (%) | 31 (60.8) |
| • Idiopathic non-specific interstitial pneumonia, n (%) | 1 (1.9) |
| • Hypersensitivity pneumonitis, n (%) | 6 (11.8) |
| • Connective tissue disease associated with ILD, n (%) | 2 (3.9) |
| • Unclassifiable ILD, n (%) | 3 (5.9) |
| • Pleuroparenchymal fibroelastosis, n (%) | 3 (5.9) |
| • Other, n (%) | 5 (9.8) |
| **Lung function** | |
| **FVC - percent predicted, mean (±SD)** | 77.6 (21.1) |
| **DLCO - percent predicted, mean (±SD)** | 54.0 (15.4) |
| **Genetic testing** | |
| **R421 Familial Pulmonary Fibrosis criteria*, n (%)** | |
| 1) Idiopathic ILD <50 y/o | 6 (11.8) |
| 2) Family history of ILD | 35 (68.6) |
| 3) Suspected telomerase complex mutations/Telomere Biology Disorder | 4 (7.8) |
| 4) More than one R421 criteria (e.g. 1+2, 1+3, 2+3) | 6 (11.8) |
| **Available results -R421 gene panel, n (% of total consenting cohort)** | 25 (50.0) |
| 1. No pathogenic variant detected | 6 (24.0) |
| 2. Pathogenic/likely pathogenic variant, n (% of 25 available results) | 3 (12.0) |
| 3. VUS, n (% of 25 available results)# | 6 (24.0) |
| 4. Risk allele (*MUC5B* gene)# | 10 (40.0) |
| **Type of gene affected (% of 25 available results)** | |
| 5. Telomere-related gene variant, n (% of 25 available results) | 9 (36.0) |
| 6. Surfactant-related gene variant, n (% of 25 available results) | 0 (0.0) |

Legend: ILD=interstitial lung disease; FVC=forced vital capacity; DLCO=diffusing capacity of the lungs for carbon monoxide; SD= standard deviation; VUS=variant of uncertain significance.
*https://www.england.nhs.uk/wp-content/uploads/2018/08/rare-inherited-disease-eligibility-criteria-v8.0.pdf (National Genomic Test Directory, latest version: v8.0 May 2025, Page 372 – R421 testing criteria).
#For primary R421 gene panel results; however, three probands had multiple variants identified.

Up to June-2025, 25 genetic results were received (50% of the consenting cohort), with an average reporting time of 5 months; 12% (3/25) had pathogenic/likely pathogenic variants and 24% (6/25) had Variants of Uncertain Significance (VUS). 52% (13/25) of all results have been discussed in the FPF MDT with 46% (6/13) of all probands being referred to Clinical Genetics services, 23% for Telomere Length testing and consideration of VUS reclassification, and 15% to another specialist service (Hepatology). For the first-degree relatives, 31% (4/13) were referred to Clinical Genetics services and 15% to specialist services (Respiratory/ILD) as per MDT consensus.

**Conclusion** Over the initial 6-month service evaluation, we identified a high prevalence of first-degree relatives potentially at risk of developing ILD through our dedicated FPF MDT. Further regional and national referrals will likely emphasize this unmet clinical need and support development of a formal NHS pathway/screening programme for first-degree relatives at risk of developing ILD.

## S157 A SINGLE CENTRE RETROSPECTIVE STUDY INVESTIGATING THE UTILITY OF BRONCHOALVEOLAR LAVAGE IN THE DIAGNOSIS OF INTERSTITIAL LUNG DISEASE

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10.1136/thorax-2025-BTSabstracts.163

**Introduction** Bronchoalveolar lavage (BAL) differential cellular analysis may be a valuable tool in the diagnostic evaluation of patients with Interstitial Lung Disease (ILD), with international consensus guidelines typically recommending consideration in those without a definite Usual Interstitial Pneumonia pattern on HRCT, however the practical utility continues to be debated.[1]

**Method** A single centre retrospective casenote review of patients undergoing BAL as part of ILD diagnostic workup.

**Results** 36 patients underwent BAL between January 2022 and June 2025. 22 (61.1%) were female and median age was 66 years. 19 (52.7%) were ex-smokers with 13 (36.1%) non-smokers and 2 (5.5%) current smokers. The mean smoking pack year history was 21.3 years. BAL was generally well tolerated with no significant complications; 2 patients (5.5%) experienced minor complications with one experiencing hypoxia and subsequent early termination of the procedure; and one experiencing excessive coughing and discomfort.

The most frequent diagnoses included acute hypersensitivity pneumonitis (25%), fibrotic hypersensitivity pneumonitis (19.4%) and connective tissue disease related ILD (8.3%). 3 patients (8.3%) had a diagnosis of unclassifiable ILD following BAL and subsequent discussion in an ILD MDT.

31 (86.1%) of patients had their management influenced through BAL analysis. 11 patients (30.5%) were commenced on immunosuppression and 6 patients (16.6%) on anti-fibrotic medications.

**Conclusion** Bronchoalveolar lavage was safe and influenced MDT discussion and management in the majority of patients in this selected cohort. Systems standardising the integration of BAL into cytopathology departments and ILD MDTs might

further increase utility and therefore aid in the diagnostic pathway of interstitial lung diseases.


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## S158 APPLICATIONS OF LUNG ULTRASOUND IN INTERSTITIAL DISEASES


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**Introduction** In diffuse interstitial lung disease (ILD) the gold standard imaging technique is chest computed tomography (CT) but less invasive techniques such as ultrasound are being considered for screening and follow-up of these patients.

**Aim** To analyse the first 54 ultrasounds in ILD performed in our Unit.

**Methods** Patients referred for ultrasound assessment (January 2023-July 2024). Data were collected from functional respiratory tests, radiological study with CT and the results obtained in the lung ultrasound. Patients were classified as mild, moderate and severe according to the Warrick scale for chest CT and the Bologna Ultrasound for Diffuse Alveolar Disease (BUDA) scale.

**Results** 54 patients (5.9% male), mean age 71±9 years. The table 1 shows the most frequent pathologies and ultrasound characteristics. Pulmonary function of our patients (averages): FVC 2283 ±877 ml, FVC% 71±18%, FEV1 1855±660 ml, FEV1% 76±18%, FEV1/FVC 82±9%, DLCO 3.29±1.65 ml/min/mmHg, DLCO% 49±21%. Radiologically 68.5% had a pattern compatible with UIP while 31.5% had a NINE pattern.

When applying the Warrick index the patients were classified as: 11.1% mild, 50% moderate and 38.9% severe. When classified with the ultrasound score (BUDA index): 35.8% mild, 28.3% moderate and 35.8% severe.

We found a moderate negative correlation between DLCO and average B lines (r = -0.408) and a slight negative correlation between DLCO and areas with irregular pleura (r = -0.292) and Warrick Score (r = -0.292), having statistical significance (<0.05).

**Conclusions**

- ILD has a number of typical ultrasound features and can be used as a clinical tool for the diagnosis and follow-up due to its low cost, availability and absence of radiation, but further studies are needed to standardise the technique.
- The best radiological/ultrasound correlation is in cases of severe involvement.

**Abstract S158 Table 1** Ultrasound characteristics according to interstitial pathology

| ILD (n=54) | AVERAGE B LINES PER AREA | AREA AREAS WITH IRREGULAR PLEURA | PLEURAGE WITH REDUCED GLIDING |
|---|---|---|---|
| Idiopathic pulmonary fibrosis (30) | 4.1±2.7 | 4.5±2.6 | 11 patients (36.7%) |
| Hypersensitivity pneumonitis (14) | 4.8±2.7 | 3.1±2.1 | 3 p. (21.4%) |
| Rheumatoid arthritis (6) | 5.4±2.9 | 5.5±1.9 | 1 p. (16.7%) |
| Fibroenfisema (1) | 8.6 | 8 | 0 p. (0%) |
| Sarcoidosis (1) | 3.1 | 1 | 1 p. (100%) |
| Unclassifiable (1) | 6.5 | 5 | 0 p. (0%) |
| Scleroderma (1) | 5.8 | 4 | 1 p. (100%) |

# Thinking outside the (asthma) biologic box

## S159 BIOLOGIC THERAPY IS GREEN! FINDINGS FROM THE ASTHMA BIO CARBON STUDY

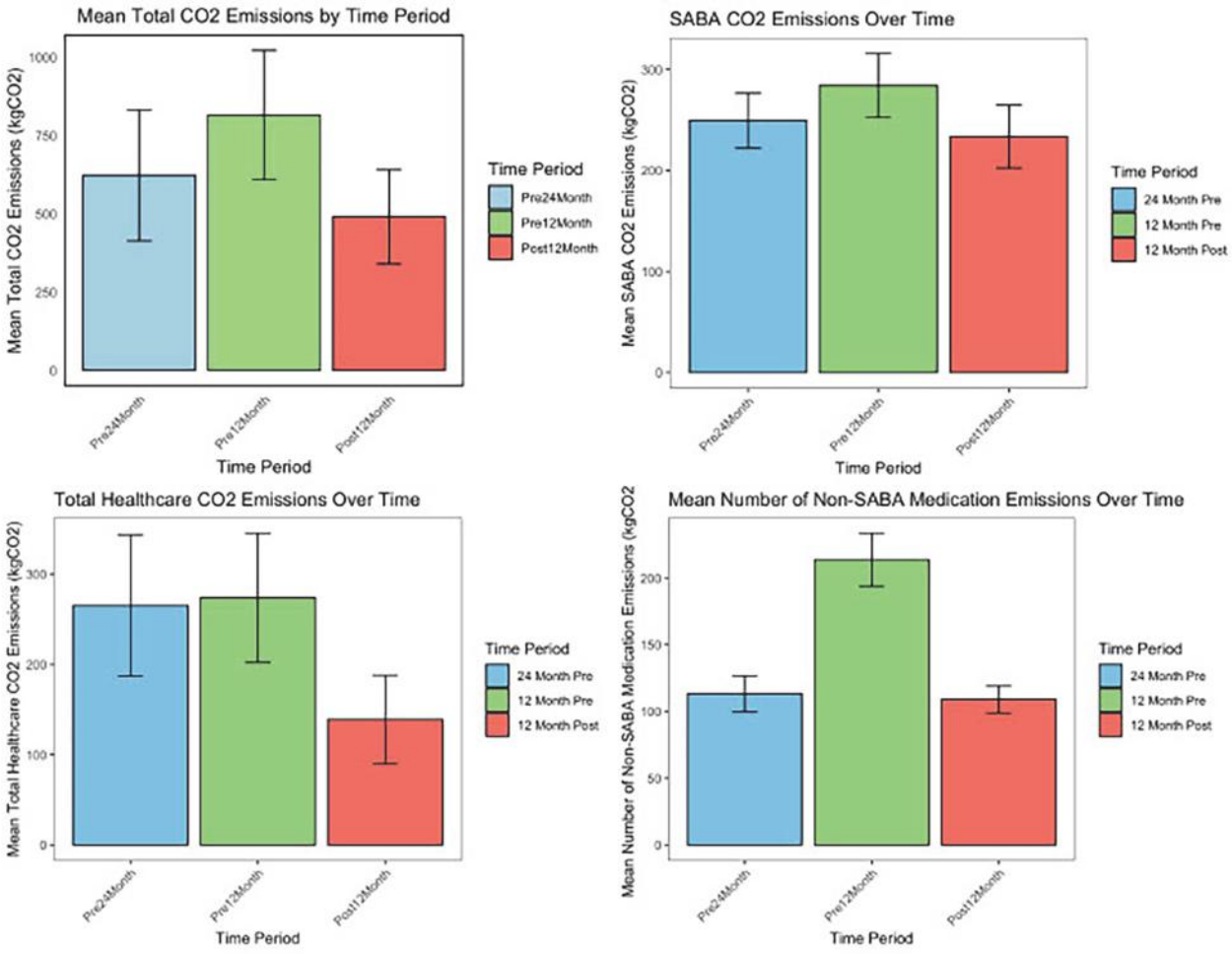

[1]DL Sykes, [1]S Faruqi, [1]H Cummings, [2]A Bahra, [2]D Sammut, [3]IJ Clifton, [3]G Baynes, [4]S Davies, [4]C Fewings, [4]H Bladon, [4]T Shemald, [5]L Jones, [1]MG Crooks. *[1]Respiratory Research Group, Hull York Medical School, Hull, UK*; *[2]Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield, UK*; *[3]Leeds Teaching Hospitals Trust, Leeds, UK*; *[4]Chesterfield Royal Hospital, Chesterfield, UK*; *[5]Mid Yorkshire Teaching NHS Trust, Wakefield, UK*


10.1136/thorax-2025-BTSabstracts.165

**Introduction** Improving asthma control and reducing the use of short-acting beta agonist (SABA) inhalers is associated with a reduction in greenhouse gas (GHG) emissions. We aimed to assess the impact of biologic treatment for people with severe asthma (pwSA) on GHG emissions from healthcare utilisation and asthma medication usage.

**Methods** In this multi-centre study, data were retrospectively collected for pwSA. Data on healthcare utilisation, medication usage, and distance travelled were collected for the following 3 periods: 24–12 months prior to, 12 months prior to, and 12 months following initiation of any biologic therapy for severe asthma.

**Results** 80 patients (55% Female, n=44) with a mean age of 59.8 (SD =13.3) were included in this study. There was a significant reduction in mean total CO2 emissions [kgCO2] 12 months after initiation of biologic therapy when compared to the previous 12 months (815.4 vs 491.15, p=0.007), however, there was no significant difference in emissions when comparing the period 24–12 months before with either 12 months before or 12 months after initiation. There were also significant reductions in mean total medication CO2 emissions [kgCO2] (498.7 vs 330.9, p<0.001) and median healthcare contact emissions (11.5 vs 1.0, p<0,001) 12 months after




**Abstract S159 Figure 1** Bar charts/Box and whisker plots demonstrating: **A)** changes in total CO2 emissions **B)** Healthcare-related emissions **C)** Emissions due to SABA inhalers **D)** Emissions due to non-SABA medications in the periods 24 months prior, 12 months prior, and 12 months following biologic initiation

initiation of biologic therapy when compared 12 months pre-biologic. There was a numerical difference in carbons emissions [kgCO2] attributable to SABA inhalers between the periods 12 months prior to and following biologic initiation, however this was non-significant (284 vs 233, p=0.129).

**Conclusions** Biologic therapy for pwSA significantly reduces total, non-SABA medication-related, and healthcare-associated carbon emissions in the 12 months following initiation, compared to the 12 months prior to starting treatment. However, there was no significant difference between the periods 24–12 months before initiation and 12 months after initiation. This suggests that poor control in the 12 months prior to initiation is associated with greater emissions and improved control with biologics reduces emissions. Previous data have shown a similar reduction in healthcare contact-related CO2 emissions but not overall emissions.[1] Our data suggest biologic treatment reduce carbon emissions from all aspects of asthma care.

## REFERENCE

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## S160 IMPROVED QUALITY OF LIFE, EARLY SYMPTOM CONTROL AND REDUCED SURGERY WITH MEPOLIZUMAB IN CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSWNP): RESULTS FROM THE SYNAPSE STUDY

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**Introduction and objectives** Mepolizumab, an anti-interleukin-5 biologic, targets type 2 inflammation in CRSwNP. While the Phase III SYNAPSE trial demonstrated the efficacy of mepolizumab in reducing nasal symptoms and surgery needs by Week-52, the timing and progression of its clinical benefits

**Abstract S160 Table 1** Summary of improvements in overall symptom VAS, SNOT-22 and nasal surgery across time points in the SYNAPSE study

| Outcome | Proportion of patients achieving overall symptom VAS responder status* (%) | Proportion of patients achieving SNOT-22 responder status† (%) | Proportion of patients requiring nasal surgery (%) |
|---|---|---|---|
| | | **N=206 (M); N=201 (P)** | |
| **Weeks 1–4** (VAS); | 6% (M) vs 7% (P) | 64% (M) vs 53% (P) | N/A |
| | OR: 0.88 | OR: 1.66 | |
| **By Week 4** (SNOT-22, surgery) | (95% CI: 0.40–1.95) | (95% CI: 1.09–2.53) | |
| | p=0.754 | **p=0.017** | |
| **Weeks 9–12** (VAS); | 35% (M) vs 20% (P) | 82% (M) vs 64% (P) | <1% (M) vs 2% (P) |
| | OR: 2.20 | OR: 2.71 | HR: 0.62 |
| **By Week 12** (SNOT-22, surgery) | (95% CI: 1.39–3.47) | (95% CI: 1.69–4.36) | (95% CI: 0.11–3.54) |
| | **p<0.001** | **p<0.001** | p=0.595 |
| **Weeks 33–36** (VAS); | 62% (M) vs 42% (P) | 75% (M) vs 61% (P) | 7% (M) vs 16% (P) |
| | OR: 2.22 | OR: 1.88 | HR: 0.50 |
| **By Week 36** (SNOT-22, surgery) | (95% CI: 1.49–3.32) | (95% CI: 1.22–2.90) | (95% CI: 0.26–0.95) |
| | **p<0.001** | **p=0.004** | **p=0.033** |
| **Weeks 49–50** (VAS); | 64% (M) vs 40% (P) | 73% (M) vs 53% (P) | 9% (M) vs 23% (P) |
| | OR: 2.64 | OR: 2.44 | HR: 0.43 |
| **By Week 52** (SNOT-22, surgery) | (95% CI: 1.76–3.97) | (95% CI: 1.60–3.73) | (95% CI: 0.25–0.76) |
| | **p<0.001** | **p<0.001** | **p=0.003** |

CI, confidence interval; HR, hazard ratio; M, mepolizumab; OR, odds ratio; P, placebo; SNOT-22, Sino-Nasal Outcome Test-22; VAS, visual analogue scale. *≥2.5 improvement; †≥8.9 improvement. Values are shown in bold where p<0.05.

remain less understood. This analysis evaluates response patterns to mepolizumab over time.

**Method** SYNAPSE randomised patients 1:1 to receive subcutaneous mepolizumab 100 mg, or placebo, with standard of care. Response timings (weeks) for clinical outcomes were assessed post hoc by overall symptom visual analogue scale (overall VAS) and Sino-Nasal Outcome Test-22 (SNOT-22) responder status (defined as a ≥2.5-point and ≥8.9-point improvement from baseline, respectively) and nasal surgery status.

**Results** Of the 407 randomised patients (206 mepolizumab, 201 placebo), mepolizumab demonstrated early efficacy in key clinical outcomes. During Weeks 9–12, a significantly higher proportion of patients in the mepolizumab group achieved clinically meaningful improvements in overall VAS scores, indicating reductions in nasal symptom burden (table 1). Similarly, improvements in SNOT-22 scores were also observed by Week-4 and -12 in the mepolizumab group. By Week-36, mepolizumab reduced the risk of nasal surgery by 50% compared with placebo (table 1).

Of those receiving mepolizumab, patients classified as overall VAS responders were significantly more likely than non-responders to subsequently be SNOT-22 responders. This association was observed as early as Week-12 (OR [95% CI]: 2.62 [1.10–6.24], p=0.030) and persisted through later time points, including Week-16 (OR [95% CI]: 3.09 [1.24–7.69], p=0.015) and Week-20 (OR [95% CI]: 2.44 [1.08–5.53], p=0.032). Furthermore, patients who achieved SNOT-22 responder status at Week-12 were significantly less likely to require nasal surgery by Week-20 (HR [95% CI]: 0.14 [0.03–0.70], p=0.016), with this association remaining statistically significant through Week-52 (p<0.02). Patients who had nasal surgery by Week-36 were more likely to require systemic corticosteroids prior to Week-36 than those who did not require nasal surgery (HR [95% CI]: 5.11 [2.38–10.96], p<0.001), with this continuing through Week-52.

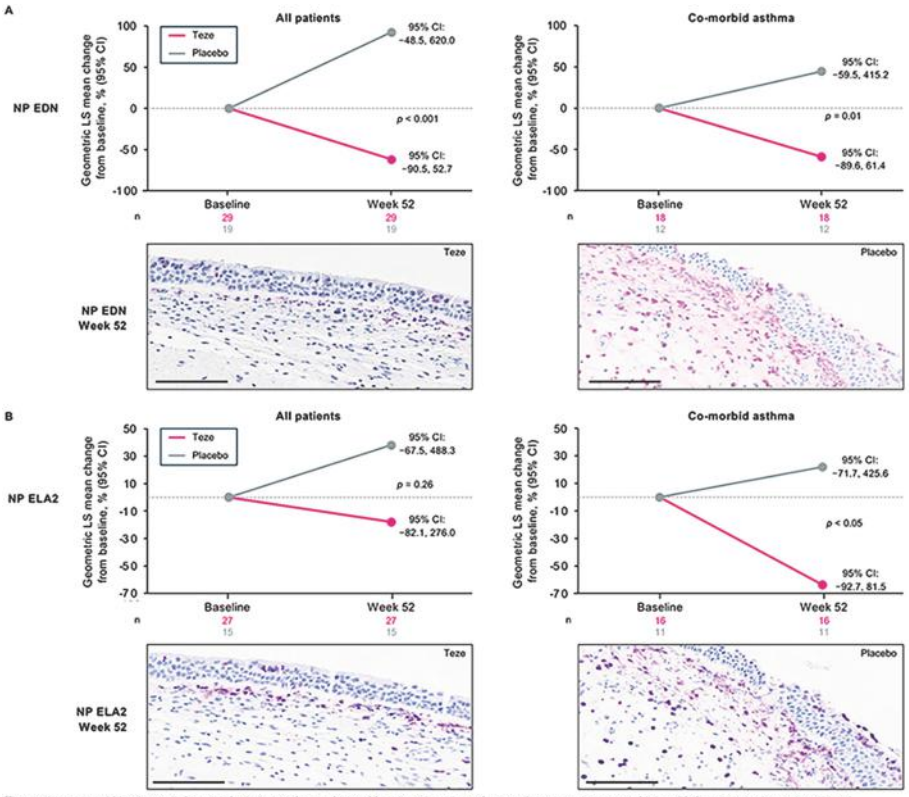
**Conclusion** Patients receiving mepolizumab achieve early responses with significant overall symptom VAS and SNOT-22 score improvements from Week-12, and nasal surgery benefits from Weeks 20–36, potentially predicting durable responses by Week-52.

## S161 TEZEPELUMAB REDUCES INFLAMMATORY BIOMARKERS IN ADULTS WITH SEVERE CHRONIC RHINOSINUSITIS WITH NASAL POLYPS WITH AND WITHOUT COMORBID ASTHMA: RESULTS FROM THE PHASE 3 WAYPOINT STUDY

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**Introduction and Objectives** In the phase 3 WAYPOINT study (NCT04851964), tezepelumab demonstrated efficacy for the treatment of severe chronic rhinosinusitis with nasal polyps (CRSwNP). This analysis aimed to evaluate the effects of




The asthma comorbid subpopulation also included patients with aspirin-exacerbated respiratory disease and non-steroidal anti-inflammatory drug-exacerbated respiratory disease. Geometric least-squares mean change from baseline in IHC-positive cells/mm² between treatment arms was analysed using a Gaussian (parametric) linear regression model with region, history of NP surgery and comorbidity status as covariates. *p* values refer to the test of systematic difference between treatment arms in terms of change from baseline. Images are representative micrographs of IHC performed on NP samples from placebo or tezepelumab recipients in the overall study population at week 52. Scale: 100µm. CI, confidence interval; EDN, eosinophil-derived neurotoxin; ELA2, neutrophil elastase; IHC, immunohistochemistry; LS, least squares; NP, nasal polyp; Teze, Tezepelumab.

**Abstract S161 Figure 1** Changes from baseline to week 52 in nasal polyp eosinophil-derived neurotoxin (A) and neutrophil elastase (B) levels overall and among patients with comorbid asthma

tezepelumab on inflammatory biomarkers in patients from the overall WAYPOINT population and those with comorbid asthma.

**Methods** Patients with CRSwNP received tezepelumab 210 mg or placebo subcutaneously every 4 weeks for 52 weeks. This pre-specified exploratory analysis assessed changes from baseline in levels of serum IL-5 and IL-13, nasal lining fluid (NLF) IgE, plasma and nasal polyp eosinophil-derived neurotoxin (EDN), and neutrophil biomarkers plasma myeloperoxidase or elastase in nasal polyp tissue.

**Results** 122/203 tezepelumab recipients and 126/205 placebo recipients had comorbid asthma. Baseline serum and NLF thymic stromal lymphopoietin (TSLP) levels were similar between treatment arms. Tezepelumab significantly reduced levels of all biomarkers, except those associated with neutrophils, compared with placebo in the overall population and in patients with comorbid asthma (figure 1).

**Conclusion** Tezepelumab reduced levels of type 2 inflammatory biomarkers compared with placebo in adults with severe CRSwNP, both in the overall study population and in patients with comorbid asthma.

## S162 EMERGING BIOLOGIC USE MAY REDUCE THE NEED FOR CORTICOSTEROID TREATMENT AND SURGERY FOR CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSWNP) IN FRANCE, GERMANY, ITALY, SPAIN AND THE UK

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**Introduction and objectives** CRSwNP patients often face a prolonged and challenging treatment journey, influenced by regional variations in healthcare systems and treatment practices. This study characterised the clinical management and treatment patterns in CRSwNP patients in routine clinical practice in France, Germany, Italy and Spain (EU4); and the UK.

**Method** This study utilised data from the Adelphi Real World CRS Disease Specific Programme™, a cross-sectional survey,

**Abstract S162 Table 1** Clinical management and treatment patterns in patients with CRSwNP by country and patient groups

| | France (N=214) | | | Germany (N=210) | | | Italy (N=241) | | | Spain (N=214) | | | UK (N=171) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Random | Surgical | Biologic | Random | Surgical | Biologic | Random | Surgical | Biologic | Random | Surgical | Biologic | Random | Surgical | Biologic |
| **Top 5 most frequently currently prescribed CRSwNP treatments for patients in each country, n (%)** | | | | | | | | | | | | | | | |
| **N** | **83** | **59** | **39** | **99** | **67** | **35** | **94** | **69** | **40** | **90** | **66** | **37** | **75** | **55** | **28** |
| Topical nasal corticosteroids (spray) | 66 (80) | 45 (76) | 28 (72) | 82 (83) | 58 (87) | 18 (51) | 73 (78) | 49 (71) | 23 (58) | 59 (66) | 47 (71) | 23 (62) | 40 (53) | 35 (64) | 17 (61) |
| Nasal saline irrigation | 35 (42) | 33 (56) | 9 (23) | 46 (46) | 38 (57) | 11 (31) | 34 (36) | 18 (26) | 7 (18) | 33 (37) | 22 (33) | 8 (22) | 28 (37) | 20 (36) | 11 (39) |
| Monoclonal antibody (i.e., anti-IL-5 or IgE) | 16 (19) | 8 (14) | 39 (100) | 2 (2) | 1 (1) | 35 (100) | 19 (20) | 10 (14) | 40 (100) | 9 (10) | 3 (5) | 37 (100) | 0 | 0 | 28 (100) |
| Oral antihistamine | 25 (30) | 22 (37) | 10 (26) | 10 (10) | 8 (12) | 6 (17) | 30 (32) | 17 (25) | 10 (25) | 38 (42) | 22 (33) | 12 (32) | 22 (29) | 22 (40) | 10 (36) |
| Systemic corticosteroids (oral) | 17 (20) | 11 (19) | 5 (13) | 15 (15) | 5 (7) | 1 (3) | 24 (26) | 15 (22) | 8 (20) | 28 (31) | 18 (27) | 8 (22) | 9 (12) | 10 (18) | 6 (21) |
| **CRSwNP biologics, n (%)*** | | | | | | | | | | | | | | | |
| **N** | **16** | **8** | **39** | **2** | **1** | **35** | **19** | **10** | **40** | **9** | **3** | **37** | **0** | **0** | **28** |
| Dupilumab | 11 (69) | 4 (50) | 27 (69) | 2 (100) | 1 (100) | 24 (69) | 9 (47) | 5 (50) | 21 (53) | 4 (44) | 3 (100) | 13 (35) | 0 | 0 | 8 (29) |
| Mepolizumab | 3 (19) | 3 (38) | 9 (23) | 0 | 0 | 6 (17) | 10 (53) | 5 (50) | 11 (28) | 4 (44) | 0 | 16 (43) | 0 | 0 | 13 (46) |
| Omalizumab | 2 (13) | 1 (13) | 3 (8) | 0 | 0 | 5 (14) | 0 | 0 | 8 (20) | 1 (11) | 0 | 7 (19) | 0 | 0 | 7 (25) |
| **Surgery** | | | | | | | | | | | | | | | |
| **Sino-nasal surgery since CRSwNP diagnosis** | | | | | | | | | | | | | | | |
| **N** | **104** | **69** | **41** | **105** | **70** | **35** | **121** | **80** | **40** | **106** | **71** | **37** | **83** | **59** | **29** |
| Patients undergone surgery, n (%) | 25 (24) | 69 (100) | 12 (29) | 17 (16) | 70 (100) | 18 (51) | 28 (23) | 80 (100) | 16 (40) | 43 (41) | 71 (100) | 21 (57) | 19 (23) | 59 (100) | 9 (31) |
| **Symptoms** | | | | | | | | | | | | | | | |
| **Top 3 CRSwNP symptoms, n (%)** | | | | | | | | | | | | | | | |
| **N** | **104** | **69** | **41** | **105** | **70** | **35** | **121** | **80** | **40** | **106** | **71** | **37** | **83** | **59** | **29** |
| Nasal blockage | 84 (81) | 55 (80) | 29 (71) | 92 (88) | 65 (93) | 29 (83) | 101 (83) | 63 (79) | 35 (88) | 95 (90) | 69 (97) | 36 (97) | 80 (96) | 48 (81) | 23 (79) |
| Need to blow nose | 65 (63) | 44 (64) | 23 (56) | 65 (62) | 49 (70) | 20 (57) | 91 (75) | 62 (78) | 29 (73) | 68 (64) | 48 (68) | 20 (54) | 61 (73) | 45 (76) | 20 (69) |
| Runny nose | 74 (71) | 38 (55) | 24 (59) | 65 (62) | 44 (63) | 18 (51) | 73 (60) | 51 (64) | 21 (53) | 70 (66) | 48 (68) | 22 (59) | 43 (52) | 29 (49) | 18 (62) |
| **SNOT-22 score (0–5)** | | | | | | | | | | | | | | | |
| **N** | **13** | **8** | **4** | **66** | **43** | **20** | **34** | **12** | **2** | **36** | **14** | **9** | **17** | **11** | **5** |
| Mean (SD) | 2.1 (1.0) | 1.6 (1.0) | 0.7 (0.6) | 1.4 (0.8) | 1.3 (0.7) | 0.9 (0.9) | 1.8 (1.0) | 2.1 (1.5) | 0.6 (0.1) | 2.0 (1.1) | 2.0 (0.8) | 1.8 (1.1) | 1.7 (1.2) | 1.7 (1.1) | 1.9 (0.8) |

*No patients were prescribed benralizumab or tezepelumab, and one patient was prescribed reslizumab in the biologic group in Spain.

FESS, functional endoscopic sinus surgery; IgE, immunoglobulin E; IL, interleukin; SD, standard deviation; SNOT-22, Sino-Nasal Outcome Test-22.

with retrospective data collection, conducted in the EU4 and UK (Mar-Sep 2023). In total, 176 physicians completed a physician survey and patient record forms for 1050 patients with moderate-to-severe CRSwNP (random group), recurrent CRSwNP with ≥1 previous surgery for polyp removal (surgical group, SG) or severe CRSwNP prescribed biologics (biologic group, BG). Patients voluntarily completed self-reported surveys assessing CRSwNP impact using validated tools (e.g. Sino-Nasal Outcome Test-22 [SNOT-22]) and the Adelphi Adherence Questionnaire. Data were pseudo-anonymously linked, with all information recorded at the survey date from medical history or specific recall period.

**Results** Most patients across EU4 and UK were prescribed CRSwNP-specific treatments (79–95%). Topical nasal corticosteroids were the most prescribed across all countries (58–79%); oral corticosteroid (OCS) use appeared to be slightly lower in the BG (3–22%) versus SG (7–27%). Biologic use was low and varied greatly across countries, with fewest patients receiving biologics in the UK. While all patients in the SG had ≥1 surgery since CRSwNP diagnosis, notably fewer patients in the BG had undergone surgery (23–57%). In the BG, 84% of patients experienced nasal blockage (86% in SG), 62% felt the need to blow their nose (71% in SG) and 57% had a runny nose (60% in SG). Average SNOT-22 score was 1.22 in the BG and 1.57 in the SG, with lower SNOT-22 scores representing reduced CRSwNP-related health burden.

**Conclusion** This cross-sectional study emphasised the persistent OCS use in CRSwNP management, even as biologics are emerging as an alternative treatment option. Despite notable variation in biologic use across countries, the findings suggest a general trend wherein patients receiving biologics reported fewer symptoms and achieved lower SNOT-22 scores, indicative of better disease management.

## S163 MANAGING ABPA WITH TEZEPELUMAB: A SINGLE-CENTRE EXPERIENCE

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**Introduction** Allergic bronchopulmonary aspergillosis (ABPA) is a complex hypersensitivity reaction to *Aspergillus fumigatus*, predominantly seen in patients with asthma or cystic fibrosis. While systemic corticosteroids remain the mainstay of treatment, their cumulative and long-term use is associated a host of significant adverse effects, prompting the need for more targeted therapies. Monoclonal antibodies have emerged as promising alternatives by modulating key pathways of type II inflammation however, limited published literature exists about the response of ABPA patients to a novel anti-Thymic Stromal Lympopoetin (TSLP) biologic, Tezepelumab.

**Methods** A retrospective, single-centre analysis was conducted on all patients diagnosed with ABPA, as per the revised 2024 ISHAM criteria, who received Tezepelumab for at least 12 months. A clinical response to Tezepelumab was defined as a ≥ 50% reduction in oral corticosteroids (OCS) requiring exacerbations or a reduction in maintenance OCS of at least 50% over a 12-month treatment period. Asthma Control Questionnaire (ACQ-5) scores before and after treatment were also captured.

**Results** A total of 20 patients received Tezepelumab with baseline characteristics listed in table 1. 80% of patients had a clinical response to Tezepelumab at 12 months and 55% had MCID decrease of 0.5 in their ACQ scores at 12 months. There was a significant reduction in annual exacerbation rate

**Abstract S163 Table 1**

| Characteristic | |
|---|---|
| **Age (y), mean ± SD** | **60.4 ± 14** |
| **Female, n (%)** | **11 (55%)** |
| **Ethnicity, n (%)** | |
| - White British | 12 (60%) |
| - White others | 4 (20%) |
| - Asian British | 1 (5%) |
| - Not stated | 3 (15%) |
| **BMI, mean ± SD (kg/m$^2$)** | **28.2 ± 6** |
| **Lung function, mean ± SD** | |
| - $FEV_1$ (Litres) | 1.64 **±1** |
| - FVC (Litres) | 2.75 **± 1** |
| **Radiology (descriptive baseline phenotype) (n = 20), n (%)** | |
| - Bronchiectasis | 14 (70%) |
| - Varicose Bronchiectasis | 4 (20%) |
| - Mucous plugging | 8 (40%) |
| - Nodules | 3 (15%) |
| - Consolidation | 3 (15%) |
| - Mycetoma | 2 (10%) |
| **Serology** | |
| - Baseline IgE (IU/L), mean ± SD | 1532 ± 1623 |
| - Baseline IgE > 1000, n (%) | 8 (40%) |
| - Peak IgE > 500, n (%) | 20 (100%) |
| - Baseline *Aspergillus*-specific IgE (kUA/L), mean ± SD | 32 ± 51 |
| - Baseline *FeNO mean* ± SD | 30 ± 26 |
| **Baseline blood eosinophils (× 10$^9$/L), mean ± SD** | **0.36** ± 0.31 |
| **No. of patients on mOCS, n (%)** | **12 (60%)** |
| **Baseline mOCS dose (mg/d prednisolone), median (IQR)** | **7.5 (5)** |
| **Receiving azole therapy, n (%)** | **13 (65%)** |
| Itraconazole | 6 (30%) |
| Posaconazole | 4 (20%) |
| Isavuconazole | 4 (20% |
| Caspofungin | 1 (5%) |

from 4.0 (IQR 2.8) to 0.0 (IQR 1.25; p=0.0007, 95% CI −6.24 to −2.30) alongside a significant median ACQ score reduction from 2.17 (IQR 1.50) to 0.84 (IQR 1.46; p=0.0004, 95% CI −1.77 to −0.62). In comparison with a prior biologic study[1] conducted at our centre, the reduction in exacerbation rate with Tezepelumab was greater than that observed with anti-IgE therapy (2.0 to 1.0) and comparable to the reductions seen with Mepolizumab (4.0 to 1.0, $p<0.0001$) and Benralizumab (5.0 to 2.0, $p=0.0156$).

**Conclusions** Overall, Tezepelumab was associated with robust clinical improvement in repeated OCS requirement, including in those on mOCS, which was comparable to, and in some cases, better than other type II monoclonal therapies.

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## S164 MEPOLIZUMAB IMPROVES AND SUSTAINS ASTHMA CONTROL AND LUNG FUNCTION IN PATIENTS WITH SEVERE ASTHMA, INDEPENDENT OF BASELINE IGE AND FENO IN THE REAL WORLD: 2-YEAR RESULTS FROM REALITI-A

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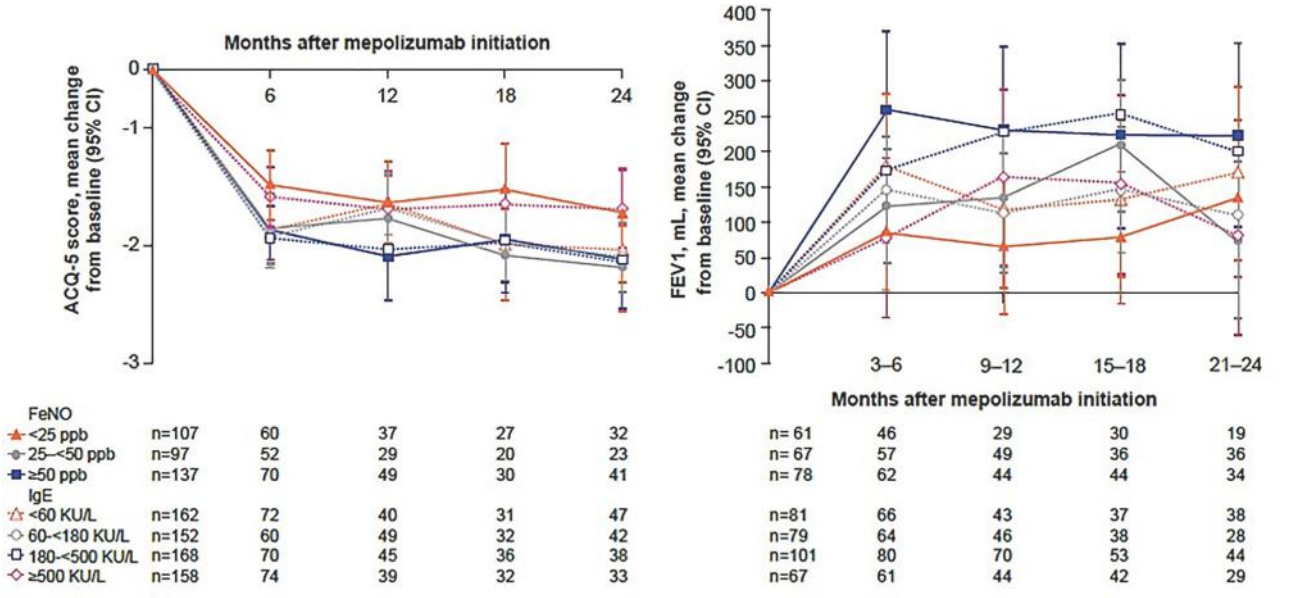
10.1136/thorax-2025-BTSabstracts.170

Introduction and objectives: REALITI-A study demonstrated mepolizumab effectiveness independent of immunoglobulin-E (IgE) and fractional exhaled nitric oxide (FeNO) levels in patients with severe asthma in preliminary 1-year analyses. However, additional longer-term real-world evidence on the effect of mepolizumab on asthma control and lung function may be useful to inform clinical decision making.

**Method** REALITI-A, a 2-year, observational, prospective study enrolled patients (≥18 years) with severe asthma, newly prescribed mepolizumab (100-mg subcutaneously). Changes from baseline in asthma control (Asthma Control Questionnaire-5 [ACQ-5] scores, minimal clinically important difference: 0.5) and lung function (forced expiratory volume in 1 second [$FEV_1$]) were assessed for baseline FeNO subgroups (<25, 25–50, ≥50 parts per billion [ppb]) and baseline IgE subgroups (<60, 60–<180, 180–<500, ≥500 KU/L) at 1-year and 2-years post-mepolizumab exposure.

**Results** ACQ-5 scores decreased across all FeNO subgroups 1-year post-mepolizumab exposure and were sustained at 2-years: <25 ppb (least squares [LS] mean change from baseline [95% CI]: -1.21 [-1.58, -0.85]), 25–<50 ppb ( -1.68 [-2.10, -1.25]), ≥50 ppb (-1.61 [-1.89, -1.34]; figure 1). ACQ-5 scores also decreased across all IgE subgroups at 1-year and were sustained at 2-years: <60 KU/L (-1.53 [-1.82, -1.24]), 60–<180 KU/L (-1.59 [-1.90, -1.27]), 180–<500 KU/L (-1.64 [-1.96, -1.31]), ≥500 KU/L (-1.19 [-1.53, -0.84]). $FEV_1$ improved at Months 9–12 post-mepolizumab exposure for FeNO <25 ppb and ≥50 ppb subgroups and was sustained at Months 21–24 (LS mean change from baseline [95% CI]: 133.1 [22.7, 243.6]; 220.6 [98.0, 343.2], respectively). $FEV_1$ also improved at Months 9–12 post-mepolizumab exposure for IgE <60 KU/L, 60–<180 KU/L and 180–<500 KU/L, and was sustained at Months 21–24 (LS mean change from baseline [95% CI]: 167.6 [81.1, 254.1], 108.8 [-31.7, 249.3], 198.1 [107.7, 288.6], respectively). $FEV_1$ improvements were seen to a lesser extent in FeNO 25–<50 ppb subgroup at 2-years (72.2 [-57.6, 202.1]) and IgE ≥500 KU/L subgroup at Months 21–24 (82.0 [-60.7, 224.8]).

**Conclusion** Treatment with mepolizumab in a real-life setting improves asthma control and lung function after 1-year, with




ACQ-5, Asthma Control Questionnaire-5; CI, confidence interval; ppb, parts per billions; $FEV_1$, forced expiratory volume in 1 second.

**Abstract S164 Figure 1** Asthma control imporves and is sustained for 2 years for all FeNO and IgE categories (left panel) and lung function improves for all FeNO and IgE categories at 2 years versus baseline, with some fluctuation in FEV1 for some categories over 2 years (right panel). Error bars represent 95% CI.

improvements sustained to 2-years, independent of baseline FeNO and IgE.

**Funding** GSK (204710).

# The role of neutrophils in respiratory disease

## S165 ACUTE SYSTEMIC HYPOXIA ALTERS CIRCULATING NEUTROPHIL BIOMECHANICAL AND TRANSCRIPTIONAL PHENOTYPE

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10.1136/thorax-2025-BTSabstracts.171

**Background** The severe high altitude illness, High Altitude Pulmonary oEdema (HAPE), occurs in some individuals exposed to hypobaric hypoxia and is characterised by neutrophil-driven lung and microvascular damage. Ultrastructural examination of lung tissue from HAPE patients shows neutrophils trapped in pulmonary capillaries. Hypoxia delays neutrophil apoptosis and increases degranulation *in vitro*, and delayed apoptosis is also seen in neutrophils from subjects who experienced 7-days systemic hypoxia at high altitude. However, the exact role of hypoxia in HAPE pathogenesis, and its impact on neutrophils *in vivo*, is poorly understood. We assessed the impact of short-term systemic hypoxia on neutrophil phenotype.

**Methods** Venous blood was collected from healthy volunteers (n=10) pre/post exposure to normobaric hypoxia (approximately 12.8% oxygen) for two hours. *Ex vivo* blood/cells were maintained at venous oxygen tension (5% oxygen) throughout. Whole blood neutrophil biomechanical profiles were measured using Real-Time Deformability Cytometry (RT-DC). Gene transcription was evaluated by RT-qPCR. Apoptosis was measured via cytospin microscopy at 24 hours. Neutrophil secretion of elastase and myeloperoxidase was assessed by activity assay.

**Results** Neutrophils exposed to pathological hypoxia (1%) *in vitro* are stiffer than those maintained at venous oxygen levels (p=0.045). Following systemic hypoxia, neutrophil number (p=0.0195) and porosity (membrane roughness, p=0.0273) were increased. Overall, neutrophils became slightly smaller and softer post-hypoxia (non-significant), with notable inter-donor variability. Expression of Hypoxia Inducible Factor (HIF)-dependent genes, GLUT1, BNIP3 and VEGF, was unchanged but CXCR4 gene expression was increased post-hypoxia (p=0.004), and higher CXCR4 expression was associated with smaller cell size ($R^2$=0.71, p=0.004). Neutrophil apoptosis remained unchanged post-hypoxia, as did secretion of elastase/myeloperoxidase, although inter-donor variability was again noted.

**Discussion** As expected, following this short *in vivo* hypoxic exposure, we did not see a HIF-dependent effect on neutrophil gene transcription or apoptosis. However, the blood neutrophilia and increased porosity suggest a primed (partially activated) phenotype. CXCR4 is known to be upregulated in aged pro-inflammatory neutrophils. Thus, higher CXCR4 expression in smaller cells may indicate enhanced endothelial

transmigration and lung injury potential. Inter-donor variability in neutrophil biomechanical and secretion profiles in response to hypoxia may, at least partly, explain the variable individual susceptibility to HAPE.

## S166 DIFFERENTIAL ROLES OF THE INTEGRINS CD11B AND CD11C IN HUMAN NEUTROPHIL EXTRAVASATION ACROSS PULMONARY AND SYSTEMIC ENDOTHELIUM

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10.1136/thorax-2025-BTSabstracts.172

**Introduction and Objectives** CD11b and CD11c are key components of the integrins Mac-1 and CR4, respectively. During inflammation, CD11b and CD11c are upregulated on the cell surface of neutrophils. Previous reports suggest both CD11b and CD11c are involved in neutrophil extravasation, but there are few data regarding human neutrophils. We hypothesised that the roles of CD11b and CD11c may vary between the pulmonary and systemic circulations.

**Methods** CD34+ haematopoietic stem cells (HPSCs) were isolated from human apheresis cones by magnetic selection and expanded. Cas9-gRNP complexes were nucleofected into the HSPCs resulting in >90% knockdown of *ITGAM* and *ITGAX* at genomic and protein levels. After subsequent directed differentiation into neutrophils in the presence of recombinant human granulocyte colony stimulating factor, CD11bKO (N = 7), CD11cKO (N = 7), and donor-matched non-targeting (scrambled) control cells (SCR) were flowed over confluent TNF-stimulated pulmonary and dermal (systemic) microvascular endothelial monolayers. Phase-contrast microscopy time-lapse images were obtained, and the phases of extravasation (rolling, adhesion, crawling and transmigration) were quantified using ImageJ, with crawling mechanics assessed using TrackMate.

**Results** Compared to SCR control neutrophils, fewer CD11bKO neutrophils engaged in rolling ($p = 0.047$) and crawling ($p = 0.031$) when interacting with pulmonary, but not systemic, microvascular endothelial cells. CD11bKO neutrophils also displayed increased crawling linearity ($p = 0.031$) when interacting with pulmonary microvascular endothelial cells.

No changes in neutrophil-endothelial interactions were observed with CD11cKO cells, when were flowed across both pulmonary and systemic microvascular endothelial cells. Similarly, no difference in crawling mechanics was seen with knockout of CD11c.

**Conclusion** We have shown for the first time in human neutrophils that CD11b mediates crawling and rolling across pulmonary, but not systemic, microvascular endothelial cells, and that CD11c is dispensable for human neutrophil extravasation in the pulmonary and systemic circulations. Our data supports the hypothesis that there is variation across endothelial beds in the requirement for integrins during human neutrophil extravasation.

## S167 A PROSPECTIVE OBSERVATIONAL STUDY INVESTIGATING NEUTROPHIL DYNAMICS IN IDIOPATHIC PULMONARY FIBROSIS

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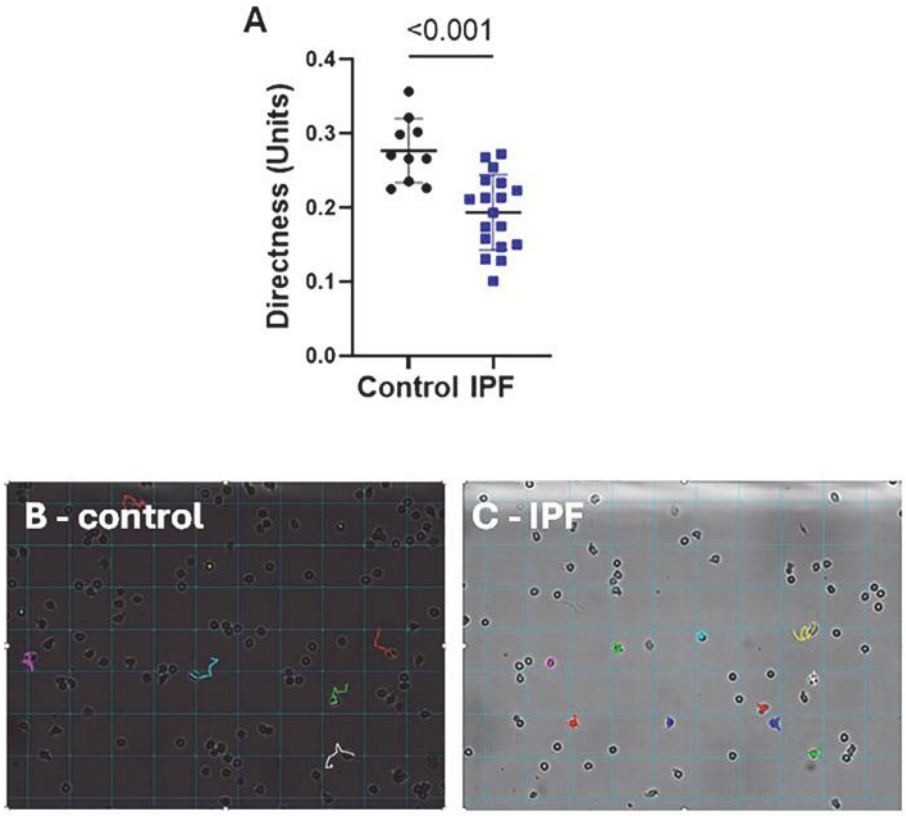
10.1136/thorax-2025-BTSabstracts.173

**Introduction and Objectives** Idiopathic pulmonary fibrosis (IPF) is characterised by a dysregulated repair response to alveolar epithelial damage. Neutrophils house an array of enzymes critical for repair and remodelling. Neutrophil counts are associated with IPF severity, and neutrophil extracellular traps are observed in fibrotic lung tissue.[1] However, the role of neutrophils in IPF remains unknown. This prospective study aimed to compare the phenotype and movement of circulating neutrophils derived from IPF patients to age-matched control participants.

**Methods** Thirty-five IPF and 19 control participants were recruited. Neutrophil and immature granulocyte counts were measured in venous blood using the Sysmex XN-1000 analyser. Neutrophils were isolated via Percoll density gradient centrifugation and cell surface markers analysed by flow cytometry. Plasma levels of Aα-Val$^{541}$, an indirect measure of proteinase 3 activity, were quantified by DELFIA.[2] Chemotaxis was measured by real-time microscopy using the Insall chamber assay.

**Results** Peripheral neutrophil counts were greater in IPF patients than controls ($4.5x10^9$/L versus $3.0x10^9$/L, $p<0.001$). IPF patients had greater immature granulocyte counts ($p<0.01$) and a lower percentage of CD10$^{positive}$ (maturity marker) neutrophils than controls ($p=0.03$). CXCR4 (chemokine receptor) and PD-L1 (co-inhibitory molecule) expression was higher in IPF-derived neutrophils ($p<0.05$). Plasma Aα-Val$^{541}$ levels were higher in IPF than control participants ($p=0.03$). Movement responses to the chemoattractants, IL-8 and fMLP were comparable, but IPF neutrophils moved less directly than controls ($p<0.001$) on exposure to the pro-fibrotic growth factor, 10 ng/mL transforming growth factor-β 1 (TGFβ1) (figure 1).

**Conclusions** IPF patients appear to have an expanded population of immature neutrophils, suggestive of emergency granulopoiesis. Dysregulated movement of IPF neutrophils in response to TGFβ1 suggests that neutrophil dynamics may be altered within the fibrotic microenvironment. Furthermore, elevated levels of the fibrinogen cleavage epitope, Aα-Val$^{541}$, implies increased neutrophil degranulation within the interstitum of IPF patients. Ongoing investigation into the




**Abstract S167 Figure 1** **(A)** Directness of neutrophil movement upon exposure to 10 ng/mL TGFβ1 assessed using the Insall chamber assay. **(B-C)** Representative microscopy images displaying tracks (lines) of neutrophil movement in response to TGFβ1 (20X magnification)

interaction between IPF neutrophils and pro-fibrotic mediators may identify a much-needed novel avenue of therapeutic manipulation.


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## S168 NEUTROPHILS DISPLAY PHENOTYPIC AND FUNCTIONAL CHANGES INDICATIVE OF ACTIVATION IN A NOVEL IN VITRO MODEL OF IDIOPATHIC PULMONARY FIBROSIS


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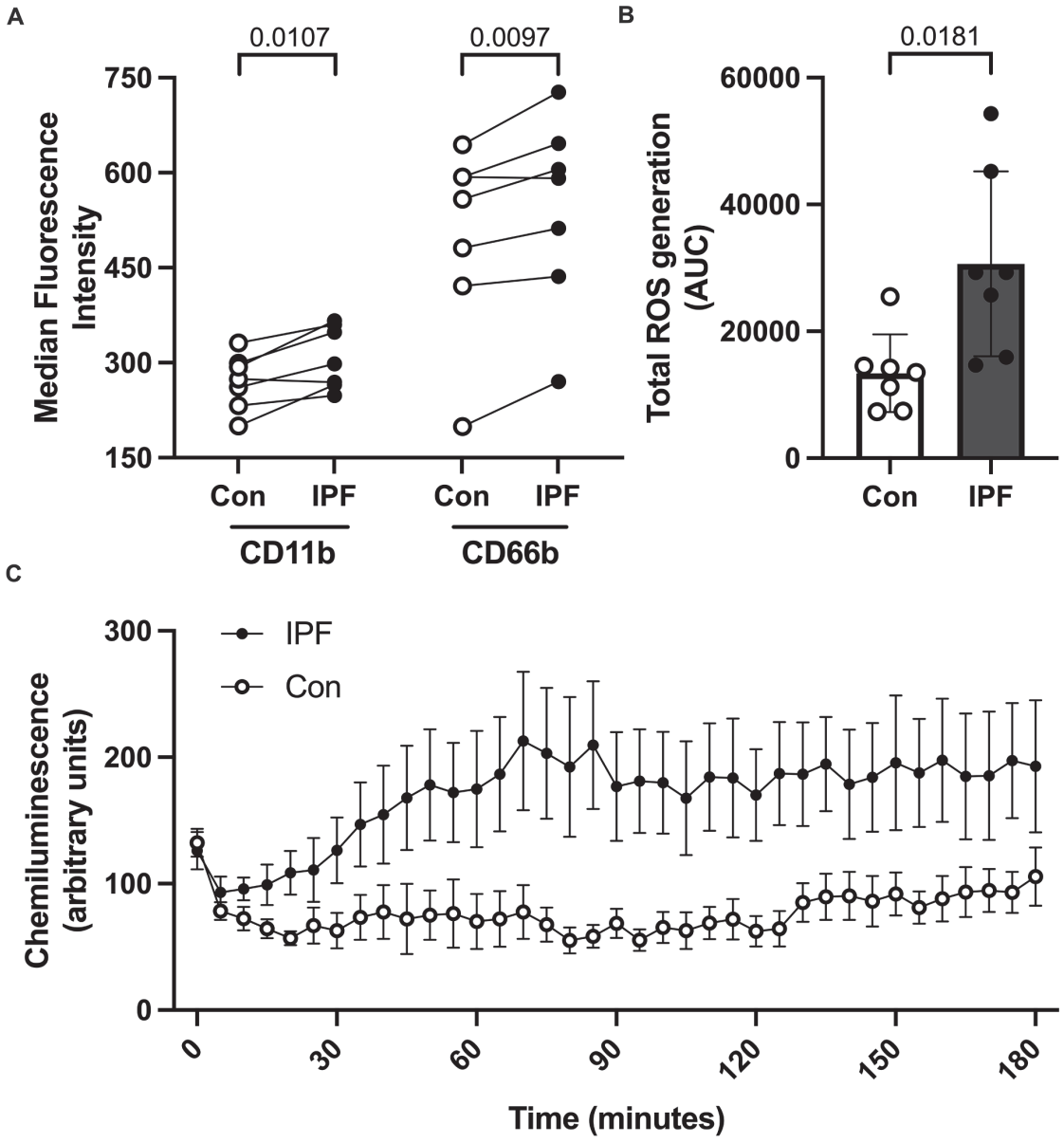

10.1136/thorax-2025-BTSabstracts.174

**Background** Neutrophil count and neutrophil extracellular trap (NET) markers are elevated within bronchoalveolar lavage fluid (BALF) of patients with Idiopathic Pulmonary Fibrosis (IPF).[1] [2] However, the activity of neutrophils within the alveolar spaces of IPF lung tissue is undetermined, as is its contribution to fibrosis. We hypothesised that the fibrotic alveolar microenvironment promotes neutrophil survival and activation, with accompanying enhanced respiratory burst and NET formation. This was investigated using a novel *in vitro* model of IPF.

**Methods** Neutrophils, isolated from venous blood of healthy donors using Percoll density-gradient centrifugation, were incubated in pooled BALF collected either from IPF patients (n=5) or age matched control participants (n=3). Flow cytometric analyses of neutrophil viability (PI/Annexin V staining) and cell surface activation marker (CD11b, CD66b, CD62L) expression were performed. Reactive oxygen species (ROS) generation and NET formation were assessed using luminol-amplified chemiluminescence and fluorometric analysis of SYTOX$^{TM}$ green stained DNA, respectively.

**Results** Fewer neutrophils entered early apoptosis during 3 hours of incubation with IPF BALF compared to control BALF ($p = 0.018$). IPF BALF exposure increased cell surface expression of the activation marker CD11b ($p = 0.011$) and degranulation marker CD66b ($p < 0.001$), compared to control BALF. CD62L expression was equivalent following exposure to control and IPF BALF ($p > 0.05$). IPF BALF exposure increased total neutrophil ROS generation over 3 hours, compared to control BALF ($p = 0.018$). Despite protein-normalised IPF BALF samples containing higher concentrations of cell free DNA (a NET marker) compared to protein-normalised control BALF samples ($p = 0.036$), NET formation by neutrophils was equivalent following exposure to pooled IPF and control BALF ($p > 0.05$).

**Conclusions** This study demonstrates that IPF BALF promotes a phenotypic shift in neutrophils towards an activated state. This corresponds with a decrease in neutrophil apoptosis and an augmented respiratory burst. The implications of these findings in IPF's pathogenesis and progression warrant further investigation. Namely, exploration of the impact of enhanced neutrophil ROS generation on TGF-β activation within the fibrotic lung is vital. Identification of neutrophil-activating components of IPF BALF may enable therapeutic modulation of neutrophil function in IPF.




**Abstract S168 Figure 1** **(A)** Flow cytometric analysis of neutrophil activation marker (CD11b and CD66b) expression following exposure to pooled IPF and control BALF. **(B)** Summary histogram and **(C)** representative trace of luminol-amplified chemiluminescence experiments assessing neutrophil ROS generation (AUC – area under the curve)


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## S169 INVESTIGATING THE EFFECT OF BARICITINIB ON NEUTROPHILIC INFLAMMATION IN AN EX VIVO LUNG PERFUSION (EVLP) MODEL OF ACUTE RESPIRATORY DISTRESS SYNDROME


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10.1136/thorax-2025-BTSabstracts.175


**Background** Acute respiratory distress syndrome (ARDS) is driven by uncontrolled neutrophilic inflammation in the alveolar space with resultant damage to the epithelial and endothelial barrier, resulting in pulmonary oedema. Baricitinib is a JAK1/2 inhibitor which inhibits inflammation in experimental lung injury in animal models and reduces mortality in COVID related respiratory failure. The effect of baricitinib on human lung injury outside of COVID is unknown.

**Hypothesis** JAK1/2 inhibition with baricitinib reduces LPS (lipopolysaccharide)-induced lung injury in ex vivo perfused and ventilated human lungs.

**Methods** Human lungs unsuitable for transplantation, for which there was consent for use in research, were ventilated and perfused ex vivo, using a modified Toronto protocol. Lungs with intact alveolar fluid clearance at baseline were injured by instilling 6 mg LPS (E coli) into a lobe and adding whole blood to the perfusate at a final concentration of 1/10. Lungs were randomised to receive either baricitinib or placebo in the perfusate (final concentration baricitinib=50ng/ml to correspond with Cmax obtained in healthy volunteers receiving 4 mg/day, the standard dose of baricitinib, and that used

in the treatment of COVID in RECOVERY). Bronchoalveolar lavage was carried out at 4 hours after LPS instillation. Total cell count in BAL was measured using an Eve Automated cell counter (NanoEntek) and differential white cell count carried out on cytospins prepared from BAL. Ethical approval was obtained from NRES (REC 14 LO 0250) and Queen's University of Belfast School of Medicine Ethics Committee (SREC14/08).

**Results** 13 pairs of lungs (26 in total) were obtained. Baseline fluid clearance was impaired in 3 lungs and these were excluded from the study. 12 lungs were randomised to receive placebo and 11 baricitinib. Baricitinib reduced BAL neutrophil count at 4 hours from median 5.76 (IQR 2.74–9.16) $x10^4$/ml to 2.02 (IQR 1.73–4.73) $x10^4$/ml (figure 1), *p=0.0489, Wilcoxon rank-sum test. The effect of baricitinib on markers of permeability, alveolar epithelial and endothelial injury will be measured.

**Conclusion** Baricitinib reduces neutrophil count in the alveolar space in a human EVLP model of LPS-induced lung injury, supporting its potential to inhibit alveolar neutrophilic inflammation in non-COVID related ARDS.

## S170 NEUTROPHIL ELASTASE AND NETS CONTRIBUTE TO DISEASE EXACERBATION IN PLEURAL INFECTION

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10.1136/thorax-2025-BTSabstracts.176

**Background** Pleural infection is a severe and complicated disease with increasing worldwide incidence. Despite being recognised for decades, currently there is only one set of guidelines for pleural infection management. Clinical outcomes remain poor and validated biomarkers to predict disease progression or response to treatment are not available. In addition to Plasminogen Activator Inhibitor 1 (PAI-1) and Soluble Urokinase Plasminogen Activator Receptor (suPAR), neutrophil elastase (NE) recently emerged as a potential fibrinolytic deficiency biomarker as it has been suggested to inhibit fibrinolysis by degrading plasminogen and thereby increasing the likelihood of septations.

**Aim** To investigate the effect of neutrophil elastase and neutrophil extracellular traps (NETs) on intrapleural fibrinolytic deficiency using fresh specimens prospectively collected from pleural infection patients at baseline and post-intrapleural fibrinolytic treatment. Also to explore the potentiality of NE and NETs as biomarker for disease management and patient stratification.

**Results** We found a negative correlation between the protein level of intrapleural neutrophil elastase and plasminogen ($r$= -0.4214, $p$= 0.0178*). Pharmacological inhibition of NE reduced plasminogen degradation (~24% reduction, $p$= 0.0372*), confirming the involvement of NE in the fibrinolytic deficiency. Pleural infection patients with significantly higher levels of NETs (NE-DNA) in their pleural fluid were more prone to develop septations ($p$= 0.0032**) and showed poorer one-year survival rate ($p$= 0.00075***). Cox regression revealed that each 1000-unit increase in NETs was associated with a hazard ratio of 1.16 (95% CI:1.02–1.32, $p$= 0.0168*). Intrapleural fibrinolytic treatment promotes fibrinolytic potential and reduces the level of NETs and neutrophil-derived proteins such as NE in pleural fluid.

**Conclusion** In conclusion, our data demonstrates the contribution of NE and NETs in intrapleural fibrinolytic deficiency. Both pose as potential important targets for disease management.

# How the eosinophil and T2 biomarkers guide decisions in COPD

## S171 CLINICAL UTILITY OF FENO IN A TERTIARY COPD CLINIC DR FIONA HICKEY, MS MICHELE CUDDIHY, PROF BREDA CUSHEN

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10.1136/thorax-2025-BTSabstracts.177

Blood eosinophil count is established as a biomarker to identify those COPD patients with an eosinophilic phenotype. These patients are most likely to respond to inhaled corticosteroids. Recent evidence supports a role for anti-eosinophilic biologic therapy in reducing exacerabations in this subgroup. FeNO is commonly used to identify Type 2 airway inflammation in asthma, but its role in COPD is uncertain. We examined the utility of measuring FeNO in a severe COPD clinic.

A retrospective review of the records of patients attending a tertiary COPD clinic was performed, and those with FeNO recorded were identified. The prevalence of FeNO≥25ppb, and FeNO ≥50ppb was established. The relationship between FeNO measurement and baseline demographic and clinical data was examined.

Data from 82 patients was included. The mean (sd) age was 56 (9) years; 55% were female. All patients had post-bronchodilator airflow obstruction with mean FEV1 54 (22.5)%, and high symptom burden, mean CAT 20.3 (8.2). The majority were ex-smokers (71%). Mean FeNO measured 19.6 (14.2) ppb, and peak blood eosinophil count 0.28 (0.22) cells/mm 3, in the previous year. An ICS was prescribed in 79%. Only 7% had a diagnosis of co-existent asthma.

An elevated FeNO≥50ppb was present in 5% of patients; 29% had a FeNO≥25ppb. There was an inverse relationship between FeNO and age. FeNO levels were higher in ex-smokers; FeNO >25 in 38% compared to 8% in current smokers ($p < 0.01$). One quarter of those prescribed ICS recorded FeNO≥25ppb. There was no significant relationship between FeNO and lung function, previous diagnosis of asthma, CAT score, or peak eosinophil count.

Almost one-third of patients referred to a tertiary severe COPD clinic have FeNO ≥25ppb suggesting FeNO is a useful adjunct in the assessment of COPD patients, regardless of smoking status, and ICS prescription. As is seen in asthma, FeNO and eosinophil count are independent biomarkers in this cohort. Integrating FeNO assessment into COPD care may enhance therapeutic precision but requires consideration of clinical context to maximise its effectiveness. More studies are needed to examine whether FeNO response to ICS differs in COPD patients as compared to those with asthma.

## S172 DIGITALLY MONITORED INHALED CORTICOSTEROIDS ADHERENCE PREDICTS CLINICAL OUTCOMES IN COPD STRATIFIED BY TYPE 2 INFLAMMATION: A COMPARATIVE ANALYSIS OF BLOOD EOSINOPHILS AND FENO

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10.1136/thorax-2025-BTSabstracts.178

**Introduction** COPD patients with type 2(T2) inflammation respond better to inhaled corticosteroids (ICS). However, it is unclear whether there is a difference in assessing treatment response using fractional exhaled nitric oxide (FeNO) or blood eosinophil count (BEC), particularly in the context of variable ICS adherence.

**Aim** To evaluate the impact of ICS adherence on exacerbations among patients with COPD, stratified by T2 inflammation using BEC and FeNO.

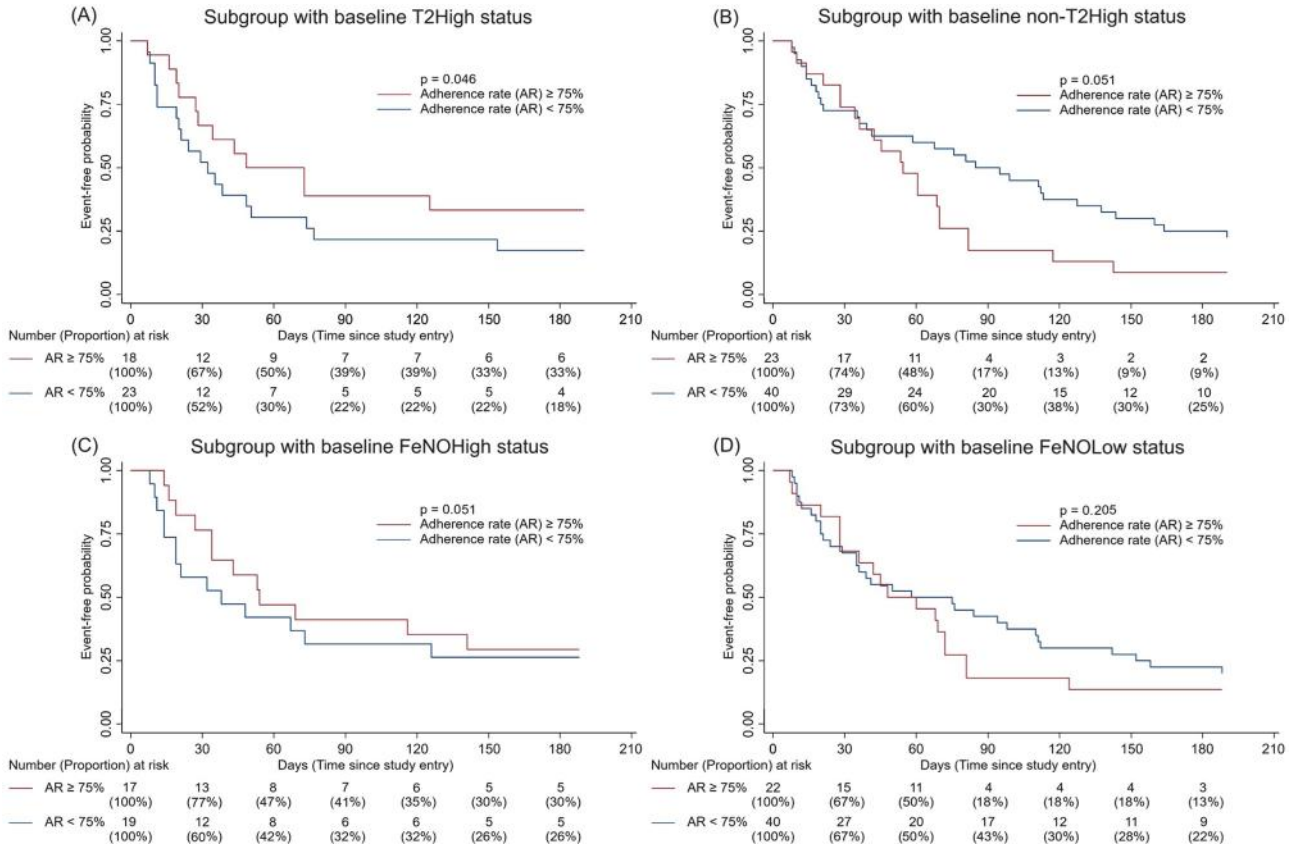
**Method** A six-month, single centre prospective cohort study (23/YH/0041) was conducted at Glenfield Hospital, Leicester, UK, targeting COPD patients receiving triple inhaled therapy with ≥2 exacerbations in the preceding year. Inhaler use was digitally monitored to objectively measure adherence. Participants were stratified by T2 inflammatory status using BEC (T2High: BEC $\geq 0.3\times10^9$/L) or non-T2High, FeNOHigh (≥20 ppb) or FeNOLow, and adherence level (AdhHigh ≥75% or AdhLow <75%). Primary outcomes included exacerbation frequency, time to first exacerbation (TTFE) and health-related quality of life via St. George's Respiratory Questionnaire (SGRQ) and COPD Assessment Test (CAT).

**Results** 104 patients (mean age 67±9 yrs and FEV1=1.1 ±0.5L) were included. The T2High-AdhHigh group (n=18) had significantly longer TTFE (HR=0.36,95%CI:0.14–0.95, p=0.046) and lower risk of hospitalisation (OR=0.14,95%CI: 0.03–0.72, p=0.02) than T2High-AdhLow group (n=23). Similarly, FeNOHigh-AdhHigh group(n=17) showed longer TTFE (HR=0.48,95%CI:0.22–1.01, p=0.051) and lower risk of hospitalisation (OR=0.06,95%CI: 0.01–0.68, p=0.02) than FeNOHigh-AdhLow group(n=19). Regardless of adherence, no difference in the hospitalisation risk was observed in FeNO-Low (n=62) or non-T2High(n=63) groups. High adherence in non-T2High participants led to a trend towards shorter TTFE (HR=1.76, p=0.05).

In the T2High group, low adherence was associated with worsening mean (SD) SGRQ scores from 62.9 (16.3) at baseline to 67.2 (13.9) at the final visit (p=0.03), and CAT scores, 24.5(7.9) to 26.8(6.4), p=0.009, exceeding the minimum clinically important difference. In contrast, high-adherence T2High and FeNOHigh groups maintained stable health status. Among non-T2High and FeNOLow participants, high adherence was paradoxically associated with increasing SGRQ and CAT scores.

FeNO and BEC demonstrated poor concordance (Cohen's κ = -0.06), suggesting they identify distinct groups of T2 inflammation.

**Conclusion** Objective ICS adherence influenced clinical outcomes differently by T2 phenotypes. FeNO was as predictive as BEC and may serve as a bedside tool.




**Abstract S172 Figure 1**

## S173 EOSINOPHILIC PHENOTYPE AND BACTERIAL LOAD IN HOSPITALISED PATIENTS WITH EXACERBATIONS OF COPD

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10.1136/thorax-2025-BTSabstracts.179

**Background** COPD is a heterogenous disease, and the eosinophilic phenotype is now well recognised as a treatable trait. However, it is less well known as to what extent bacterial infections affect this group of patients.[1]

**Aim** Our primary aim was to look at the incidence of bacterial growth in eosinophilic and the non-eosinophilic phenotype within our cohort of patients with COPD.

**Method** A retrospective analysis was conducted on patients coded has having been admitted to our Trust with COPD exacerbations from October 2020 to April 2021. Historic sputum culture results were collected from our web-based patient portal. Patients were included in the analysis if they had a sputum culture showing bacterial growth at any time. Eosinophilic phenotypes (EP) were defined as having a blood eosinophil count $\geq 0.3x10^9$/L and non-eosinophilic phenotypes (NEP) as having a blood eosinophil count$<0.3x10^9$/L.

**Results** In the study period, 337 unique patients were admitted with COPD exacerbations. They had a mean age (SD) of 73 (9) years, 49.6% (167/337) were female and 64.1% (216/337) were EP.

47% (n=157/337) patients had at least one positive sputum culture. Of these, 68.8% (n=108/157) were classified as EP.

72%(n=108/150) of EP had a positive sputum culture compared to 70% (n=49/70) of NEP; Odds Ratio 1.10 (95% CI 0.59–2.06); Chi-Square 0.021; p=0.88.

When compared, NEP had higher burden of H. Influenzae, Strep Pneumoniae and Moraxella (59%, 20% and 20% vs 55%, 19% and 15%, respectively) whereas EP had a higher burden of Coliforms, Pseudomonas sp and S.aureus (32%, 30% and 15% v 25%, 25% and 8%, respectively).

**Conclusion** Our findings suggest that in COPD patients requiring hospital admission, there is no significant difference between the bacterial burden of EP and NEP. Hence, future treatments of EP should not only include biologics but also focus on the role of bacteria in preventing exacerbations.

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## S174 REDUCTION OF EXACERBATIONS ACCORDING TO TYPE 2 INFLAMMATORY BIOMARKERS WITH DUPILUMAB TREATMENT IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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10.1136/thorax-2025-BTSabstracts.180

**Purpose** In BOREAS and NOTUS, add-on dupilumab reduced moderate-or-severe exacerbation rates and improved lung function in patients with chronic obstructive pulmonary disease (COPD) and type 2 inflammation. This post hoc analysis explored the predictive value of baseline blood eosinophil counts and fractional exhaled nitric oxide (FeNO) for response to dupilumab treatment in patients with COPD and type 2 inflammation.

**Methods** BOREAS (NCT03930732) and NOTUS (NCT04456673), Phase 3, randomized, placebo-controlled trials, enrolled 1874 patients (aged 40–85) COPD with moderate-to-severe airflow obstruction, and type 2 inflammation (screening blood eosinophil count $\geq$300 cells/mL) on triple inhalation therapy. Patients received dupilumab 300 mg every two weeks (q2w) or placebo for 52 weeks. Treatment rate ratios of dupilumab vs placebo and adjusted annualized moderate-or-severe exacerbation rates over a range of type 2 biomarkers were evaluated.

**Results** The treatment rate ratio for moderate-or-severe exacerbations was 0.70 (95% confidence interval [CI]:0.61–0.81), 0.71 (0.61–0.83), 0.71 (0.61–0.83), and 0.72 (0.61–0.83) for patients with baseline blood eosinophil counts of 300, 500, 700, or 900 cells/µL, respectively (continuous biomarker-by-treatment interaction $P$=0.09). Reductions in exacerbation rates were observed over a range of baseline blood eosinophil counts (dupilumab: 0.57 [95% CI:0.51–0.63] for 300 cells/mL to 0.58 [0.51–0.65] for 900 cells/mL]; placebo: 0.81 [0.73–0.89] for 300 cells/mL to 0.81 [0.73–0.89] for 900 cells/mL). Furthermore, the treatment rate ratio for moderate-or-severe exacerbations was 0.69 (95% CI:0.59–0.80) and 0.67 (0.53–0.85) for patients with baseline immunoglobulin E (IgE) levels 100 IU/mL and 1000 IU/mL, respectively (continuous biomarker-by-treatment interaction $P$=0.83); reductions in exacerbation rates were observed regardless of baseline IgE levels (dupilumab: 0.56 [95% CI:0.50–0.63] for 100 IU/mL to 0.53 [0.44–0.63] for 1000 IU/mL; placebo: 0.82 [0.74–0.90] for 100 IU/mL to 0.78 [0.67–0.92] for 1000 IU/mL). Reductions in treatment rate ratio for exacerbations were also observed with increasing baseline FeNO levels from 0.69 (95% CI:0.60–0.80) for 20 parts per billion (ppb) to 0.56 (0.46–0.69) for 40 ppb (continuous biomarker-by-treatment interaction $P$=0.006), indicating a significant predictive value.

**Conclusion** Dupilumab vs placebo reduced exacerbations independently of baseline blood eosinophil counts and IgE. Higher baseline FeNO levels were associated with a greater exacerbation reduction with dupilumab treatment compared with placebo.

## S175 TRACKING EOSINOPHILS FOLLOWING PREDNISOLONE OR BENRALIZUMAB AT THE TIME OF AN EXACERBATION IN THE ABRA TRIAL

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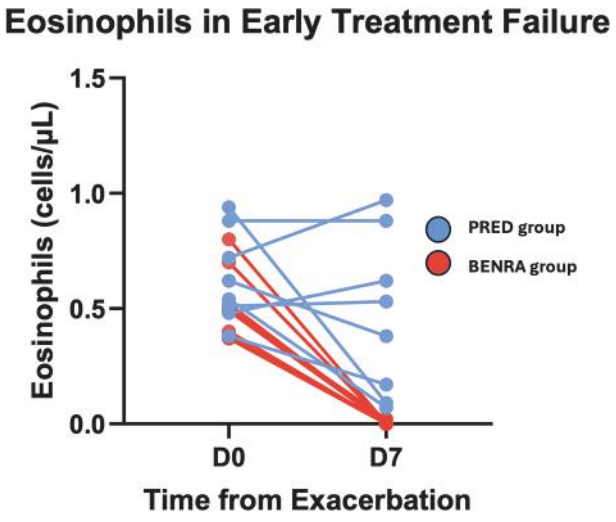
10.1136/thorax-2025-BTSabstracts.181

**Background** Prednisolone is the standard of care for treatment of exacerbations of asthma and COPD and has been shown to deplete eosinophil counts in severe asthma at stable state

(Moran, AJRCCM 2020). The ABRA trial was a multicentre, phase 2, double-blind, placebo-controlled trial. Eosinophilic exacerbations (eosinophils ≥300 cells/μL) were randomised 1:1:1 to: (1) benralizumab + placebo (BENRA), (2) placebo + prednisolone (PRED), or (3) benralizumab + prednisolone (BENRA+PRED).

**Objective** We explored the trajectory of the eosinophil count in ABRA patients who experienced early treatment failure compared to those who did not.

**Results** Early treatment failure (treatment failure within the first 5 days), occurred in 17 (19.5%) of patients (n=4 (23%) BENRA; n=5 (29%) BENRA+PRED; n=8 (47%) PRED). The pooled BENRA group demonstrated a mean difference of -0.514 (95%CI -0.627 to -0.402; p= <0.001) in eosinophils, whereas PRED group demonstrated a mean change of -0.170 (95%CI-0.469 to 0.129, p=0.221, see figure 1) between exacerbation and day 7. Within the group of patients for whom no treatment failure occurred, eosinophils within the pooled BENRA group demonstrated a mean change of -0.646 (95%CI -0.75 to -0.54, p≤0.001) and within the prednisolone group -0.221 (95%CI -0.53 to 0.09, p=0.143).




**Abstract S175 Figure 1** Eosinophils count in PRED group vs pooled BENRA group at exacerbation (D0) and first clinic visit (D7)

**Conclusion** Prednisolone, unlike benralizumab had no effect on reducing eosinophil count during eosinophilic exacerbations. Whether this relates to treatment failures needs to be further elucidated.

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## S176 A SUBGROUP ANALYSIS OF COPD-HELP – MEPOLIZUMAB FOR COPD WITH EOSINOPHILIC PHENOTYPE FOLLOWING HOSPITALISATION – STRATIFIED BY EOSINOPHIL LEVELS

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10.1136/thorax-2025-BTSabstracts.182

**Background** Mepolizumab, a monoclonal antibody to interleukin-5, has been shown to reduce exacerbations (Sciurba, *NEJM* 2025) in people with chronic obstructive pulmonary disease (COPD) and increased blood eosinophils in the past year, but did not improve time to readmission or death following hospitalisation (Flynn, *NEJM Evidence* 2025). We hypothesised that eosinophil count at index admission and functional capacity influence the effect of mepolizumab in this hospitalised population.

**Methods** We performed post-hoc analyses on data from COPD-HELP, a double-blind, placebo-controlled, randomised trial where 238 patients hospitalised with AECOPD and evidence of eosinophilic phenotype (blood eosinophil count ≥300 cells/μL at least once in prior year) were randomised to mepolizumab 100 mg or placebo for 48 weeks. All participants had extended MRC dyspnoea score (eMRC [range: 1–5b]) ≥2, were on inhaled corticosteroids and had significant smoking history. Primary outcome was time to rehospitalisation or death. Cox proportional hazards models were fitted for the overall population and the subgroup with eMRC 2–4 (n=184), including treatment group, eosinophil group at admission ('low' <300 cells/μL or 'high' ≥300 cells/μL) and their interaction.

**Results** Of 238 participants, 119 were allocated mepolizumab (54 [45%] eosinophilic at admission) and 119 placebo (65 [55%] eosinophilic). In those with a 'high' blood eosinophil count mepolizumab was associated with a hazard ratio (HR) of 0.69 (95% CI 0.44 to 1.09) compared to placebo. In contrast, for those with a 'low' blood eosinophil count the mepolizumab versus placebo HR was 1.3 (0.82 to 2.05). In those with eMRC 2–4 and a 'high' blood eosinophil count at admission, the HR for mepolizumab versus placebo was 0.61 (95% CI 0.36 to 1.03; n=99). In those with 'low' blood eosinophils at admission and eMRC 2–4 the HR was 1.47 ( 95% CI 0.86 to 2.52; n=85). The hazard ratio for the eosinophil group-by-treatment interaction was 0.53 (95% CI 0.28 to 1.01; p=0.053) in the overall population and 0.41 (0.19 to 0.86; p=0.019) in the eMRC 2–4 subgroup.

**Conclusions** Post-hoc analyses of COPD-HELP suggest that mepolizumab initiated at severe exacerbation conveys greater benefit in those with evidence of eosinophilic inflammation at admission, particularly in combination with better functional status.

# From primary to secondary care: evolving management of COPD

## P1 CURRENT PRACTICES, PERCEPTIONS AND FUTURE CHALLENGES IN COPD MANAGEMENT

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10.1136/thorax-2025-BTSabstracts.183

**Introduction** There is growing evidence to support the use of interventions beyond inhaled medications in COPD. Clinician attitudes and access to these therapies are crucial to informing COPD care pathways and the development of personalised care.

**Aims** We sought to explore current attitudes, practices, future directions and challenges in the management of moderate-severe COPD in the Republic of Ireland (ROI).

**Methods** We created a custom online survey comprising 38 questions, divided into 6 domains: responder demographics, pulmonary rehabilitation (PR), Long-term Non-invasive Ventilation (LTNIV), lung volume reduction (LVR), biologics therapies and lung transplantation. This was disseminated by email and text messaging to respiratory consultants in the ROI through the Irish Thoracic Society consultant contact list.

**Results** 37 responses were returned (24.5% response rate) between 4th December 2024 and 13th February 2025, with 36 eligible for inclusion. Completion rate was 97.2% (35/36). Only a minority reported sufficient access to PR (30%), or a dedicated COPD multidisciplinary team (MDT) (31%). 34% infrequently or never consider LTNIV despite 78% perceiving the quality of evidence on its benefit in reducing hospitalisations to be high or moderate. 59% infrequently or never consider LVR despite perceiving it to be moderately or highly beneficial towards improving exercise capacity (92%) and QoL (88%). 79% infrequently refer patients with COPD for lung transplant assessment. 31% were unsure about the emerging evidence for biologics in COPD. Perceived challenges to future integration of biologics into COPD care in ROI include the current absence of national drug reimbursement agreements, lack of dedicated COPD MDTs, and administrative burden.

**Conclusion** Our study provides insights into the current landscape of COPD management in the ROI. While significant progresses have been made in the diagnosis and management of COPD, access to essential services, such as pulmonary rehabilitation and a dedicated COPD MDT, remain suboptimal. There exists significant heterogeneity in the attitudes towards LTNIV and LVR, and uncertainty in relation to the role of biologics in COPD. Infrastructural limitations and lack of local guidelines are amongst the various issues that need to be addressed in order to optimise COPD care in the ROI.

## P2 DISEASE STABILITY, MANAGEMENT AND TREATMENT GOALS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE AMONG NEWLY DIAGNOSED VS LONG-TERM DIAGNOSED PATIENTS: A PATIENT SURVEY

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10.1136/thorax-2025-BTSabstracts.184

**Introduction and Objectives** To align healthcare professionals' (HCPs) priorities with patients' treatment goals, this survey aimed to uncover perspectives on factors influencing management and stability from patients with COPD and comorbidities, focusing on newly diagnosed (ND) and long-term diagnosed (LD) patients.

**Methods** A cross-sectional online survey explored patients' COPD experiences including symptoms and management, disease stability, treatment goals and expectations, self-management awareness, healthcare professional (HCP) communication and education needs. The 15-minute survey had 25 items with a combination of multiple choice, ranking, Likert scale and free text responses. The survey was administered across 11 countries to patients (n=330) aged ≥40 with a diagnosis of COPD for at least one year.

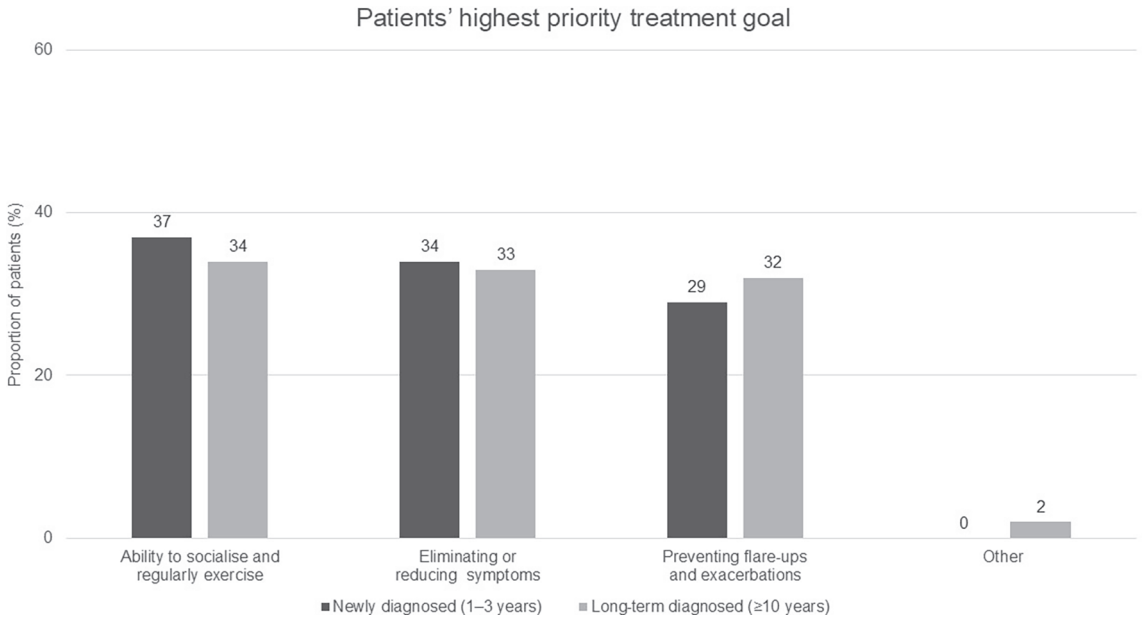
**Results** The analysis compared ND (those diagnosed 1–3 years ago, n=96) with LD (those diagnosed ≥10 years ago, n=113) patients. The most reported comorbidities were

**Abstract P1 Table 1** Perceived challenges in integrating biologic therapies into care pathways for patients with COPD

| Challenge to integrating biologics into COPD care | Ranked top (n, %) | Ranked in top 3 (n, %) |
|---|---|---|
| Lack of reimbursement under the High Tech Drug Arrangements | 10 (55.6%) | 17 (94%) |
| Absence of dedicated institutional COPD MDT | 2 (11.1%) | 13 (72%) |
| Administrative burden that comes with biologic prescribing | 1 (5.6%) | 10 (55%) |
| Others* | 4 (22.2%) | 6 (0.0%) |
| Prefer not to say | 0 (0.0%) | 0 (0.0%) |

Section responses: n = 18.

**Others: lack of smoking cessation support/fear biologics would be used in sub-optimally managed patients (i.e., tobacco smokers), inappropriate prescribing due to knowledge deficits among prescribers, nurse specialist workload/burden, cost to state due to high volume of patients., unequal access in private prescribing setting.*




**Abstract P2 Figure 1**

asthma, hypertension and anxiety. ND patients reported higher exacerbation rates than LD patients. ND patients expressed feeling less confident in recognising early flare-up signs. Primary reasons why patients would visit a healthcare provider were similar in both groups and included routine follow-ups and visits to discuss uncontrolled symptoms. Both ND and LD patients were somewhat or very satisfied with communication about COPD with their HCP. 62% of respondents reported that a COPD-specific treatment goal had been set by their HCP, although this was higher in ND than LD patients. When ranking their own treatment goals, both ND and LD patients closely ranked: 1) ability to socialise and regularly exercise; 2) eliminating/reducing symptoms; and 3) preventing flare-ups/exacerbations (figure 1). Qualitative analysis uncovered that descriptions of disease stability most commonly fell into the theme 'improvement in specific COPD related symptoms', followed by 'improvement in general disease state'. LD patients were more likely than ND patients to report that they feel they are achieving their treatment goals 'very well'.

**Conclusions** This survey highlights many similarities in the experiences of ND and LD patients with COPD, though there are some key differences that may inform better management. HCPs and patients should work together on treatment goals to ensure a patient-centred approach to disease management and stability.

**Funding** GSK

## P3 INTEGRATING AI-ASSISTED SPIROMETRY: VALIDATION AND USER EXPERIENCE IN A COPD DIAGNOSTIC PATHWAY

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**Introduction** Accurate interpretation of spirometry is essential for diagnosing chronic obstructive pulmonary disease (COPD). AI-based tools have the potential to enhance diagnostic accuracy and streamline workflow. ArtiQ.Spiro is an AI-based software that provides spirometry quality feedback and diagnostic support. As part of efforts to integrate ArtiQ.Spiro into our COPD diagnostic pathway in Glasgow, we conducted validation studies comparing its performance with clinician interpretation and assessed user experience.

**Methods** We retrospectively analysed spirometry data from two separate cohorts totalling 248 patients who attended for direct-access COPD diagnostic pathway in 2022 and 2024. The diagnostic support outputs of ArtiQ.Spiro were compared to interpretations by ARTP-registered clinicians and final diagnostic pathway outcomes to determine concordance. Additionally, we conducted a prospective validation where ArtiQ.Spiro's interpretations were obtained by assistant physiologists during test acquisition and initial interpretation for 125 consecutive tests. User experience feedback was collected from both trainee and senior assistant physiologists.

**Results** ArtiQ. Spiro demonstrated excellent concordance with pathway outcomes in the retrospective cohort. There was near-complete agreement between a 'normal' AI interpretation and a 'normal' clinician-reported spirometry and pathway outcome (NPV = 0.942), with good agreement by Artiq.Spiro alone for COPD identification (PPV = 0.69). In the prospective validation, 90% of tests were correctly interpreted by both assistant physiologists and ArtiQ.Spiro. Notably, 10% of initial assistant physiologist interpretations were incorrect but were correctly identified by ArtiQ.Spiro. Trainee assistant physiologists reported significant benefits from ArtiQ.Spiro's quality control and reporting components, highlighting its value in improving learning regarding test selection, interpretation, and reversibility decision-making. Senior assistants provided positive feedback, valuing continued access to the software and expressing willingness to issue reports for normal spirometry when spirometry was technically adequate, and assistant and ArtiQ.Spiro physiological interpretations were concordant.

**Conclusions** Based on these validation results and positive user experience across different clinician groups, we will proceed with implementing ArtiQ.Spiro in our COPD diagnostic

pathway, which currently manages over 600 patients per month. AI-assisted interpretation has the potential to significantly support trainee and experienced clinicians, improve diagnostic accuracy, optimise resource utilisation, and enhance early accurate diagnosis of COPD, while releasing senior clinician time for complex cases.

## P4 MULTIDIMENSIONAL CLASSIFICATION FOR SEVERITY OF EXACERBATIONS OF COPD: THE COPD EXACERBATION BAT (BASELINE, ACUITY, AND TRIGGER) CONCEPTUAL MODEL

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10.1136/thorax-2025-BTSabstracts.186

**Background** The severity of COPD exacerbations results from a combination of acute deterioration and baseline conditions initiated by an intrinsic or extrinsic trigger. Current classifications, such as the 2023 GOLD-adopted Rome classification, focus mainly on treatment given and hospitalisation, overlooking the dynamic relationship between baseline health and exacerbation events.

**Aim** To develop and evaluate a COPD exacerbation severity stratification model that addresses acute presentations and transitions between clinical stability and worsening disease.

**Method** A model to incorporate different dimensions, all that contributing to the severity of exacerbations, was developed through a series of workshops across European clinicians and patient feedback, developed through the CICERO ERS Clinical Research Collaboration. The model is currently being validated in cohorts of hospitalised patients in the UK and Belgium.

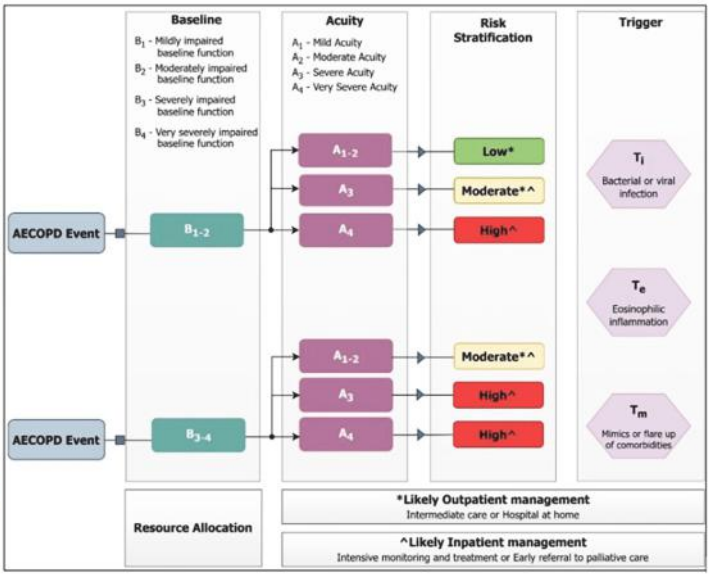
**Result** The BAT (Baseline, Acuity, Trigger) model incorporates three domains: Baseline (B), functional status of chronic disease; Acuity (A), severity of the acute illness; and Trigger (T), aetiologies triggering the event, allowing personalised treatment (left of figure 1). The BAT model stratifies exacerbations into low, moderate, or high risk, guiding healthcare facility, likelihood of deterioration, and recovery timelines (right of figure 1). Although data analysis is ongoing, early findings indicate that the BAT model outperforms the Rome classification in predicting mortality, with stronger alignment between severity grading and patient outcomes.

**Conclusion** The BAT classification may allow more individualised prognostic and therapeutic implications. Further validation across both community and hospital settings is needed to confirm its broader utility and applicability.

| Baseline | | | | |
|---|---|---|---|---|
| | Points on the severity of baseline function | | | |
| **Baseline Variables (0-3) points** | **0** | **1** | **2** | **3** |
| Dyspnoea (mMRC) scale | 0-1 | 2 | 3 | 4 |
| BMI (kg/m²) | > 21.0 | 18.5- 21.0 | < 18.5 | |
| $FEV_1$ (% predicted) | ≥ 65 | 50–64 | 36–49 | ≤ 35 |
| Frailty level (CFS) | ≤ 3 | 4 | 5-6 | 7-9 |

| Acuity | | | |
|---|---|---|---|
| **A1** | **A2** | **A3** | **A4** |
| Worsening dyspnoea compared to baseline for 48 hrs:<br><br>change in ΔmMRC <1 or ΔCAT <2 | Worsening dyspnoea compared to baseline for 48hrs:<br><br>change in ΔmMRC ≥1 or ΔCAT ≥2 | As per A2 plus any of<br><br>New respiratory failure AND/OR Evidence of multi-organ strain | As per A3 plus<br><br>Acute ventilatory failure (or acute on chronic)<br><br>$PaCO_2$ >6.0 KPa and pH<7.35 |

| Trigger | | |
|---|---|---|
| Respiratory infections (Ti) | Eosinophilic inflammation (Te) | Mimics (Tm) |




**Abstract P4 Figure 1** Multidimensional classification of severity of acute exacerbation of COPD (AECOPD) according to 'Baseline' (B), 'Acuity' (A) and 'Triggers' (T): The BAT model stratifies AECOPD events into low, moderate, or high risk by assessing three key domains: Baseline (B): Evaluate baseline health using lung function test (FEV1), functional disability due to breathlessness (mMRC scale), clinical frailty scale, and body mass index (BMI). Acuity (A): Assess the degree of worsening dyspnoea burden, multi-organ involvement, and acute ventilatory failure on admission. Trigger (T): Gather information on the exacerbation triggers (e.g., respiratory infection, eosinophilia, or mimics) to formulate personalised treatment for the current event and phenotype/mitigate the risk of future AECOPD events

P5

## IMPACT OF PHARMACOTHERAPY WITH INHALED CORTICOSTEROIDS AND LONG-ACTING BETA-2 AGONISTS ON SYMPTOM CONTROL AND OSCILLOMETRY PARAMETERS AMONG INDIAN PATIENTS WITH EARLY COPD DUE TO SMOKING AND NON-SMOKING CAUSES: INDIAN PRE COPD ASSESSMENT NETWORK (IECAN) STUDY

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**Background** Pre-COPD is seen in 6–8% smokers but difficult to diagnose/treat in clinical settings due to normal spirometry. We wished to study whether Oscillometry help identify Pre-COPD and help assess response to treatment.

**Methods** In this 12-week, randomized, double-blind double dummy, placebo-controlled trial across 10 Indian centers, adults aged 35–55 without previous history of or treatment for Asthma, with more than 10 pack years of smoking or equivalent biomass fuel exposure, respiratory symptoms and CAT score>10, normal spirometry, and elevated AX (>6 $cmH_2O/L$) were enrolled. Participants underwent HRCT, received salbutamol inhaler as needed and randomized to once-daily Fluticasone Furoate/Vilanterol or placebo. All smokers were provided smoking cessation advice.

Assessments performed at baseline, 6, and 12 weeks included CAT score, spirometry, and oscillometry (using Tremoflo).

**Results** 149 individuals screened, 93 eligible patients were randomized (Active Group: FF/V, n=43;

Placebo group, n=50). 34 participants lost to follow-up, data from 59 analyzed (Active 28; Control: 31). Well matched groups with mean age 44, CAT score 15 and median Ax 13.6 cmH2O/L. There was a significant within-group reductions in CAT scores. Between-group difference of 1.7 was not significant (p=0.18). Spirometry showed no significant change. However, oscillometry parameters, (Ax, R5, R5-R20), showed significant improvement in Active group compared to baseline and compared to Placebo (p<0.05, figure 1).

Of the 59 participants, 41 were smokers (20 Active and 21 control) and 18 had biomass fuel exposure. 31of the 41 smokers reported that they had stopped smoking. Ax improved significantly in those who stopped smoking, compared to those who continued to smoke but no significant change was noted in R5 and R5–20.

**Conclusion** Oscillometry helped diagnose pre-COPD and was able to objectively detect response to FF/V therapy as well as to smoking cessation. Pre-COPD can potentially be defined in at risk patients with normal spirometry if they have CAT>10 and Ax>10. A larger study is needed to assess whether these findings are reproducible and generalisable.

| Group | Active (43) | Control (50) | $=Between Group difference |
|---|---|---|---|
| Age (years) | 45.4 | 43.7 | NS |
| Gender : M (%) | 33 (77%) | 42 (84%) | NS |
| BMI ($Kg/m^2$) | 25.3 | 25.8 | NS |
| Smoking pack years | 14.9 | 14.1 | NS |
| Biomass Exposure Years | 19.8 | 20.4 | NS |
| Comparative Analysis Results (Baseline to End of Treatment Visit) | | | |
| CAT score (mean ± SD) | 15.4 ± 3.2 / 5.7 ± 3.8 * | 14.5 ± 3.6 / 6.5 ± 4.8 * | 1.7, p = 0.181 |
| R5 $CmH_2O/L/S$ (median) | 3.9/3.4 * | 4.0/ 4.1 | $, p = 0.008 |
| R5-20 $CmH_2O/L/S$ (median) | 0.77/0.66 * | 0.83/ 0.95 | $, p = 0.010 |
| Ax Pre $CmH_2O/L$ (median) | 13.8/ 10.1 * | 13.4/ 11.9 | $, p = 0.006 |
| FEV1% predicted (mean ± SD) | 91.3 ± 13.2 / 92.2 ± 15.4 | 89.9 ± 12.3 / 90.5 ± 14.5 | NS |
| FVC% predicted (mean ± SD) | 92.6 ± 13.8 / 94.8 ± 18.4 | 93.2 ± 10.7 / 94.3 ± 12.1 | NS |
| Ratio (mean ± SD) | 0.82 ± 0.05 / 0.81 ± 0.06 | 0.8 ± 0.06 / 0.8 ± 0.07 | NS |

* Within group difference, $ between group difference

**Abstract P5 Figure 1**

## P6 RELATIVE LUNG AVAILABILITY AND TOTAL SYSTEMIC EXPOSURE AFTER INHALATION OF MEDIUM OR HIGH STRENGTH DOSES OF BECLOMETASONE DIPROPIONATE/FORMOTEROL FUMARATE, FORMULATED AS PMDI WITH A NEXT GENERATION PROPELLANT

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10.1136/thorax-2025-BTSabstracts.188

**Background** Growing concern around the global warming potential (GWP) of hydrofluoroalkane 134a (HFA-134a) propellant used in pressurized metered dose inhaler (pMDI) for asthma and COPD has prompted research into alternatives like HFA-152a, with a significantly lower GWP.

A new formulation of CHF1535 [beclometasone dipropionate (BDP)/formoterol fumarate (FF)] 100/6 µg medium strength (MS) and 200/6 µg high strength (HS) has been developed with HFA-152a. Bioequivalence with CHF1535 HFA-134a marketed formulation should be demonstrated for BDP, B17MP (active metabolite of BDP) and Formoterol.

**Methods** Two single-dose, double-blind, randomized, 4-way crossover studies with CHF1535 MS or HS were conducted in healthy volunteers, administered with a single dose of 4 inhalations of each formulation with HFA-152a or HFA-134a, with and without the use of the AeroChamber Plus® spacer. Primary PK endpoints were $C_{max}$, $AUC_{0\text{-}t}$ and $t_{max}$ for all analytes and $AUC_{0–30\ min}$ for Formoterol only, to assess lung availability and systemic effect. Safety was monitored.

**Results** The 90% confidence intervals of all PK parameter ratios between HFA-152a and HFA-134a for lung availability and total systemic exposure were within the bioequivalence acceptance interval (80.00–125.00%) for both strengths, with and without the spacer. There was no shift in median $t_{max}$. No safety signals were observed.

**Conclusions** Therapeutic equivalence between the two formulations was demonstrated, supporting the timely introduction of therapeutic options enabling a seamless transition for patients, while minimizing climate impact.

## P7 SUSTAINABLE CARE: WHAT PEOPLE WITH COPD ARE OFFERED A LOWER CARBON INHALER DEVICE AND WHO IS MORE LIKELY TO SUCCEED IN SWITCHING?

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10.1136/thorax-2025-BTSabstracts.189

Inhalers contribute significantly to carbon emissions in healthcare. Metered Dose Inhalers (MDIs) have a larger carbon footprint compared to Dry Powder Inhalers (DPIs). As a result, the NHS has prioritized transitioning patients from MDIs to DPIs when appropriate. However, there is limited understanding of COPD patient characteristics that may determine a clinician's decision to offer a switch and indeed whether the switch is a success or failure.

Asthma + Lung UK asked people with a respiratory disease to complete a wide-ranging online survey in 2023. Participants were asked if they had been offered the opportunity to switch to a DPI, and if so if it had been successful. We compared demographic, economic and clinical factors between people who successfully switched versus those who changed back to an MDI.

4,821 people with COPD replied (64.6% female; age 67.8 ±13.0). 1,767 (36.6%) were offered a switch from MDI to DPI of whom 1,573 (89%) accepted. There were no differences in gender, age, race, or income between those who had and had not been offered a switch. Those offered a DPI were more likely to have been aware of the carbon impact of inhalers (62.5% vs 47.9%) and had had planned healthcare attendances (63% vs 52.3%). The change to DPI was successful in 969 (61.6% of switchers). Unsuccessful switchers were more likely to be female, and have uncontrolled disease, with increased unplanned healthcare utilisation and exacerbation frequency. Awareness of the carbon impact of MDIs was not significantly different between the two groups (60.3% vs 63.6%).

This data provides insight that most patients who expressed a view are willing to consider a switch in inhaler. Most patients offered a switch will succeed and those with well controlled COPD are those most likely to successfully change from an MDI to a DPI.

## P8 REAL-WORLD STUDY ON CAUSES OF DEATH IN PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) IN ENGLAND

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10.1136/thorax-2025-BTSabstracts.190

COPD is characterized by persistent respiratory symptoms and airflow obstruction. While previous studies have evaluated the mortality burden of COPD, there is limited recent research describing the leading causes of death. This study sought to describe mortality in people with COPD and identify the leading causes of death, overall and by sub-groups of interest.

In this longitudinal cohort study of people with COPD in the CPRD Aurum database linked with Hospital Episode Statistics and death data from the Office for National Statistics from England (1 Jan 2014–10 Mar 2020), mortality rates (per 1000 patient years [PYs]) are described overall, by the top five causes (based on 3-digit ICD10 codes) and for pre-specified causes. Patients were stratified by groups, including COPD severity levels.

Of 369,910 people with COPD (median age 69 years, 47% women, 93% White), 24.7% died over a median of 3.7 years follow-up; 67.4 (95%CI 67.0–67.9) deaths per 1000 PYs.

Cause-specific rates per 1000 PYs were 17.4 (17.2–17.6) for COPD-related mortality, 9.6 (9.4–9.7) for CVD, 7.9 (7.7–8.0) for lung cancer, 1.9 (1.9–2.0) for pneumonia, and 1.9 (1.8–2.0) for dementia. Men with COPD had a numerically higher rate of CVD-related mortality, and women with COPD for dementia-related mortality. People with severe COPD had a numerically higher overall mortality rate.

COPD, CVD and lung cancer were the leading primary causes of death among people with COPD. These findings provide up to date information on the mortality burden of COPD, and highlight the importance of comprehensive multidisciplinary management strategies to address both respiratory and comorbid conditions for improved survival in COPD.

## P9 ADVANCED CLINICAL PRACTITIONER (ACP) HOT AIRWAYS CLINIC: PROOF OF CONCEPT TO ASSESS DEMAND AND OUTCOMES OF EOSINOPHILIC AIRWAYS DISEASE (EAD)

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10.1136/thorax-2025-BTSabstracts.191

**Background** The ACP secondary care airways clinic supports primary and emergency care with the diagnosis and optimisation of airways disease patients in a large inner city teaching hospital. Eosinophilic airways disease (EAD) is highlighted within the national guidance to seek early optimisation to reduce exacerbation, improve quality of life and prevent avoidable long-term airways damage.

157 new patients have attended the clinic with their outcomes reviewed following new diagnosis or optimisation to understand the impact of poorly controlled EAD management on secondary care respiratory referrals.

**Objectives** 1. Identify the number of EAD being referred for optimisation/diagnosis

2. Determine use of best practice guidelines for EAD in our local area.

**Methods** Case notes of new patient's referred to the clinic were reviewed over a 12-month period. Criteria for referral included suspected/poorly controlled airways disease from either Primary care or Emergency/Ambulatory care. presenting complaints, initial eosinophils >0.30 x10(9) and primary diagnoses were reviewed. Escalation of treatment was as per national guidance.

**Results** 157 patient referrals were reviewed: median age 60 years (IQR 21–86); female 54%; BAME 25%. Referral source: 6% from urgent care; 7 GP referrals; 87% Airways Team and 6% from other Respiratory Consultants. New diagnosis 103 (66%) of which 62% were eosinophilic with 32% ICS naive and initiated as part of their management. 15% were encouraged to improve compliance with their existing ICS.

21% (N=33) received disease optimisation of which 73% were eosinophilic (n=24). 52% (n=17) had their ICS escalated. 7% had a change of ICS device.

Other non-airways diagnoses were made in 4%. 5% self-resolved by the time of review.

**Conclusion** The recognition of EAD as a need for referral is evident but the management of EAD within primary and emergency care is lacking despite the production of local, national and international guidelines. A 'one stop' clinic to serve and optimise patients has proved integral to manage this patient cohort and streamlining secondary care respiratory referrals. Ongoing work within primary/emergency care is needed to reduce poor outcomes by applying best practice particularly to our EAD is cohort who have increased risk of exacerbation, healthcare utilisation and poor outcomes.

## P10 BREATHLESS SYSTEMS: COPD PRESSURES IN HOSPITALS ACROSS ENGLAND

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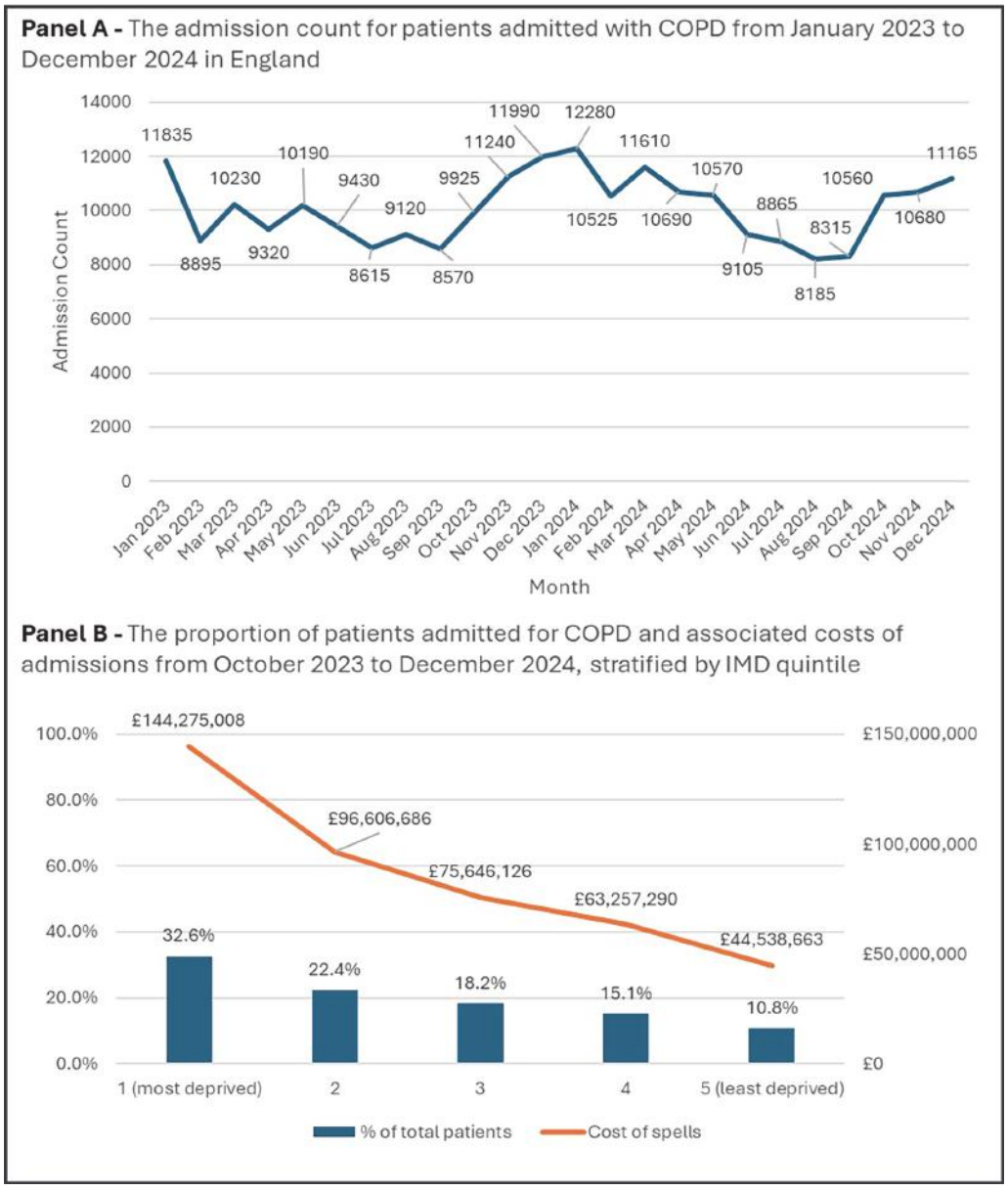
10.1136/thorax-2025-BTSabstracts.192

**Introduction** Chronic obstructive pulmonary disease (COPD) – a progressive disease characterised by persistent airflow limitation – causes a substantial healthcare burden. This analysis aims to quantify the burden of COPD in secondary care settings in England and the impact of seasonality and deprivation.

**Methods** This was an exploratory, retrospective analysis of the Hospital Episode Statistics (HES) data for patients with a primary diagnosis of COPD (J44.0, J44.1, J44.9) admitted between January 2023 and December 2024. Hospital admissions, length of stay (LOS), and costs were calculated; stratifications included month, patient age, and index of multiple deprivation (IMD). Longitudinal analysis was conducted; patients with an admission for COPD within the 2023 calendar year (index admission) were followed for 1-year following their index date; repeat admissions for COPD in the year after admission were assessed.

**Results** Across 2023 and 2024 there were 153,800 patients identified in England accounting for 241,925 admissions from COPD, costing £673,076,864, with an average LOS of 6.39 days and cost per admission of £2,782. There were 46,405 admissions in Winter (December 23-March 24) compared with 26,105 in Summer (June-September 24). Monthly admission counts are shown in figure 1A. Between October 2023 and December 2024, patients in the most deprived IMD quintile had the highest number of admissions and greatest cost (figure 1B). In 2024, patients aged <65 represented 23% of the admissions across England (average LOS 5.03 days). Longitudinal analyses showed that of 86,210 patients in the cohort in 2023, 26,460 (30.7%) were readmitted within 1 year for Acute Exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD); of these patients, 12,425 (47%) had at least two admissions and 6,920 (26.2%) had three admissions.

**Conclusions** Burden is influenced by seasonality, with 35% more admissions in winter compared with summer. The most deprived are disproportionately impacted, accounting for three times more admitted patients than the least deprived quintile. Furthermore, nearly a quarter of patients admitted were of working age. This analysis demonstrates that COPD continues to represent a substantial burden on patients, the NHS, and




**Abstract P10 Figure 1**

society; there is an urgent need to focus the optimisation of care on these high burden groups.

## P11 COPD AND RESPIRATORY VIRAL INFECTION IN HOSPITALISED ADULTS: LONGER SYMPTOM DURATION, BLUNTED PERCEPTION, AND POOR EARLY OUTCOMES

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10.1136/thorax-2025-BTSabstracts.193

**Background** COPD is a known risk factor for poor outcomes following respiratory viral infection (RVI), but its impact on patient-reported symptom burden and early hospital trajectory remains under-characterised. Using data from the UK UNIVERSAL study—a prospective multicentre cohort of hospitalised adults with PCR-confirmed RVI—we compared clinical profiles, symptom severity and short-term outcomes in individuals with and without COPD.

**Methods** Adults (≥18 years) enrolled from August 2022 to May 2025 were stratified by clinician-diagnosed COPD. FLU-PRO Plus domain scores (0–100 scale) and symptom duration were recorded at admission. Outcomes were: (i) length of stay (LOS: ≤2, 3–7, 8–14, ≥15 days), (ii) respiratory high-dependency unit (RHDU) or GICU admission, (iii) 30-day readmission for acute respiratory illness (ARI), and (iv) 60-day all-cause mortality. Between-group differences were assessed using Mann-Whitney U, $\chi^2$, or Fisher's exact tests.

**Results** Of 836 participants, 234 (28%) had COPD. They were older (median 71 vs 63 years, $p<0.001$) and reported significantly lower symptom intensity scores across most domains: nasal (28.5 vs 33.1, $p=0.015$), throat (14.4 vs 22.1, $p=0.003$), gastrointestinal (9.0 vs 15.6, $p<0.001$), and systemic (31.4 vs 37.8, $p=0.003$); eye and chest/respiratory scores were comparable. Despite this, COPD was associated with more prolonged symptom duration across multiple domains ($p<0.03$), and significantly worse clinical outcomes: LOS ≥15 days (8.5% vs 5.7%, $p=0.003$), RHDU admission

**Abstract P11 Table 1** Patient characteristics and outcomes by COPD status

| Characteristic | COPD (n=253) | No COPD (n=583) | p-value |
|---|---|---|---|
| RSV | 43 (17.1%) | 68 (11.6%) | 0.134 |
| Influenza | 54 (21.4%) | 149 (25.5%) | |
| Rhinovirus/Enterovirus | 51 (20.2%) | 115 (19.7%) | |
| SARS-CoV-2 | 56 (22.2%) | 161 (27.6%) | |
| Other virus infection | 31 (12.3%) | 61 (10.4%) | |
| Viral co-infection, n (%) | 17 (6.7%) | 30 (5.1%) | |
| Age, median [IQR] (years) | 71 [64–77] | 60 [48–75] | <0.001 |
| Male sex | 131 (52.0%) | 262 (44.9%) | 0.061 |
| Pneumococcal vaccinated | 151 (60.9%) | 220 (38.3%) | <0.001 |
| Symptom duration prior to hospitilisation, median [IQR] (days) | 7.1 [3.0–8.0] | 6.2 [3.0–7.0] | 0.007 |
| 60-day mortality | 16 (7.3%) | 11 (2.1%) | <0.001 |
| 30-day readmission with respiratory syndrome | 26 (12.0%) | 36 (6.8%) | 0.013 |
| RHDU admission | 6 (2.6%) | 4 (0.7%) | 0.036 |

(2.6% vs 0.7%, $p=0.036$), 30-day ARI readmission (12.0% vs 6.8%, $p=0.019$), and 60-day mortality (7.3% vs 2.1%, $p<0.001$). COPD patients also more frequently reached WHO OSCI $\geq 5$ (72.6% vs 66.2%, $p=0.068$).

**Conclusion** Adults with COPD hospitalised for RVI demonstrated paradoxically lower perceived symptom severity despite more prolonged symptom duration and significantly worse early outcomes. These findings highlight the need for enhanced monitoring and targeted intervention strategies—including antiviral trials—focused on this high-risk population.

## P12 CHARACTERISTICS AND OUTCOMES OF PATIENTS WITH COPD AND ACUTE HYPERCAPNIC RESPIRATORY FAILURE (AHRF) ADMITTED TO RESPIRATORY SUPPORT UNIT (RSU)

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10.1136/thorax-2025-BTSabstracts.194

**Background** Mortality from COPD with AHRF remains high and several prognostic factors have been previously identified.

**Abstract P12 Table 1**

| Characteristics | Group I (n=19) | Group II (n=26) | Group III (n=25) |
|---|---|---|---|
| Mean age (in years) | 75 | 68 | 71 |
| Male | 11 (58%) | 15 (58%) | 7 (28%) |
| Female | 8 (42%) | 11 (42%) | 18 (72%) |
| Eosinophils <0.3 x 10^9/L | 18 (95%) | 19 (73%) | 22 (88%) |
| Eosinophils ≥0.3 x 10^9/L | 1 (5%) | 7 (27%) | 3 (12%) |
| FEV1 < 30% | 3 (16%) | 10 (38%) | 4 (16%) |
| FEV1 30 – 49% | 1 (5%) | 7 (27%) | 6 (24%) |
| FEV1 50 – 80% | 3 (16%) | 3 (12%) | 3 (12%) |
| No FEV1 record | 12 (63%) | 6 (23%) | 12 (48%) |
| Current smoker | 6 (32%) | 13 (50%) | 11 (44%) |
| Ex-smoker | 11 (58%) | 13 (50%) | 13 (52%) |
| Never smoker | 2 (10%) | 0 (0%) | 1 (4%) |
| Heart failure | 10 (53%) | 5 (19%) | 8 (32%) |
| Diabetes mellitus | 8 (42%) | 6 (23%) | 7 (33%) |
| ≥2 comorbidities | 14 (74%) | 10 (38%) | 8 (32%) |

*all numbers are representative of n= unless stated otherwise

**Aim and objectives** We aim to demonstrate certain patient characteristics and associated outcomes in COPD patients with AHRF admitted to RSU.

**Methods** We retrospectively collected data of 70 patients with COPD and AHRF admitted to RSU between 1/1/2023 and 1/10/2023. Recorded variables are demographics, clinical data and 3-month outcome. Data was analysed using Excel.

**Results** We classified our patients' 3-month outcomes into Group I (death, n=19; 27%), Group II (readmission, n=25; 36%) and Group III (no readmission, n=26; 37%) with mean age 75, 68 and 71 respectively. Eosinophilia (Eosinophils ≥0.3 x 10^9/L) was present in 5% of Group I, 27% of Group II and 12% of Group III. Eosinophilia was absent in current smokers, contrary to expectations. 74% of the mortality group had ≥2 comorbidities. 76% (13/17) of patients with FEV1 <30% either died or were readmitted, though most patients lacked FEV1 data. Details are shown in table 1.

**Conclusion** Mortality is higher in males and patients with ≥2 comorbidities. Eosinophilia is not associated with mortality confounded by old age and comorbidities. Male sex, very severe lung function, and eosinophilia correlate with 3-month readmission. Further research is required to identify prognostic factors for treatment optimization.

## P13 SEASONAL VARIATION IN EXACERBATION RISK FOLLOWING SEVERE AECOPD

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10.1136/thorax-2025-BTSabstracts.195

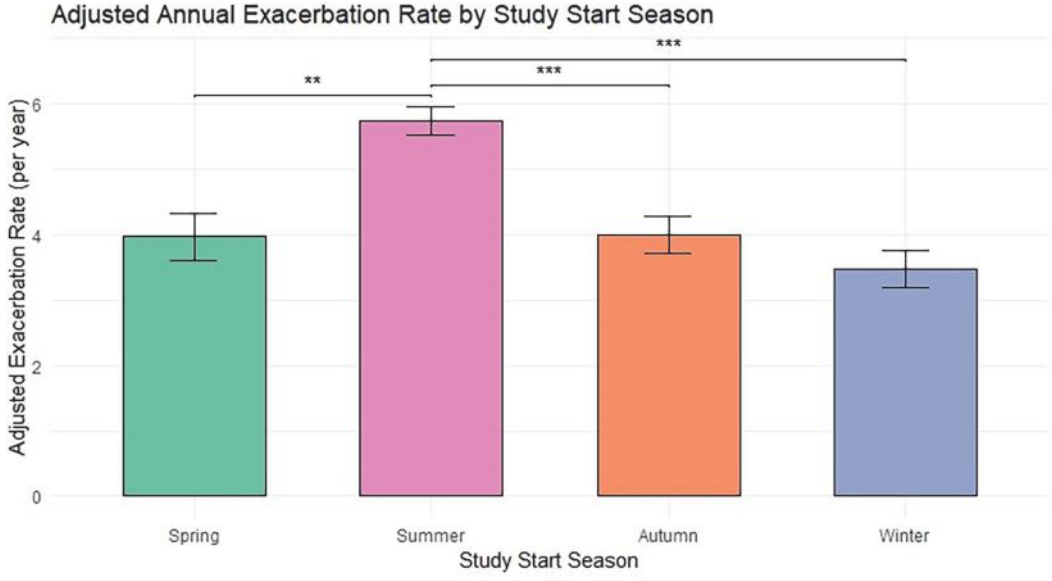
**Introduction** Admission to hospital with a severe exacerbation of COPD (AECOPD) is associated with an increased risk of further exacerbations, readmission and death. Seasonality can affect exacerbation phenotype (Aung *et al*, 2024), but it remains unclear how season of onset affects ongoing risk following severe AECOPD.

**Methods** COPD-HELP was a single-centre, double-blind, randomised, placebo-controlled trial that recruited 238 participants during hospitalisation for AECOPD and allocated them 1:1 to mepolizumab or placebo for 48 weeks. All participants had an eosinophil count ≥300 cells/μL on at least one occasion in the prior 12 months, were established on inhaled corticosteroids and current or ex-smokers. Full details are published elsewhere. (Flynn *et al.*, 2025)

For this analysis, 119 participants on placebo were included. Exacerbation rates were analysed based on an individual's season of entry into the trial.. Entry into trial occurred within a week of a severe AECOPD for all participants. Exacerbation rates were calculated as the total number of exacerbations per participant, offset by time in trial. A linear regression model was fitted, using season of study enrolment as the primary predictor variable and exacerbation rate in the preceding 12 months as a covariate

**Results** Adjusted annual exacerbation rates varied significantly by season, with the highest rate in summer (adjusted mean 5.76, 95% CI, 5.33 – 6.19), followed by autumn (4.02, 95% CI, 3.47 – 4.57), spring (3.98, 95% CI, 3.28 – 4.69), and lowest in winter (3.48, 95% CI, 2.92 – 4.04). This represents a relative increase of 65% following an exacerbation in summer compared to winter.

**Conclusions** In an eosinophilic COPD population, severe exacerbations in summer were associated with a higher rate of exacerbations in the following year than those in any other season. This group may be particularly susceptible to environmental exposures such as increased pollen levels, pollution index or other seasonal allergens. Greater understanding of seasonal dynamics and mechanisms driving recurrent exacerbations could play a role in risk reduction and post exacerbation care and guidance.




**Abstract P13 Figure 1**

## P14 UNDER PRESSURE: PATIENT CHARACTERISTICS ASSOCIATED WITH PRIMARY CARE RESPIRATORY TREATMENT REVIEWS BEFORE RESPIRATORY WINTER PRESSURES IN THE UK

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10.1136/thorax-2025-BTSabstracts.196

**Introduction** Chronic obstructive pulmonary disease (COPD) and asthma present substantial clinical and economic burdens and are the leading cause of admissions and 'winter pressures' on the UK National Health Service. Our study will explore epidemiological factors associated with respiratory treatment review in primary care, before respiratory winter pressures.

**Methods** This interim analysis of a retrospective cohort study uses Clinical Practice Research Datalink (CPRD) and Hospital Episode Statistics (HES) linked datasets. Adult patients alive at index (01/11/2022) with a history of asthma or COPD were included. Index characteristics stratified by primary care treatment review codes in the 12-months prior to index are reported.

**Results** Key demographics are presented in table 1.

Of 1,412,720 patients with asthma, 32.5% (459,651) had received a treatment review. Reviewed patients were older (48 vs 39 years), had more comorbidities (Charlson Comorbidity Index [CCI] 1.7 vs 1.4, 32% vs 18.9% with cardiovascular comorbidities), had a higher proportion with exacerbations in the year prior (13.8% vs 4.9%) and had a greater number of respiratory medications; a higher proportion received any ICS (89% vs 74%), ICS+LABA (47% vs 14%), and SABA (99.5% vs 91.4%).

Of the 186,297 patients with COPD, 52.0% (96,949) received a treatment review. Patients reviewed tended to be older (72 vs 67 years), had a higher burden of comorbidities (CCI 3.0 vs 2.5, 68% vs 58.9% with cardiovascular comorbidities), and a higher proportion of patients receiving more advanced therapies (e.g. triple therapy, 30.2% vs 13.4%). Patients reviewed also had higher burden of moderate exacerbations (30.0% moderate, 2.9% severe vs 16.5% moderate, 1.9% severe). Notably, of the 43,758 patients with moderate and 4,507 patients with prior severe exacerbations, only 66% and 63% respectively were reviewed in the prior year.

**Conclusions** Treatment reviews for asthma and COPD predominantly targeted patients at higher disease risk, yet notably, some high-risk individuals—such as those with prior hospitalisation—did not receive a review. Outcomes during winter and the effects of treatment review and disease control will be addressed in forthcoming analyses. Preliminary findings emphasise the need to investigate missed opportunities for review and to identify further strategies to improve patient care before winter.

**Abstract P14 Table 1** Patient demographics for the overall asthma and COPD cohorts stratified by review status in the 12 months prior to index

| | *Asthma* | | | *COPD* | | |
|---|---|---|---|---|---|---|
| ***Characteristic*** | **Overall (N=1,412,720)** | **Not Reviewed (N=953,069)** | **Reviewed (N=459,651)** | **Overall (N=186,297)** | **Not Reviewed (N=89,348)** | **Reviewed (N=96,949)** |
| **Age, years, mean (SD)** | 42 (± 21) | 39 (± 21) | 48 (± 22) | 70 (± 14) | 67 (± 17) | 72 (± 10) |
| **Gender (female), n (%)** | 756,223 (53.5%) | 487,817 (51.2%) | 268,406 (58.4%) | 86,716 (46.5%) | 42,205 (47.2%) | 44,511 (45.9%) |
| **Charlson Comorbidity Index, mean (SD)** | 1.5 (± 1.2) | 1.4 (± 1.1) | 1.7 (± 1.3) | 2.8 (± 2.0) | 2.5 (± 2.0) | 3.0 (± 1.9) |
| **Cardiovascular comorbidities†, n (%)** | 326,982 (23.1%) | 179,808 (18.9%) | 147,174 (32.0%) | 118,736 (63.7%) | 52,662 (58.9%) | 66,074 (68.2%) |
| **Current Smoker, n (%)** | 209,994 (14.9%) | 155,167 (16.3%) | 54,827 (11.9%) | 65,127 (35.0%) | 31,058 (34.8%) | 34,069 (35.1%) |
| **Prior SABA, n (%)** | 1,328,277 (94.0%) | 871,081 (91.4%) | 457,196 (99.5%) | 160,526 (86.2%) | 68,103 (76.2%) | 92,423 (95.3%) |
| **Prior ICS, n (%)** | 1,115,216 (78.9%) | 706,763 (74.2%) | 408,453 (88.9%) | — | — | — |
| **Prior ICS+LABA, n (%)** | 348,957 (24.7%) | 132,664 (13.9%) | 216,293 (47.1%) | — | — | — |
| **Prior LABA+LAMA, n (%)** | — | — | — | 30,389 (16.3%) | 9,323 (10.4%) | 21,066 (21.7%) |
| **Prior Fixed Triple Therapy, n (%)** | — | — | — | 41,217 (22.1%) | 11,956 (13.4%) | 29,261 (30.2%) |
| **Prior Open Triple Therapy, n (%)** | — | — | — | 11,573 (6.2%) | 4,281 (4.8%) | 7,292 (7.5%) |
| **IMD Quintile, n (%)** | | | | | | |
| ***1*** | 237,479 (16.8%) | 159,162 (16.7%) | 78,317 (17.0%) | 21,636 (11.6%) | 10,638 (11.9%) | 10,998 (11.3%) |
| ***2*** | 223,585 (15.8%) | 149,089 (15.6%) | 74,496 (16.2%) | 26,740 (14.4%) | 13,170 (14.7%) | 13,570 (14.0%) |
| ***3*** | 213,085 (15.1%) | 142,763 (15.0%) | 70,322 (15.3%) | 28,032 (15.0%) | 13,800 (15.4%) | 14,232 (14.7%) |
| ***4*** | 228,464 (16.2%) | 154,020 (16.2%) | 74,444 (16.2%) | 33,254 (17.8%) | 15,996 (17.9%) | 17,258 (17.8%) |
| ***5*** | 231,530 (16.4%) | 157,925 (16.6%) | 73,605 (16.0%) | 40,326 (21.6%) | 18,348 (20.5%) | 21,978 (22.7%) |
| ***Missing*** | 278,577 (19.7%) | 190,110 (19.9%) | 88,467 (19.2%) | 36,309 (19.5%) | 17,396 (19.5%) | 18,913 (19.5%) |
| **FEV1 (litres), mean (SD)** | 2.6 (± 0.88) | 2.7 (± 0.91) | 2.5 (± 0.85) | 1.8 (± 0.76) | 1.9 (± 0.79) | 1.8 (± 0.74) |
| **Asthma exacerbation during baseline, n (%)** | 110,424 (7.8%) | 47,165 (4.9%) | 63,259 (13.8%) | — | — | — |
| **Moderate Exacerbation* during baseline, n (%)** | 104,968 (7.4%) | 45,071 (4.7%) | 59,897 (13.0%) | 43,758 (23.5%) | 14,721 (16.5%) | 29,037 (30.0%) |
| **Severe Exacerbation** during baseline, n (%)** | 5,456 (0.4%) | 2,094 (0.2%) | 3,362 (0.7%) | 4,507 (2.4%) | 1,661 (1.9%) | 2,846 (2.9%) |

†Includes

*Defined as (1) in asthma: a course of OCS, or a code for asthma exacerbation (for asthma), or (2) in COPD: a course of OCS and antibiotics on the same day, or a COPD exacerbation code

**Defined as an inpatient hospitalisation for COPD or Asthma

COPD = Chronic obstructive Pulmonary Disease; FEV1 = Forced Ejection Volume in 1 second; ICS = Inhaled Corticosteroid; IMD = Index of Multiple Depravation; LABA = Long-Acting Beta Agonist; LAMA = Long-acting muscarinic antagonists; SABA = Short-Acting Beta Agonist; SD = Standard Deviation.

## P15 UNDERSTANDING INCREASED HOSPITALISATIONS IN INTEGR-COPD: A SECONDARY ANALYSIS OF ADMISSION DATA

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10.1136/thorax-2025-BTSabstracts.197

**Introduction and Objectives** The INTEGR-COPD study demonstrated adherence to COPD management guidelines can be improved through the integration of respiratory specialists into general practice to deliver annual COPD reviews.[1] Unexpectedly, the study found a statistically significant increase in hospitalisations among the intervention group (IRR 1.86, 95% CI 1.38 to 2.52; $p<0.001$) contrary to findings from a previous systematic review.[2] This secondary analysis aims to explore the potential reasons for this finding.

**Methods** All patients with 'COPD-related Hospitalisation' during the 12 month follow-up period were identified from the original intention-to-treat dataset. Secondary care electronic patient records from the primary hospital site in the locality were reviewed to detail each admission episode. One record was removed due to missing data limiting further analysis. Statistical analysis was performed using zero-inflated poisson regression using STATA v19.5 (STATACorp).

**Results** In the intervention group, 91/180 (51%) original listed admission episodes were identified retrospectively, compared to 157/167 (94%) in the control group. Although there is a small possibility that patients may have been admitted to other hospitals either regionally or nationally, geographical mapping shows that the secondary care system utilised represented the closest hospital trust. New analysis now shows an IRR of 0.74, 95% CI 0.55 to 1.01, $p=0.055$, adjusting for age, sex, baseline hospitalisation and clustering.

**Conclusions** Our findings now show that integrated COPD care is likely to have reduced hospitalisation episodes rather than increased them as initially reported. These results are likely to reflect methodological differences in the original study such that the intervention group recorded patient reported outcomes whilst the control group used hospital records to overcome missing data. This highlights the importance of triangulating patient reported data with electronic records to ensure reliability and the importance of consistent methodology across study arms.

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2. Poot CC, Meijer E, Kruis AL, Smidt N, Chavannes NH, Honkoop PJ. Integrated disease management interventions for patients with chronic obstructive pulmonary disease. *Cochrane Database of Systematic Reviews* 2021;(9):CD009437. DOI:10.1002/14651858.CD009437.pub3.

# Monitoring, managing and treating ILD to improve outcomes

## P16 A NATIONAL SURVEY MAPPING THE PROVISION OF SUPPORTIVE AND PALLIATIVE CARE SERVICES FOR INTERSTITIAL LUNG DISEASE PATIENTS ACROSS THE UNITED KINGDOM

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10.1136/thorax-2025-BTSabstracts.198

**Introduction and Objectives** Interstitial lung diseases (ILDs) are commonly associated with progressive symptoms, reductions in quality-of-life and survival. Although the literature supports the importance of supportive and palliative care (SPC) in the patient journey, there remains no consensus/national guideline on SPC provision in the United Kingdom (UK). We hypothesised that there are disparities in SPC provision across the UK and aimed to map services in hospitals and the community.

**Methods** A cross-sectional online survey devised by members of the UK Supportive Palliative Care ILD Network was distributed to respiratory and SPC health-care professionals (HCP) from across the UK. Patient representatives contributed to the planning meeting. The questions covered SPC referral pathways, and presence of SPC-dedicated MDTs and clinics within hospitals. Community site-related questions covered the provision for ILD patients of holistic services, and end-of-life discharge facilitation from hospital.

**Results** Ninety-one responses were received from across the UK between November 2024 and May 2025. Seventy-four respondents were from secondary care and covered 51 hospital sites/trusts (table 1). Twenty-three specialist ILD centres responded. 32% of respondents reported a dedicated ILD SPC MDT meeting was operational in their hospital site. SPC outpatient services appeared to vary across the UK by frequency, duration and HCP. 28% of respondents reported the presence of joint SPC-respiratory clinics for ILD patients. Conflicting answers in sites which received $\geq$2 responses, and respondents answering 'unknown' to questions suggested poor understanding by HCPs of local services. Only 17 responses were received from community-based sites, 8 of which were from hospice settings.

**Conclusions** This national survey provides the first insights into SPC services available for ILD patients across the UK and highlights variabilities in the provision of care. Unfortunately, limited responses were received regarding community services. Respondents may not have replied if the SPC services in their locality are accessible and utilised by ILD patients, but are not

**Abstract P16 Table 1** Table summarising number and percentage of respondents that report the provision of supportive and palliative care (SPC) services for ILD patients at hospital sites across the United Kingdom

| Region | Sites (n) | Respondents (n) | Formal referral pathway to SPC services for ILD inpatients (n, %) | Formal referral pathway to SPC services for ILD outpatients (n, %) | SPC MDT for ILD patients (n, %) | Physician-led SPC clinics for ILD patients (n, %) | Nurse led SPC clinics for ILD patients? (n, %) | Joint palliative care & respiratory clinics for ILD patients? (n, %) |
|---|---|---|---|---|---|---|---|---|
| North East & Yorkshire | 7 | 10 | 7 ( 70.0) | 6 (60.0) | 5 (50.0) | 7 (70.0) | 5 (50.0) | 5 (50.0) |
| North West England | 9 | 15 | 7 (46.7) | 5 (33.3) | 2 (13.3) | 6 (40%) | 5 (33.3) | 4 (26.7) |
| Midlands | 12 | 14 | 7 (50.0) | 11 (64.7) | 3 (21.4) | 4 (28.6) | 4 (28.6) | 3 (21.4) |
| East of England | 1 | 3 | 1 (33.3) | 1 (33.3) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 3 (100.0) |
| South West England | 4 | 6 | 5 (83.3) | 3 (50.0) | 5 (83.3) | 3 (50.0) | 3 (50.0) | 1 (16.7) |
| South East England | 4 | 5 | 2 (40.0) | 2 (40.0) | 2 (40.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| London | 3 | 5 | 3 (60.0) | 5 (100.0) | 3 (60.0) | 4 (80.0) | 3 (60.0) | 2 (40.0) |
| Scotland | 2 | 2 | 2 (100.0) | 1 (50.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Wales | 3 | 3 | 1 (33.3) | 0 (0.0) | 1 (33.3) | 1 (33.3) | 3 (100.0) | 1 (33.3) |
| Northern Ireland | 6 | 8 | 3 (37.5) | 3 (37.5) | 1 (12.5) | 1 (12.5) | 4 (50.0) | 0 (0.0) |
| Unknown | . | 3 | 2 (66.6) | 1 (33.3) | 2 (66.6) | 1 (33.3) | 1 (33.3) | 2 (66.6) |
| **Total** | **51** | **74** | **40 (54.1)** | **38 (51.4)** | **24 (32.4)** | **27 (36.5)** | **28 (37.8)** | **21 (28.4)** |

ILD specific services. Further engagement is needed including research that focuses on qualitative sampling of patient/carer stakeholders in collaboration with community support groups/charities. This survey highlights the need for consensus and guidance in the UK on SPC service provision for ILD patients.

## P17 IMPACT OF VIRTUAL NURSE/PHARMACIST-LED MONITORING IN PATIENTS WITH INTERSTITIAL LUNG DISEASE (ILD) ON ANTIFIBROTIC TREATMENT IN A UK ILD SPECIALIST CENTRE

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10.1136/thorax-2025-BTSabstracts.199

**Background** Antifibrotic medications are prescribed for Idiopathic Pulmonary Fibrosis (Pirfenidone or Nintedanib) and Progressive Fibrosing ILD (Nintedanib) with the aim of slowing disease progression. Unfortunately, side effects of these medications can be significant.

**Aim** To understand if regular ILD nurse/pharmacist (RN/RPh) telephone contact with patients taking antifibrotic medication can positively impact therapeutic tolerance.

**Methods** Retrospective antifibrotic prescribing data was collected from October 2020 to October 2024 from our ILD specialist centre. We analysed patient demographics, duration on antifibrotic therapy and number of RN/RPh telephone contacts.

**Results** Data from 859 patients prescribed antifibrotics was analysed with 462 patients (54%) still on treatment in October 2024. During the study period, a total of 140 patients (16% of cohort) and 35% of patients who discontinued treatment died. Mean duration on treatment (days) in 2021 was 141 vs 305 in 2024; a 116% increase with an average annual rise of 33%. Longest use (days) was at 3192 for Nintedanib and 3127 for Pirfenidone. Since inception, RN/RPh-led monitoring has increased from 769 total contacts in 2021 to 1483 in 2024, an average annual rise of 25%.

**Conclusion** Our preliminary ILD data demonstrates an annual increase in length of antifibrotic treatment since 2020, which could be related to the initiation of virtual RN/RPh-led monitoring. A comparative analysis of pre-initiation data would provide further insights into the role of RN/RPh in improving antifibrotic tolerance, as well as explore other variables that may influence sustained therapy maintenance.

## P18 IMPLEMENTATION OF REGIONAL INTERSTITIAL LUNG DISEASE MULTIDISCIPLINARY MEETINGS BETWEEN TERTIARY AND SECONDARY CARE

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10.1136/thorax-2025-BTSabstracts.200

**Introduction** Interstitial lung diseases (ILD) are a group of progressive conditions with an unpredictable trajectory, requiring both early referral and intervention from specialist care. Diagnosis is often complex and challenging, with many patients being initially misdiagnosed, despite increased awareness. Multidisciplinary (MDT) discussion in previous studies has demonstrated a new or altered pre-existing diagnosis or change in clinical management. Subsequently, effective management of ILD requires a collaborative approach between secondary care and ILD specialist centres.

**Aims and Objectives** To explore how specialist ILD input via regional MDT can improve patient care locally and facilitate earlier appropriate referral to tertiary care. We also hope to improve ILD management with a collaborative approach between secondary care and ILD specialist centres, in line with NHS England directives.

**Methods** From 2023 to mid-2025, regional MDT meetings were organised between the tertiary service and all the major referring trusts. The timing, duration and frequency of the MDT were decided by secondary care, in accordance with local needs. Attendees included an ILD Consultant from tertiary care, local respiratory physicians, radiology and, where possible, rheumatology and respiratory nurses.

Subsequent to successful implementation in 2023/4, 4 criteria were developed and used as part of a clinical audit for 8 referring trusts to identify outcomes for each case discussion. Outcome data was measured from January-June 2025.

**Results** Please see table 1.

**Conclusion** Regional MDM work had a positive impact on care delivery by facilitating the following:

1. Reducing potentially unnecessary referrals to tertiary care (CAT 1 & 2)
2. Limiting travel and cost expenses for patients.
3. Reduced waiting times for specialist centre (CAT 3 & 4) - routine referral waiting time reduced by 8 weeks, urgent referrals now seen 1–2 weeks.
4. Increasing ILD education for consultants and trainees with better pre-referral diagnostic work-up.
5. More patients required a single visit only to tertiary care.
6. Improved interprofessional collaboration with rheumatology and allied healthcare professionals, strengthening multidisciplinary working.
7. Earlier and safe discharge from tertiary service to secondary care.

This model of care effectively aligns with the future requirements of NHS England to provide high-quality care for ILD closer to home.

**Abstract P18 Table 1**

| REGION | CATEGORY-1 | CATEGORY-2 | CATEGORY-3 | CATEGORY-4 | REGION TOTAL |
|---|---|---|---|---|---|
| A | 19 | 41 | 33 | 2 | 95 |
| B | 19 | 49 | 25 | 1 | 94 |
| C | 41 | 48 | 11 | 2 | 102 |
| D | 7 | 25 | 19 | 2 | 53 |
| E | 5 | 14 | 9 | 0 | 28 |
| F | 9 | 22 | 12 | 0 | 43 |
| G | 3 | 15 | 5 | 0 | 23 |
| H | 7 | 2 | 2 | 1 | 12 |
| TOTAL | 110 | 216 | 116 | 8 | 450 |

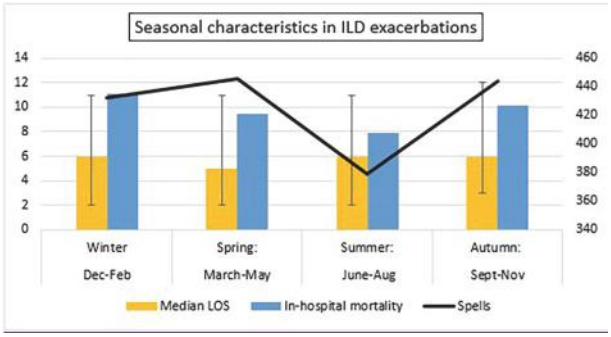
Category:
1. Minimal ILD/ILA/not ILD – no ILD FU required
2. ILD for local care/observation, maybe with guidance/more tests
3. Routine referral to tertiary centre for specialised diagnostic/treatment including antifibrotics
4. Urgent referral for complex/rapidly progressive ILD to tertiary care centre

## P19 SEASONAL PATTERNS OF INTERSTITIAL LUNG DISEASE HOSPITALISATIONS IN A UK TERTIARY CARE CENTRE

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10.1136/thorax-2025-BTSabstracts.201

**Background** Hospitalisations related to interstitial lung disease (ILD) are associated with significant morbidity and healthcare burden. While seasonal variation has been well documented in other respiratory conditions, its influence on ILD-related hospital admissions remains unclear.[1] This study aimed to assess seasonal trends in admission frequency, length of stay (LOS), and in-hospital mortality among patients admitted with ILD.




**Abstract P19 Figure 1**

**Methodology** This retrospective study was conducted at a single UK tertiary ILD centre, analysing all primary-coded ILD related hospitalisations between December 2017 and December 2024. Seasons were defined as winter (Dec-Feb), spring (Mar-May), summer (Jun-Aug), and autumn (Sep-Nov). Outcomes included the number of admissions per season, median LOS, and in-hospital mortality. Statistical comparisons were performed using the Kruskal-Wallis and chi-squared tests.

**Results** A total of 1701 admissions were included (mean age 71.7, SD14.0 years; 50.5% male). The seasonal distribution of admissions was: 432 (25.4%) in winter, 446 (26.2%) in spring, 379 (22.3%) in summer, and 444 (26.1%) in autumn (p=0.213). Median LOS was 6 days (IQR 2–11) in winter, 5 days (IQR 2–11) in spring, 6 days (IQR 2–11) in summer, and 6 days (IQR 3–12) in autumn (p=0.232). In-hospital mortality was11.1% in winter, 9.4% in spring, 7.92% in summer, and 10.1% in autumn (p=0.392). No statistical significant seasonal differences were observed in admission frequency, LOS, or mortality.

**Conclusion** This large single-centre study found no evidence of seasonal variation in ILD-related hospital admissions, length of stay, or in-hospital mortality. These findings suggest that seasonal factors may play a limited role in hospitalisation risk, supporting consistent year-round planning of ILD services. Further research is warranted to explore other drivers of hospitalisation in this population.

## REFERENCE

1. Trends and seasonal variation of hospitalization and mortality of interstitial lung disease in the United States from 2006 to 2016

## P20 HOW DIETETIC INTERVENTIONS ARREST WEIGHT LOSS IN MALNOURISHED PATIENTS WITH FIBROTIC INTERSTITIAL LUNG DISEASE (ILD)

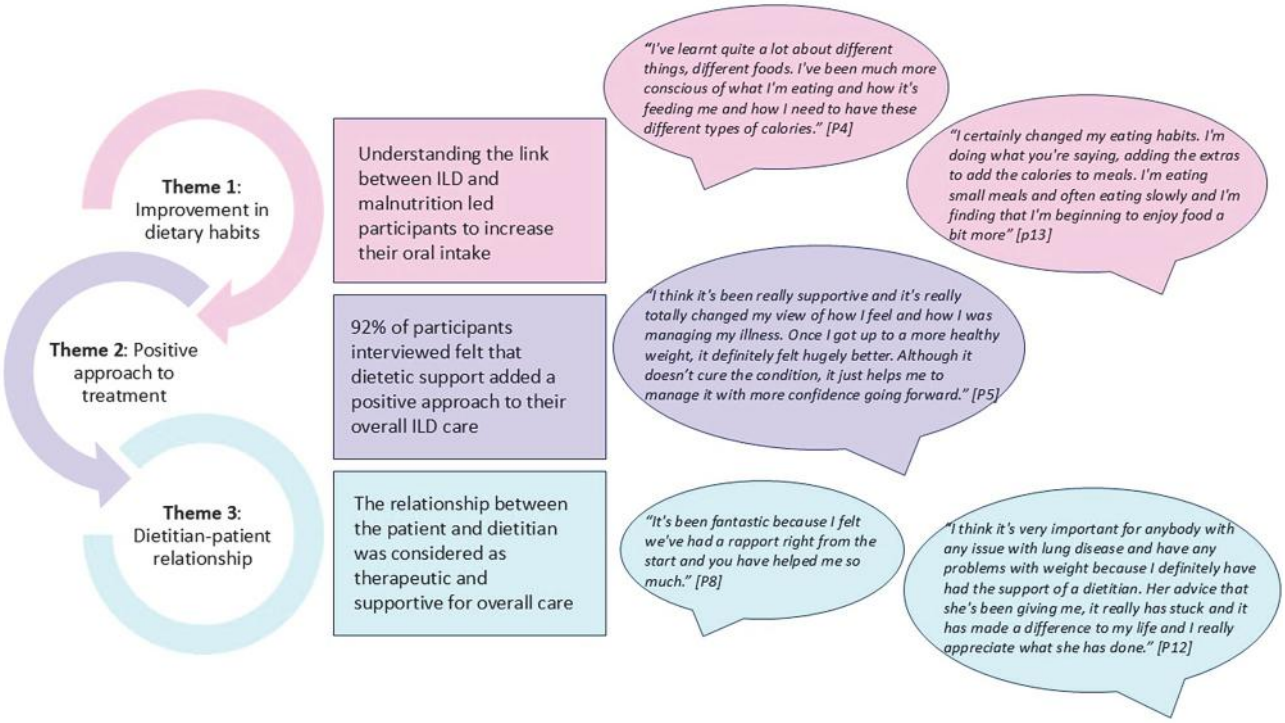
[1]R Kahai, [1]G Castelli, [1]F Danzo, [1]L Ferreira, [1]F Mirabelli, [1]M Morviducci, [1]A Srirangan, [1]C Roberts, [1]C Stock, [1]S Bax, [1]R Hewitt, [1]M Kokosi, [1]P George, [1]F Chua, [1]V Kouranos, [1]P Molyneux, [1]R Jenkins, [1]W Banya, [1]G Korff, [1]A Wells, [2]A Duncan, [3]P Sestini, [1]E Renzoni. *[1]Royal Brompton Hospital, London, UK; [2]Guy's and St Thomas', London, UK; [3]University of Siena, Siena, Italy*

10.1136/thorax-2025-BTSabstracts.202

**Introduction** Malnutrition in ILD is associated with reduced survival. In a pilot feasibility trial (NCT06016959), malnourished patients (n = 40) were randomised to a 12-week individualised dietetic intervention or receiving a dietary leaflet (control). Dietetic interventions but not control reversed ongoing weight loss.

**Aim** To explore the impact of a dietetic intervention trial in malnourished ILD patients.

**Methods** Semi-structured research interviews were conducted with participants at trial end, 13 in the dietetic and four in




**Abstract P20 Figure 1**

the control arm. Participants discussed their experience of dietetic care, research participation, and recommended changes to study design. Interviews were audio recorded, transcribed verbatim and coded thematically (Braun & Clarke, 2006).

**Results** Analysis revealed three key themes: 1) dietary habits were improved to increase weight; 2) a positive approach to treatment was fostered; 3) the dietitian-patient relationship was viewed as supportive, and strengthened the link between ILD and diet. However, some patients reported a feeling of forcing themselves to eat.

Participants randomized to control would have preferred earlier dietetic intervention. However, they did report closer monitoring of their nutritional status. For future research, there was a preference towards digital questionnaires and food diaries.

**Conclusion** Participants viewed dietetic care as highly supportive and valued this to facilitate changing their dietary habits. Although sample size was small, analysis suggests that increased self-monitoring of nutritional status without support does not correlate with positive outcomes.

## P21 IMPACT OF TREATMENT ON QUALITY-OF-LIFE MEASURES IN SARCOIDOSIS

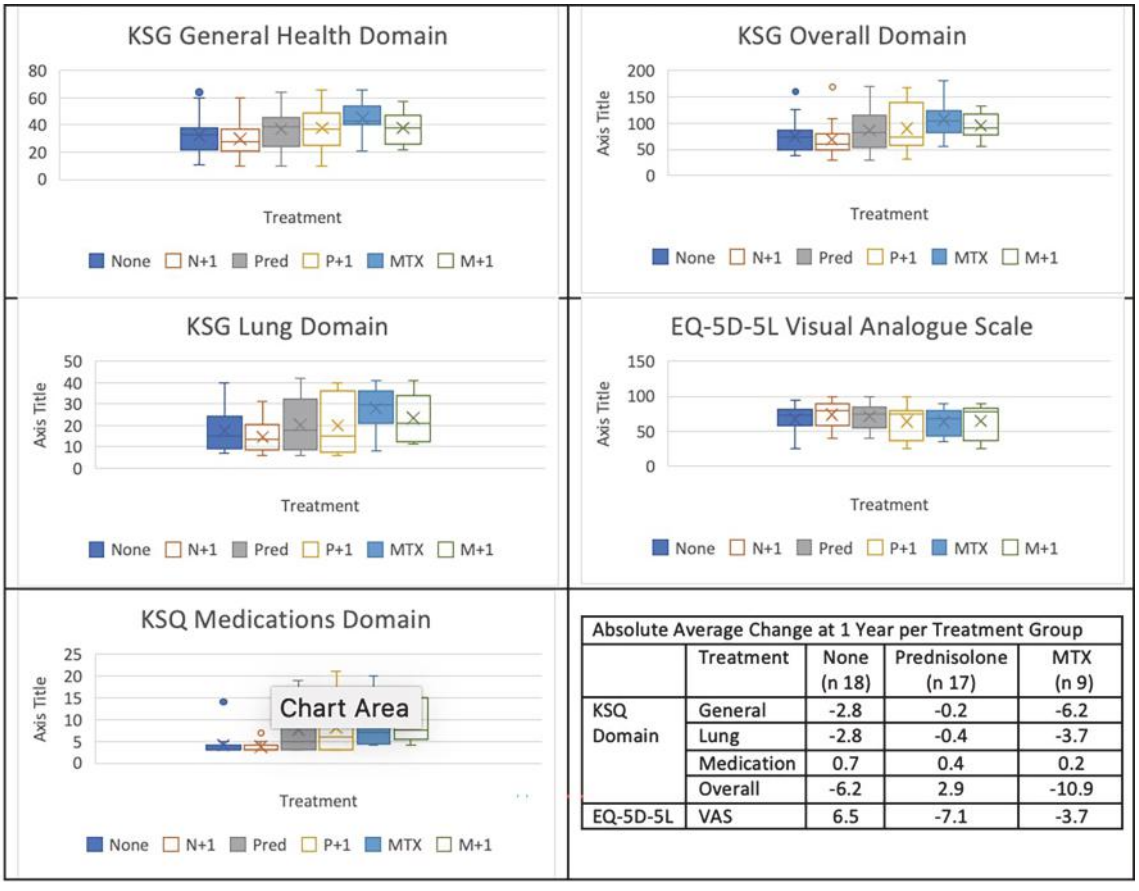
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10.1136/thorax-2025-BTSabstracts.203

**Introduction** Within Interstitial Lung Disease management there is increasing recognition of glucocorticoid toxicity. Recent literature suggests that initial Methotrexate (MTX) treatment may be noninferior to Prednisolone in Pulmonary Sarcoidosis. We aim to examine the impact different treatments have on Patient Reported Outcomes.

**Methods** 116 participants underwent clinical assessment, selected blood testing, plain CXR evaluation and Quality of Life (QoL) measurement including the Kings Sarcoidosis Questionnaire (KSQ) and EQ-5D-5L during enrolment in the TEM Sarcoidosis Project (IRAS 309950). They were followed for 1 year with repeat assessment and investigation. Participants were stratified dependent on treatment; none, single agent Prednisolone, MTX (± additional treatment), Hydroxychloroquine (HCQ), and anti-TNF.

**Results** At enrolment mean age was 59.6 (SD 11.3), 66% were Male, with median disease duration 7.3 years (IQR 3.4–11.9), 42% did not require treatment. There were no significant differences in terms of age of diagnosis, duration of disease, gender and burden of disease (Scadding stage) across all participants. Within the treatment group 31% were treated with single agent Prednisolone (n 26), 10% were treated with MTX (n 12), 11% were treated with HCQ (n 13), and 3% received anti-TNF treatment. QoL measures were globally better in treatment naïve participants. Within the treatment group there were no significant differences between Prednisolone and Methotrexate in terms of age at diagnosis, gender and burden of disease. The Prednisolone group trended towards a longer duration of disease (10.6 v 8.7 p 0.452), whereas the MTX group had higher BMI (28.2 v 34.3 p 0.016). There were no significant differences in QoL measurements between these groups at enrolment. Figure 1 presents Follow up data at one




| Absolute Average Change at 1 Year per Treatment Group | | | | |
|---|---|---|---|---|
| | Treatment | None (n 18) | Prednisolone (n 17) | MTX (n 9) |
| KSQ Domain | General | -2.8 | -0.2 | -6.2 |
| | Lung | -2.8 | -0.4 | -3.7 |
| | Medication | 0.7 | 0.4 | 0.2 |
| | Overall | -6.2 | 2.9 | -10.9 |
| EQ-5D-5L | VAS | 6.5 | -7.1 | -3.7 |

**Abstract P21 Figure 1** Change in King's Sarcoidosis Questionnaire Score, and EQ-5D-5L at Enrolment and 1 Year

year, available for 47 participants. MTX compared favourably with Prednisolone with greater improvements in KSQ domains and a smaller decline in Visual Analogue Scale.

**Conclusion** Raised BMI in the MTX group suggests preferential use as a steroid sparing agent, possibly to limit hyperglycaemic complications. The results need interpreted with caution as most participants were cotreated with Prednisolone or HCQ, nevertheless the data is favourable and gives reassurance of a positive impact.

## P22 PROGRESSION PATTERNS IN NINTEDANIB-TREATED INDIVIDUALS WITH PROGRESSIVE PULMONARY FIBROSIS (PPF)

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10.1136/thorax-2025-BTSabstracts.204

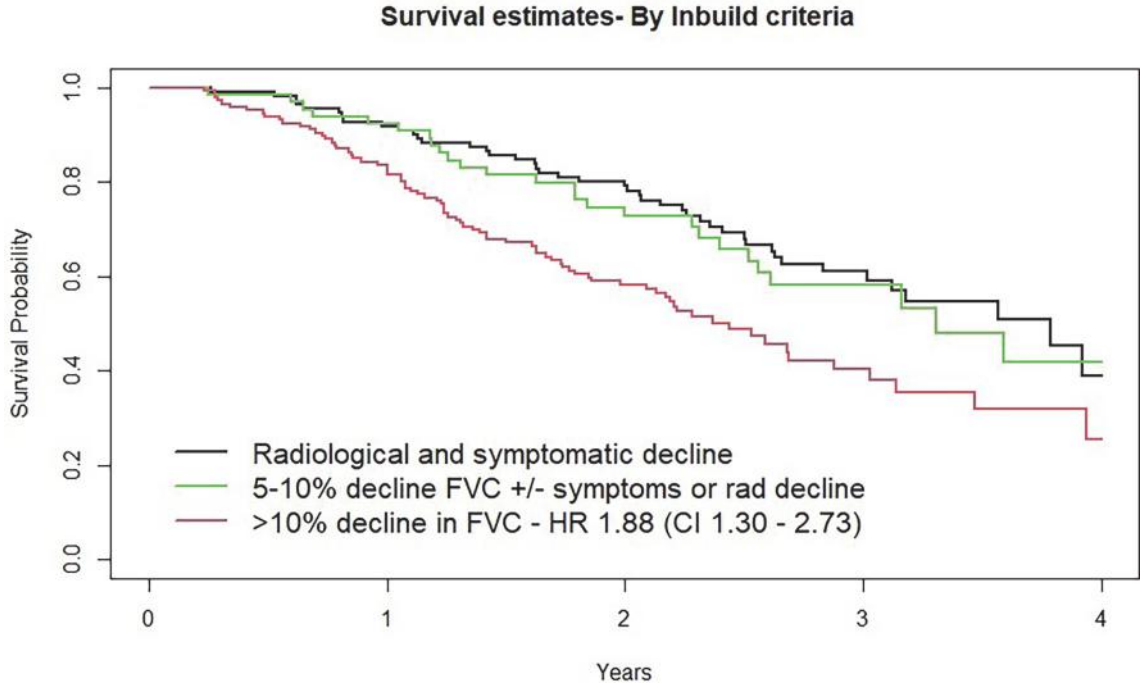
**Introduction** Antifibrotic therapy with nintedanib has become the pharmacologic standard of care for patients with PPF. However, it remains unclear to what extent progression patterns of the underlying interstitial lung disease (ILD) or mortality may be altered by such treatment in practice.

**Methods** A retrospective analysis of nintedanib-treated patients meeting INBUILD criteria for PPF (2018–2023) was undertaken. Baseline was defined as the date of lung function or computed tomography (CT) when criteria for PPF were met. Time-to-event analysis was performed using R Studio.

**Results** 322 patients [50.3% male; median age 69.5 (IQR 61–75)] were included. The commonest ILD subtypes were fibrotic hypersensitivity pneumonitis/FHP and connective tissue disease associated-ILD/CTD-ILD (121 each; 38%). In the overall cohort, ≥10% relative FVC decline was met by 146 (45.3%) patients whose risk of mortality was increased compared to patients (111/322; 34.5%) who met only radiological and symptomatic criteria for PPF [HR 1.88 (CI:1.30–2.73) (figure 1). Progression denoted by ≥15% relative decline in diffusion capacity/TLco and age >65 years at PPF diagnosis were also associated with higher mortality [HR 1.86 (CI:1.24–2.78)] and HR 1.48 (CI:1.19–1.83) respectively]. Compared with FHP, a CTD-ILD diagnosis was associated with a better prognosis [HR 0.65 (CI:0.44–0.97)]. Conversely, patients with unclassifiable ILD had a poorer outcome compared to those with FHP [HR 2.00 (CI:1.07–3.72)]. Nearly a fifth (59/322; 18%) of patients were unable to tolerate nintedanib due to adverse effects.

41 patients (12.7% of cohort) died within a year of satisfying PPF criteria. Patients with a relative decline in ≥10% FVC or ≥15% TLco or who could not complete gas transfer studies in these 12 months were at an increased risk of mortality [HR 3.34 (CI:1.75–6.38)]. A ≥10% relative FVC decline alone over that interval was associated with a higher risk of death [HR 2.82 (CI:1.57–5.07)]

**Conclusions** Survival following satisfaction of PPF criteria varies depending on the progression pattern and the underlying disease. Progression defined by ≥10% relative FVC decline is associated with worse outcome both at baseline and if experienced as a subsequent event after commencing nintedanib therapy.




**Abstract P22 Figure 1**

## P23 NINTEDANIB IN SYSTEMIC SCLEROSIS-ASSOCIATED INTERSTITIAL LUNG DISEASE: REAL-WORLD MULTICENTER COHORT STUDY ON TOLERABILITY AND DISCONTINUATION

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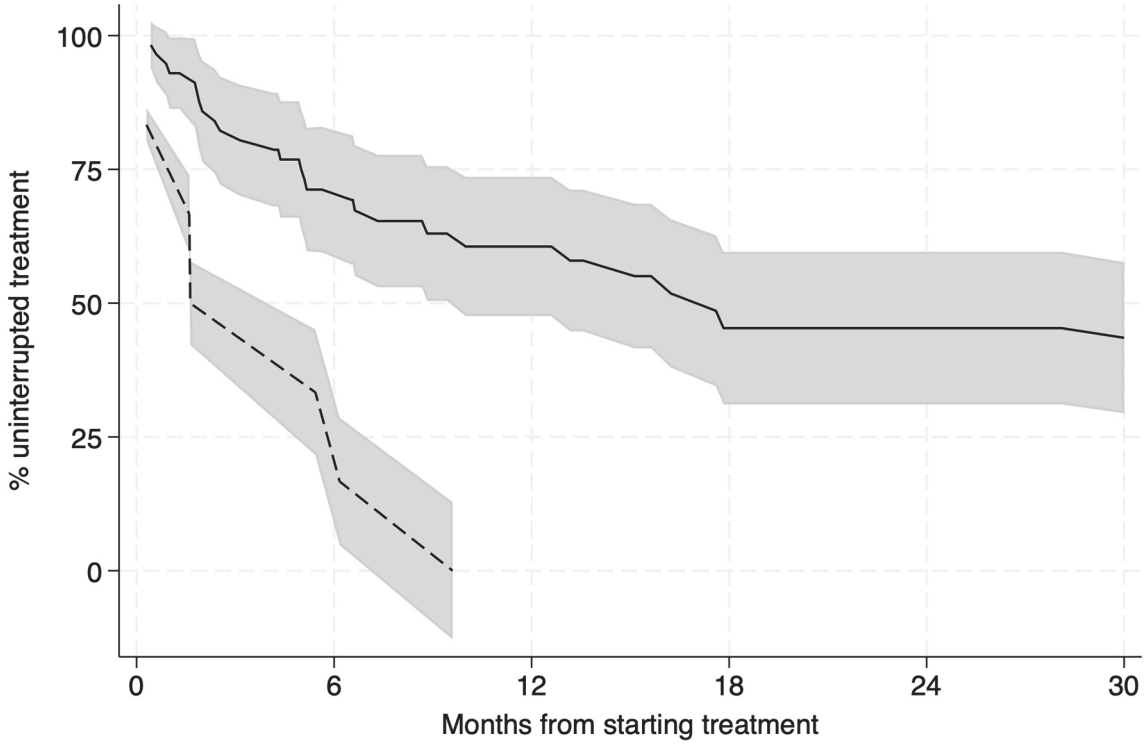
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**Background** Nintedanib slows progression of systemic sclerosis-associated interstitial lung disease (SSc-ILD), but its tolerability is a potential concern.

**Methods** This multicenter study with patients followed up in specialist respiratory and rheumatology units evaluated tolerability and treatment-related interruption in SSc-ILD patients receiving nintedanib. Logistic regression identified factors associated with interruption, with multivariable adjustment for potential confounders. Fisher's exact test was used when complete separation precluded logistic regression. Time to first treatment interruption was analysed by Cox regression. Wilcoxon signed-rank test was used to compare weight changes before and after treatment.

**Results** Among 63 patients (mean age 56.5±13.8 years, 22% male), 76.2% received nintedanib 150 mg twice daily with the remainder 100 mg twice daily. Concomitant mycophenolate mofetil use and baseline gastrointestinal (GI) symptoms were reported in 85.7%. Baseline patient-reported GI symptoms after nintedanib included diarrhoea (14%), reflux (48%), and nausea/vomiting (32%). Over a median follow up of 22.2 months (95% CI 18.1–26.2), 51% of patients interrupted nintedanib due to diarrhoea (33%), nausea/vomiting (21%), reflux (2%), weight loss (9.5%), and abnormal liver function (5%) (multiple reasons allowed). Older age ($p<0.03$) and BMI $<18.5$ kg/m$^2$ ($p=0.024$) were independently associated with interruption. Patients with BMI $<18.5$ kg/m$^2$ was also significantly linked to shorter time to interruption (figure 1), even after adjustment for FVC or CPI and demographic variables. Baseline GI symptoms and concurrent disease-modifying antirheumatic drugs used were not associated with interruption. Most patients who experienced interruption were able to restart treatment, either at reduced or full dose. Permanent discontinuation occurred in 22.2%, with no significant predictors. Mean weight loss over 12 (±6) months after initiation (2.05 kg) was significantly greater than that observed over the same period before treatment (1.09 kg) ($p=0.001$).

**Conclusion** Among patients with SSc-ILD treated with nintedanib, adverse effects are common, and weight loss frequently led to treatment interruptions. Older age and lower BMI were significant predictors of interruption, while no clear predictors of permanent discontinuation were identified, such as baseline GI symptoms, larger studies are needed to confirm these findings. Most patients were able to resume treatment, underscoring the need for close monitoring with access to healthcare professional support.




**Abstract P23 Figure 1**

## P24 COMPARISON OF NINTEDANIB OUTCOMES IN RHEUMATOID ARTHRITIS ASSOCIATED INTERSTITIAL LUNG DISEASE (RA-ILD) AND IDIOPATHIC PULMONARY FIBROSIS (IPF)

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**Background** Nintedanib slows pulmonary fibrosis progression and has been licensed for IPF patients since 2014. Nintedanib is now licensed for treating progressive pulmonary fibrosis of known causes, including rheumatoid arthritis, based on the Inbuild trial. Approximately 25% of patients in the Inbuild study had connective tissue-associated interstitial lung disease (ILD); however, the only immunosuppressant permitted was prednisolone. Disease-modifying antirheumatic drugs (DMARDs) are the mainstay treatment for articular and extra-articular manifestations of rheumatoid arthritis. These include conventional synthetic DMARDs such as Methotrexate and Sulfasalazine as well as biological agents such as Adalimumab and Rituximab. Here, we therefore compare real-world experiences of Nintedanib in IPF and RA-ILD patients, including patients treated with DMARD therapies.

**Methods** We conducted a retrospective study of all patients initiated on Nintedanib for IPF (n=72) and RA-ILD (n=18) from our tertiary centre database between April 2023 and April 2024. Mean values were compared using Wilcoxon rank sum analysis and Chi-squared or Fisher's exact test for categorical data.

**Results** Patients with RA-ILD had more severe disease at treatment initiation compared to those with IPF; FVC (80%±4.1 vs 88%±6.9, p=0.064) and TLCO (48±6.9 vs 58±3.7, p=0.025) (table 1). At 6 months both groups showed relative stability of FVC with changes <5% suggesting treatment efficacy. A greater proportion of RA-ILD patients discontinued Nintedanib (59% vs 36%, p=0.086), however, the total duration of treatment was longer (13.2±3.0 vs 5.0±0.97 months, p<0.001).

**Conclusion** RA-ILD patients tend to start Nintedanib at a later point in their disease compared to IPF, reflecting the differences in licensing. Our results support Nintedanib delaying progression of disease. We demonstrate a higher rate of treatment

**Abstract P24 Table 1** Comparison of Nintedanib outcomes in IPF vs RA-ILD

| Characteristic | IPF N = 72[1] | RA-ILD N = 18[1] | p-value[2] |
|---|---|---|---|
| Age | 76 (8) | 71 (7) | 0.007 |
| Gender | | | 0.2 |
| Male | 56 (78%) | 11 (61%) | |
| Female | 16 (22%) | 7 (39%) | |
| BMI | 28.1 (4.7) | 33.3 (14.4) | 0.11 |
| FVC before Rx (%pred) | 88 (18) | 80 (15) | 0.064 |
| FVC at 6 months (%pred) | 87 (20) | 86 (17) | 0.8 |
| Percentage change in FVC | -3 (10) | 3 (12) | 0.15 |
| TLCOc before Rx (%pred) | 58 (16) | 48 (15) | 0.025 |
| TLCOc after Rx (%pred) | 55 (16) | 49 (15) | 0.2 |
| Percentage change in TLCOc | -14 (49) | -8 (21) | 0.7 |
| Total length of treatment (months) | 5.0 (4.2) | 13.2 (6.5) | <0.001 |
| Treatment stopped | 26 (36%) | 10 (59%) | 0.086 |
| Dose reduced | 15 (21%) | 5 (28%) | 0.5 |
| Death | 12 (18%) | 5 (29%) | 0.3 |
| Side effects | 49 (69%) | 13 (72%) | 0.8 |
| Gastrointestinal side effects | 34 (47%) | 11 (61%) | 0.3 |
| Liver function test derangement | 7 (9.7%) | 2 (11%) | >0.9 |

[1]Mean (SD); n (%)

[2]Wilcoxon rank sum test; Fisher's exact test; Pearson's Chi-squared test

cessation in the RA-ILD group than was seen in the INBUILD study warranting more research to understand whether this relates to disease severity at initiation or the co-prescription of DMARDs.

## P25 IMPACT OF TIERED SERVICE DELIVERY MODEL ON ANTIFIBROTIC ADHERENCE AND MORTALITY AMONG PATIENTS WITH INTERSTITIAL LUNG DISEASE

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**Introduction** Regional models of care using shared care and tiered delivery may tackle health inequalities for patients with interstitial lung disease (ILD) and provide high-standard care closer to home [ILD Care Pathway, One Voice ILD, April 2024]. The Merseyside ILD network have used a shared care tiered service delivery model for several years, with Aintree University Hospital (Tier 3) supporting Tier 1 and 2 sites across the region - see table 1. In anticipation of changes to ILD service delivery, we report the outcomes of shared care within our cohort.

**Methods** We conducted a retrospective observational study of patients with idiopathic pulmonary fibrosis (IPF) and progressive fibrotic ILD (PF-ILD) started on antifibrotic (AF) therapy 01/01/2022–31/12/2023. We collected baseline demographics, spirometry, and outcomes, including AF discontinuation and mortality, until 01/01/2025. Patients were cohorted based on parent hospital tier, and outcomes compared using Kaplan Meier analyses.

**Results** 275 patients commenced AF therapy through our ILD centre (mean age 72, 69% male, 68% on nintedanib). 60 (22%) were followed up by Aintree directly, 125 (45%) by a tier 2 site, and 90 (33%) by shared care with a tier 1 site. Patients with PF-ILD were significantly younger, with significantly lower FEV1, FVC, and TLCO at baseline compared to those with IPF. There was no difference in patient characteristics between tiers.

After exclusion of patients who had physician-recommended AF discontinuation (e.g. due to transaminitis), 1-year adherence was 67% (95% CI 61–74%). While there was no difference in discontinuation rate between tiers, pirfenidone had a significantly higher discontinuation risk compared to nintedanib (HR 1.60, 95% CI 1.05–2.44, p=0.029).

121 patients (44%) died between AF start and end of follow up, with 12-month survival estimated at 79% (95% CI 74–84%). There was no significant survival difference between tiers. Patients who discontinued their AF during the follow up period had a significantly increased mortality risk (HR 1.82, 95% CI 1.23–2.69, p=0.002).

**Conclusions** Our data suggest shared care tiered service delivery for AF delivery is safe, without difference in adherence or mortality between specialist and non-specialist sites. These findings support rollout of tiered service delivery within the UK.

## P26 REAL-WORLD EVALUATION OF PATIENT CHARACTERISTICS IMPACTING TOLERABILITY OF ANTIFIBROTIC TREATMENT IN INTERSTITIAL LUNG DISEASES

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**Background** Pirfenidone and Nintedanib are antifibrotic medications licensed in the UK for Idopathetic Pulmonary Fibrosis (IPF), with Nintedanib also approved for Progressive Fibrosis Interstitial Lung Diseases (PFILD). However adverse drug reactions (ADRs) frequently lead to treatment discontinuation. This study aimed to identify baseline patient characteristics associated with tolerability of antifibrotic therapy.

**Method** A retrospective analysis was conducted at a UK tertiary centre, reviewing all patients initiated on Pirfenidone and Nintedanib between May 2020 and April 2023. Tolerators were defined as patients who continued therapy for at least six months; non-tolerators discontinued within six months. Baseline demographics, pulmonary function tests, comorbidities, blood results and treatment initiation strategies were extracted. Comparative analyses were performed to explore associations between baseline characteristics and

**Abstract P25 Table 1** Overview of services offered by tier status in Merseyside ILD service

| Tier | Description | Staffing | ILD MDT | Prescribing Centre |
|---|---|---|---|---|
| Tier 3 | NHSE commissioned specialist ILD service with a dedicated ILD clinic | • Specialised respiratory consultants with expertise in ILD<br>• Specialised ILD nurses<br>• Specialised thoracic radiologists | Regional | Yes |
| Tier 2 | Dedicated ILD clinic within respiratory directorate | • Local ILD lead respiratory consultant<br>• Specialised respiratory nurse with expertise in ILD<br>• Tier 3 sites provide specialised ILD nurse and pharmacist support | Local | Can be prescribing or non-prescribing (all Merseyside sites non-prescribing) |
| Tier 1 | ILD care delivered within general respiratory clinica | • Respiratory consultant without expertise in ILD<br>• Tier 3 sites provide specialised ILD nurse and pharmacist support | None | No |

tolerability outcomes. ADRs were classified as any undesirable effects attributed to the antifibrotic medications, as recorded in follow up consultations. Dispensing records were cross-referenced to verify dosing history and treatment discontinuation.

**Results** Among 260 patients included (72% IPF, 28% PFILD), 75% (n=195) were classified as tolerators. Tolerability was higher with Nintedanib than Pirfenidone (79.6% vs. 67.3%; p=0.027). Among tolerators, ADRs were common (59%) with no difference between nintedanib and pirfenidone. Age was the only baseline demographic associated with tolerability (mean age 72.4 vs. 74.8 years, p=0.44). Sex, body mass index, ethnicity, smoking status, and lung function (FVC and DLCO) showed no significant associations. Comorbidities including hypertension, diabetes, gastrointestinal disorders, anxiety and depression, and ischaemic heart disease were

| | | Tolerators (n=195) | Non-Tolerators (n=65) | *p*-values |
|---|---|---|---|---|
| **Antifibrotic Indication** | IPF, n (%) | 135 (72.19%) | 52 (27.81%) | 0.094 |
| | PFILD, n (%) | 60 (82.19%) | 13 (17.18%) | |
| **Antifibrotic Medication** | Nintedanib, n (%) | 129 (79.6%) | 33 (20.3%) | 0.027 |
| | Pirfenidone, n (%) | 66 (67.3%) | 32 (32.7%) | |
| **Tolerators with ADRs Reported** | Nintedanib, n (%) | 74 (57%) | - | 0.550 |
| | Pirfenidone, n (%) | 41 (62%) | - | |
| **Initiation Dosing schedules** | Nintedanib Standard Initiation, n (%) | 111 (77.63%) | 32 (22.38%) | 0.112 |
| | Nintedanib Reduced Dose Initiation, n (%) | 16 (94.12%) | 1 (5.88%) | |
| | Pirfenidone Standard Initiation, n (%) | 195 (65.4%) | 65 (34.52%) | |
| | Pirfenidone Gradual up-titration schedule on Initiation, n (%) | 55 (76.92%) | 29 (23.08%) | |
| **Demographics** | Age (mean) years | 72.4 (8.78) | 74.8 (6.67) | 0.044 |
| | Male, n (%) | 142 (72.8%) | 47 (72.3%) | 0.936 |
| | White, n (%) | 148 (77.1%) | 48 (76.2%) | 0.810 |
| | Asians (including Indian, Pakistani, Bangladeshi, and Chinese), n (%) | 33 (17.2%) | 10 (15.9%) | |
| | Black, n (%) | 11 (5.73%) | 5 (23.8%) | |
| | BMI, kg/m$^2$, mean (SD) | 27.97 (6.10) | 26.66 (7.21) | 0.211 |
| **Smoking Status** | Current and ex-smoker, n (%) | 105 (56.45%) | 31 (48.44%) | 0.267 |
| **Baseline Spirometry** | FVC, % predicted (SD) | 67.6% (13.02) | 66.38% (13.25) | 0.485 |
| | DLCO, % predicted (SD) | 55.80% (19.55) | 51.41 (10.28) | 0.480 |
| **Baseline Blood Report** | Urea, mmol/L, mean (SD) | 6.07 (2.04) | 6.79(5.68) | 0.142 |
| | Creatinine, µmol/L, mean (SD) | 84.1 (1.9) | 64 (3.58) | 0.884 |
| | Alanine Aminotransferase, U/L, mean (SD) | 24.1 (14.3) | 28 (23.3) | 0.117 |
| | Alkaline Phosphatase, U/L, mean (SD) | 86.9 (28.9) | 88.2 (27.6) | 0.767 |
| | Bilirubin, µmol/L, mean (SD) | 10.77 (7.29) | 10.63 (4.81) | 0.894 |
| | Albumin, g/L, mean (SD) | 43.94 (3.43) | 43.62 (3.50) | 0.532 |
| **Comorbidities** | Hypertension, n (%) | 75 (79.8%) | 19 (20.2%) | 0.180 |
| | Diabetes, n (%) | 40 (71.43%) | 16 (28.57%) | 0.486 |
| | Ischaemic Heart Disease, n (%) | 34 (69.4%) | 15 (30.6%) | 0.314 |
| | Gastrointestinal Disorders, n (%) | 27 (84.37%) | 5 (15.6%) | 0.282 |
| | Anxiety / Depression, n (%) | 13 (6.67) | 7 (10.77) | 0.282 |
| **Concurrent Medications** | Immunomodulators, n (%) | 18 (9.23%) | 8 (12.3%) | 0.474 |
| | Proton pump inhibitors (PPIs) , n (%) | 111 (73.5%) | 40 (26.49%) | 0.514 |
| | Antiplatelet Medications, n (%) | 55 (75.3%) | 18 (24.66%) | 0.936 |
| | Oral corticosteroids, n (%) | 28 (14.36%) | 4 (6.15%) | 0.081 |
| **Healthcare Professional who Initiated Antifibrotic Therapy** | Consultant, n (%) | 5 (2.58%) | 3 (4.62%) | 0.613 |
| | Specialist Pharmacist, n (%) | 123 (63.4%) | 37 (56.92%) | |
| | Specialist Registered Nurse, n (%) | 65 (33.51%) | 24 (36.92%) | |
| | Respiratory Registrar, n (%) | 1 (0.52%) | 1 (1.54%) | |

**Abstract P26 Figure 1** Correlation between patient characteristics, clinical features, initiation dosing on antifibrotic therapy tolerance. This table shows statistical association between baseline patient characteristics (age, gender, body mass index (BMI), ethnicity), clinical parameters (pulmonary function results, comorbidities, blood report), antifibrotic initiation strategies (dosing schedules, and initiating healthcare professional) with antifibrotic tolerability, defined as continuation of therapy for at least 6 months. Values represent averages, proportions or percentages corresponding to p-values to indicate significance

similarly distributed between groups. No significant associations were found between tolerability and baseline blood markers, or concurrent medications such as proton pump inhibitors, antiplatelet medications, immunomodulators or corticosteroids. Antifibrotic initiation strategy (standard vs. reduced dose) and prescriber type (consultant, specialist pharmacist, or nurse) did not impact tolerability.

**Conclusion** In this real-world cohort, antifibrotic therapy was tolerated by most patients, with better persistence observed in younger individuals and those on Nintedanib. Other baseline factors showed no clear association with tolerability.

## P27 CAN ANTIFIBROTIC SIDE-EFFECT DECISION AIDS IMPROVE PATIENT TOLERANCE AND CONTINUATION OF ANTIFIBROTIC MEDICATION?

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**Background and Aims** Antifibrotic treatments for idiopathic pulmonary fibrosis (IPF) and progressive pulmonary fibrosis (PFILD) have several side effects which can limit patients'

**Abstract P27 Table 1**

| | Pre-Decision Aids n=50 | Post-Decision Aids n=50 | p=value |
|---|---|---|---|
| **Diagnosis** | | | |
| IPF | 39(78%) | 42(84%) | 0.611 |
| PFILD | 11(22%) | 8(16%) | |
| **Antifibrotic** | | | |
| Nintedanib | 45(90%) | 47(94%) | 0.715 |
| Pirfenidone | 5(10%) | 3(6%) | |
| **Initial Dose** | | | |
| Full | 42(84%) | 36(72%) | 0.227 |
| Reduced | 8(16%) | 14(28%) | |
| **Demographics** | | | |
| Age (years)* | 75.8(73.4 – 78.2) | 73.5(71.7 – 75.4) | 0.139 |
| BMI (kg/m$^2$)* | 28.3(27.1 – 29.5) | 29.3(28.0 – 30.6) | 0.260 |
| **Changes to antifibrotic medication within 3 months** | | | |
| No changes | 28(56%) | 37(74%) | 0.059 |
| Break for GI side effects | 17(34%) | 5(10%) | **0.038** |
| Break for deranged LFT | 2(4%) | 0 | 0.495 |
| Break for other reasons | 3(6%) | 3(6%) | 1.000 |
| Dose increase | 0 | 5(10%) | **0.001** |
| **Outcome at 3 months** | | | |
| Full dose | 31(62%) | 39(78%) | 0.081 |
| Reduced dose | 7(14%) | 8(16%) | 0.779 |
| Permanent stop | 12(24%) | 3(6%) | **0.023** |
| **Contacts outside AF Pathway** | | | |
| No contacts | 23(46%) | 37(74%) | **0.004** |
| 1 | 18(36%) | 5(10%) | **0.002** |
| 2+ | 9(18%) | 8(16%) | 0.790 |

**mean(95%confidence interval)*

ability to continue treatment. A previous service evaluation at our Tertiary ILD centre identified gastrointestinal (GI) side effects and deranged liver function tests (LFTs) as the main causes of patients permanently stopping medication. Due to this we developed an Antifibrotic Side-effect Decision Aid for use by all team members to help identify severity, standardise advice and guide management of common side effects, including pages for diarrhoea, nausea and abnormal LFTs. The Decision Aids were presented at local and regional meetings from August 2023 and approved by local clinical governance committees prior to deployment in May 2024. We aimed to review the impact of the Decision Aids on antifibrotic tolerance, and impact on clinical contacts outside our established antifibrotic pathway.

**Methods** We reviewed 2 cohorts of consecutive patients who initiated antifibrotic medication at our Tertiary ILD centre, 50 pre-Decision Aid between May-July 2023 and 50 post-Decision Aid between July-October 2024. We reviewed medical records for 3 months after starting medication and identified any dose changes, reasons for changes, and patient outcomes at 3 months. We documented contacts to the specialist nurses outside our standard antifibrotic pathway via our telephone contact line. Chi-squared or Fishers exact tests were used as appropriate for group comparisons.

**Results** There were no significant differences in antifibrotic used, initial dose, age or BMI between the pre and post-Decision Aid cohorts (table 1). Fewer patients had treatment breaks for GI side effects in the post-Decision Aid cohort ($p=0.038$) with more patients increasing their dose ($p=0.001$). Fewer patients permanently stopped medication in the post-Decision Aid cohort (6% vs 24%, $p=0.023$). There was no increase in patient contacts outside the established antifibrotic pathway, with a higher proportion of patients not utilising the contact line within the first 3 months ($p=0.004$).

**Discussion** The Decision Aids have had a positive impact on patient tolerance and treatment continuation during the initial 3-month period without a significant increase in patient contacts to the specialist nurse contact line.

## P28 LITTLE WOMEN LOOKING INTO ANTIFIBROTIC TOLERANCE IN RELATION TO WEIGHT, DOSE, FEMALE GENDER AND ETHNICITY

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**Introduction** Nintedanib is an antifibrotic medication indicated for progressive fibrotic lung disease. Patients are usually started on full dose Nintedanib irrespective of their age, gender, body mass index (BMI) or ethnicity. Many patients are intolerant of full dose and medication is either reduced or discontinued. Our aim was to demonstrate clinically available predictive factors of full dose intolerance in a real-world cohort.

**Method** We reviewed 127 patients initiated on Nintedanib at a single prescribing centre between December 2021 and October 2023. Patient data was obtained from electronic clinical records. Data was collected until there was dose reduction, discontinuation, or the last clinical entry prior to completion of the review in March 2025.

The interval from Nintedanib initiation to the point of data collection was between 20–39 months.

**Results** 51 patients (39.8%) tolerated 150 mg BD Nintedanib throughout, 71 (55.4%) required dose reduction and 5 (3.9%) were initiated at 100 mg.

Female gender and an IPF diagnosis were significant predictive factors of intolerance of nintedanib at full dose ($p<0.001$

**Abstract P28 Table 1** Demographics and results

| | Reduced dose Nintedanib (n=76) | Full dose Nintedanib (n=51) | p value |
|---|---|---|---|
| | | | |
| **Age at start of treatment (n, sd)** | 71.45 (7.36) | 70.37 (9.85) | 0.051 |
| **Gender (n, %)** | Female = 25 (32.9%)<br>Male = 51 (67.1%) | Female = 9 (17.6%)<br>Male = 42 (82.4%) | <0.001 |
| **Ethnic group (n, %)** | Caucasian = 49 (98%)<br>Other ethnicities = 1 (2%)<br>*(26 missing values)* | Caucasian = 30 (100%)<br>Other ethnicities = 0<br>*(21 missing values)* | 0.119 |
| **Diagnosis (n, %)** | IPF = 50 (65.8%)<br>PF-ILD = 26 (34.2%) | IPF = 28 (54.9%)<br>PF-ILD = 23 (45.1%) | 0.042 |
| **Weight at baseline (n, sd)** | 79.06 (16.04)<br>*(3 missing values)* | 88.08 (14.15) | 0.503 |
| **BMI at baseline (n, sd)** | 27.78 (4.60)<br>*(3 missing values)* | 30.55 (5.07) | 0.615 |
| **FVC % predicted at baseline (n, sd)** | 75.55 (15.47) | 75.02 (15.45) | 0.771 |
| **TLCO % predicted at baseline (n, sd)** | 49.48 (14.59)<br>*(2 missing values)* | 53.78 (15.17)<br>*(2 missing values)* | 0.677 |

IPF = Idiopathic Pulmonary Fibrosis; PF-ILD = Progressive Fibrosing Interstitial Lung Disease

and p=0.042); older age was a near significant predictive factor (p=0.051).

Treatment was given for a mean of 26.52 months (SD=5.38) with dose reduction occurring after 8.14 months (SD=6.72). Discontinuation occurred after 19.59 months (SD=6.82).

Nintedanib was paused or reduced due to diarrhoea (39.4%), deranged liver function (9.2%) and weight loss (5.7%).

Nintedanib was discontinued in 27 patients and was due to diarrhoea 38.4%, death 34.6%, weight loss 15.3%, patient choice 7.7% and nausea in 3.8%.

20 patients passed away by the time of review with 10 in each group.

**Conclusion** This single centre, retrospective real-world study has demonstrated significant rates of side effects and intolerance to Nintedanib 150 mg BD.

It suggests that female gender and older age are predictive factors of intolerance of full dose Nintedanib.

These findings warrant further investigation in a UK wide cohort which is planned for 2026, to further explore and consolidate these findings and potentially highlight others including ethnicity which wil be vital to guide clinicians to enable prescription of a dose that will be better tolerated from the outset and improve patient experience.

## P29 EFFICACY OF PULSED CYCLOPHOSPHAMIDE IN INTERSTITIAL LUNG DISEASE

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**Introduction** Interstitial lung disease (ILD) is characterised as a group of conditions that cause inflammation and fibrosis in the lung. Cyclophosphamide (CYC) is considered for patients with rapidly worsening ILD to potentially slow down the progression of the lung disease. The use of cyclophosphamide is fraught with concerns due to efficacy and side effects and the decision to treat is based on a risk to benefit ratio.

**Objectives** To assess the role and tolerability of intravenous (IV) pulsed cyclophosphamide in patients with ILD.

**Methods** We retrospectively analysed ILD patients treated with CYC at our centre from 2021 to 2024. Response to treatment was correlated with lung functions and extent of radiological involvement. Scans were analysed by cardiothoracic radiologist based on the extent of ground glass opacity or fibrosis on CT scans. Data are presented as median (IQR).

**Results** A total of 21 patients were included. The median age was 62 years (56–72); 11 were male. Diagnoses included CTD-ILD (n=6), chronic hypersensitivity pneumonitis (n=7), fibrotic NSIP (n=2), fibrotic organising pneumonia (n=3), PR3-positive vasculitis (n=1), progressive sarcoidosis (n=1), and unclassified progressive fibrosis (n=1). The median follow-up duration was 38 (20–61) months.

All patients underwent pre-treatment investigation as per requirements. CYC doses ranged from 10–15 mg/kg; everyone received concurrent intravenous pulsed methylprednisolone (500 mg to 1 g daily for 3 days). The median FVC reduced from 2.2 L to 2.1 L, however there was an improvement in FVC predicted% from 59% to 63%. There was no change in the TLCO 39% (33–48%) to 38.5% (20–34%) pre and post treatment. Treatment was discontinued in two patients due to haematuria and neutropenia. Side effects included haematuria, neutropenia, diarrhoea and abdominal pain, nausea, and chest infection. The median duration between treatment initiation and follow-up imaging was 9 (6–14) months. Correlation between lung function and radiology following treatment are shown in table 1.

**Conclusion** There was improvement in both FVC and TLCO in patients treated with CYC if the fibrosis is or GGO was < 50% on imaging. The values were difficult to compare in the more extensive group due to small numbers.

**Abstract P29 Table 1**

| Group | ΔFVC(L) | ΔFVC (%) | ΔTLCO (%) |
|---|---|---|---|
| Fibrosis < 50% (n=14) | +0.19 | +7.0 | +11.7 |
| Fibrosis > 50% (n=1) | +1.19 | +28.0 | +6.0 |
| GGO < 50% (n=10) | +0.19 | +6.1 | +14.6 |
| GGO > 50% (n=5) | - 0.03 | -1.0 | -2.0 |

FVC: Forced Vital Capacity, GGO:Ground-Glass opacity, TLCO: Transfer Factor for Carbon Monoxide

## P30 EFFECT OF DIETARY NITRATE SUPPLEMENTATION ON EXERCISE PERFORMANCE IN HYPOXIC IPF: THE EDEN-OX 3 STUDY

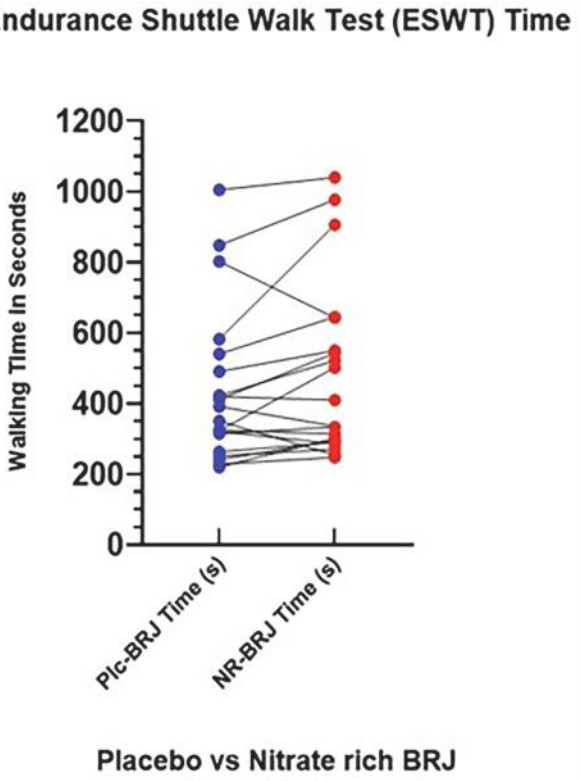
[1,2,3]M Majrshi, [2,4]A Alzahrani, [2,1]P George, [1,2]K Philip, [5]A Alsulayyim, [1,2]L Price, [1,2]M Polkey, [1,2]N Hopkinson. *[1]National Heart and Lung Institute, Imperial College London, London, UK; [2]Respiratory Medicine, Royal Brompton and Harefield Hospitals, Guy's and St Thomas' NHS Foundation Trust, London, UK; [3]Respiratory Therapy Unit, King Abdulaziz University Hospital, Jeddah, Saudi Arabia; [4]Respiratory Therapy Department, Medical Rehabilitation Sciences College, Taibah University, Medinah, Saudi Arabia; [5]Respiratory Therapy Program, Department of Nursing, College of Nursing and Health Sciences, Jazan University, Jazan, Saudi Arabia*

10.1136/thorax-2025-BTSabstracts.212

**Background** Idiopathic pulmonary fibrosis (IPF) is a progressive interstitial lung disease characterised by breathlessness and reduced exercise capacity. Dietary nitrate ($NO_3^-$) supplementation, in the form of beetroot juice (BRJ), increases nitric oxide (NO) bioavailability, which may enhance vascular function and oxygen delivery. This study investigated the acute effects of nitrate-rich BRJ (NR-BRJ) supplementation on exercise performance in individuals with confirmed IPF.

**Methods** Patients with IPF who required ambulatory oxygen or demonstrating exercise-induced desaturation were enrolled in a randomized, double-blind, placebo-controlled crossover study. Each participant completed screening followed by two interventional visits. Participants received 140 mls nitrate-rich beetroot juice (NR-BRJ) on one occasion and nitrate-depleted placebo beetroot juice (Plc-BRJ) at the other, with a 7–30 day washout period. Primary endpoint was the Endurance Shuttle Walking Time (ESWT). Endothelial function was assessed using flow-mediated dilatation (FMD). Data normality was assessed with the Shapiro-Wilk test. Paired t-tests and Wilcoxon signed-rank tests were applied as appropriate.

**Results** The cohort included 20 patients with IPF (mean age: 70.3 ± 11.3 years; 75% male). Mean FVC was 3.21 ± 0.75 L (88% ±16.3% predicted) and TLco 48.6 ± 7.9% predicted. ESWT performance improved by median difference of 31 s (IQR: –9.5 to 100.0; p = 0.043); with small to moderate effect (d = 0.44). Median [IQR] values were 373 s [291.3–619.8] for NR-BRJ and 370.5 s [276.0–527.5] for Plc-BRJ. A statistically significant increase in endothelial function, as measured by FMD, was observed 180 minutes post-consumption (NR-BRJ: +2.97 ± 5.46%; Plc-BRJ: –0.71 ± 4.72%; $p$ = 0.043).




**Abstract P30 Figure 1**

**Conclusion** These findings suggest benefit from acute dietary nitrate supplementation with NR-BRJ in IPF patients with hypoxia on exercise and vascular function, consistent with previous findings in COPD and pulmonary hypertension. Longer term studies are needed to see if this translates into sustained clinical benefit.

# Enhancing outcomes in chronic respiratory disease: the role of rehabilitation and physiotherapy

## P31 A QUALITATIVE EXPLORATION OF THE PERCEPTION OF MAINTENANCE NEEDS THREE MONTHS AFTER REHABILITATION IN PATIENTS WITH LONG COVID

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10.1136/thorax-2025-BTSabstracts.213

**Introduction and Objectives** Existing evidence suggests there is an appetite for a structured maintenance programme following Covid rehabilitation to maintain functional ability.[1] The objective of this study was to explore whether, three months on, this perception had changed, and whether maintenance warrants further exploration.

**Methods** Covid rehabilitation completers were invited for a face-to-face visit three months following discharge. During the visit, patients completed objective outcomes (previously reported[2]), and a semi-structured interview. Interviews, conducted by a research assistant were recorded and transcribed verbatim. Reflexive thematic analysis was conducted using NVivo (V1.6.1), and themes reviewed by two research team members who specialise in qualitative research.

**Results** A total of 19 interviews were conducted (mean age 57.8±7 years; 79% white; 74% women).

Three themes were generated (figure 1):

1. I've got what I need to proceed (with caution)

Participants felt they had progressed, had good understanding of their symptoms and how to manage them, and therefore had the confidence to proceed exercising with caution, taking into account their fatigue.

2. I've found the onward path that works for me

Participants felt motivated to continue exercising in the way they had chosen. Individualised options were selected based on accessibility, what mattered to them, and how it worked alongside their own routine.

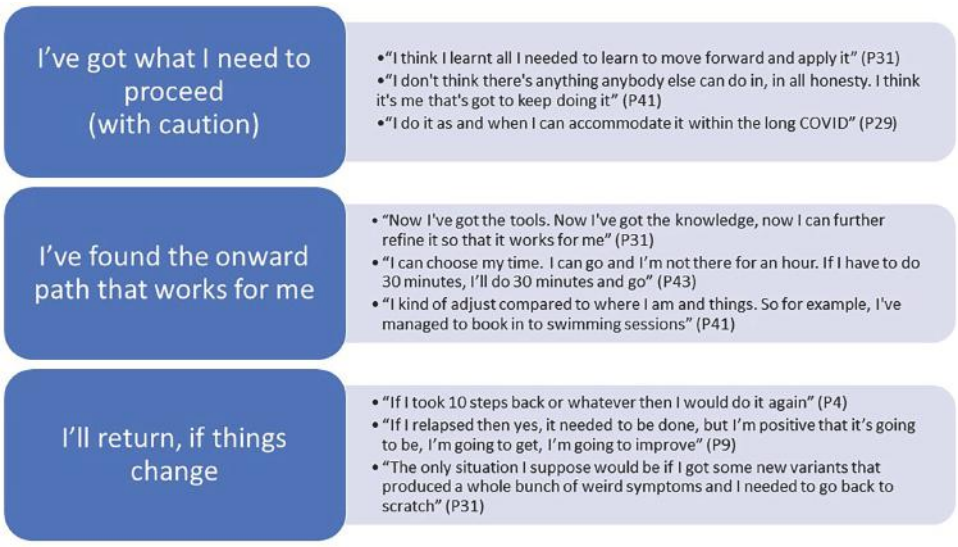
3. I'll return, if things change

Patients felt symptoms had improved, but accepted they remained variable. They appreciated this may be the case and expressed appetite to return to rehabilitation should things change.

**Conclusion** Despite rehabilitation completers expressing an appetite for maintenance at point of discharge, three months later, most continued exercising independently, albeit with caution. Participants only felt they would need further input if there was a change to their symptoms, suggesting rehabilitation provided the skills and knowledge for effective onward self-management of Long Covid.

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2. Hong A, *et al*. 'P28-Incremental and endurance shuttle walk test performance and functional assessment of chronic illness therapy fatigue scale are maintained or improved at 3-months following a 6-week in-person or online covid rehabilitation programme'. *Thorax*. 2023;**78**(Suppl 4), A123-A123.




**Abstract P31 Figure 1** Themes with illustrative quotes

## P32 PREDICTING ENROLMENT TO PULMONARY REHABILITATION IN THE UK

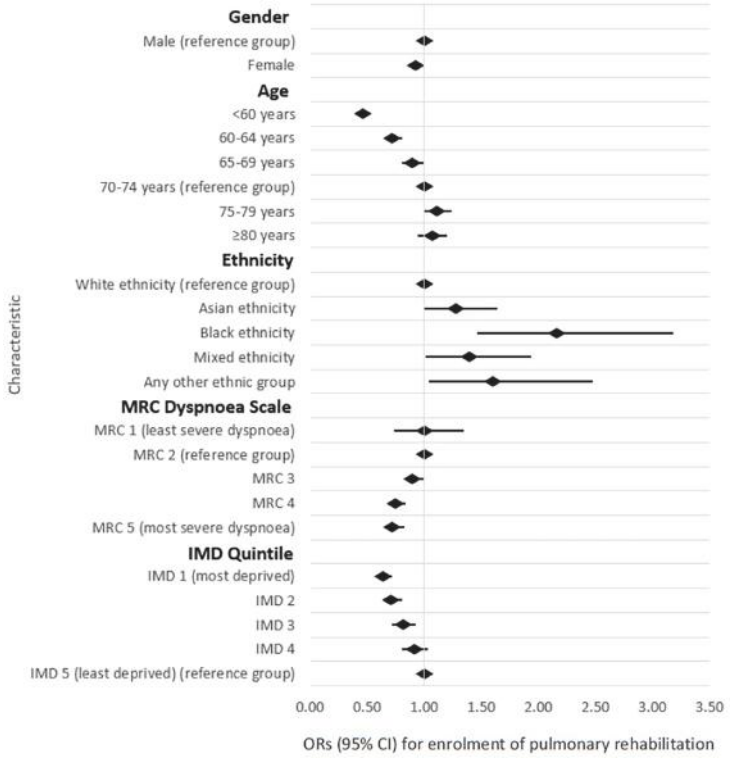
[1,2]H Drover, [1,2]L Wang, [1,2]MW Orme, [1,2]M Richardson, [3]JK Quint, [1,2]R Evans, [1,2]SJ Singh, [1,2]E Daynes. *[1]Department of Respiratory Sciences, University of Leicester, Leicester, UK; [2]Centre for Exercise and Rehabilitation Science, NIHR Leicester Biomedical Research Centre – Respiratory, University Hospitals of Leicester NHS Trust, Leicester, UK; [3]School of Public Health, Imperial College London, London, UK*

10.1136/thorax-2025-BTSabstracts.214

**Background** Despite the known benefits of Pulmonary Rehabilitation (PR), a significant proportion of those that attend an assessment do not enrol in a programme. The characteristics of those declining a programme remain unexplored.

**Aim** To compare the characteristics of those enrolling into a PR programme using the UK National Respiratory Audit Programme dataset for England and Wales.

**Methods** 88,446 individuals with COPD attended a PR assessment between March 2019 and September 2023. A




**Abstract P32 Figure 1** Plot showing odds ratios for enrolment into a pulmonary rehabilitation programme in individuals living with COPD that attended a pulmonary rehabilitation assessment by characteristic. Reference groups for each characteristic are shown with an odds ratio of 1. MRC: Medical Research Council; IMD: index of multiple deprivation

generalised linear mixed model with a logit link function was used to explore the characteristics associated with PR enrolment. Factors included age, gender, ethnicity, Medical Research Council (MRC) dyspnoea grade and index of multiple deprivation (IMD) quintile. Enrolment was defined as attending at least one PR session in any mode of delivery.
**Results** 72,085 (82%) individuals that attended a PR assessment enrolled into a PR programme. Individuals that identified as having a Black ethnicity had significantly greater odds of enrolling in a PR programme. Age<65 years, female gender, MRC grade 4/5 or IMD1–3 quintile groups had significantly lower odds of enrolment (figure 1). Individuals aged<60 years, those from the most deprived areas (IMD1) or with an MRC grade 5 were 54% (49–58), 36% (28–44) and 28% (18–36) less likely to enrol into a programme respectively.
**Conclusions** There are inequities in the enrolment of a PR programme across characteristics, particularly in age, deprivation and MRC grade. Approaches targeted at individuals with characteristics less likely to enrol in PR may facilitate their access to optimal care.

## P33 THE USE OF LAY HEALTH WORKERS IN SUPPORTING PATIENTS WITH COPD TO ATTEND PULMONARY REHABILITATION: A QUALITATIVE STUDY EXPLORING THE PATIENT EXPERIENCE

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10.1136/thorax-2025-BTSabstracts.215

**Introduction and Objectives** Pulmonary rehabilitation (PR) is the most effective treatment for the symptoms of chronic obstructive pulmonary disease (COPD), yet uptake and completion are low. The IMPROVE trial aimed to assess the effectiveness of recruiting and training patients who had completed PR to become lay health workers (i.e., PR-buddies), to support patients newly referred to PR. PR-buddies were trained to assess potential barriers to participation and help patients overcome them, emphasise the benefits of PR, and provide advice. The aim of this study was to explore the experiences of patients who received support from PR-buddies and the acceptability of PR-buddies from patients.
**Methods** Patients who participated in the IMPROVE trial were invited to an interview to discuss their experiences. Interviews were semi-structured, audio-recorded, and transcribed verbatim for coding and inductive thematic analysis.
**Results** Twenty patients from 11 IMPROVE trial sites across England participated. Three interviews were in-person, and the rest conducted by phone. Patients were happy with the support provided by their PR-buddy, which increased their confidence in attending PR. Patients liked the fact that PR-buddies had completed PR previously and so understood how they were feeling and could explain what to expect. Patients valued PR-buddies with good communication skills. The timing and mode of contact between PR-buddies and patients varied. Most patients preferred in-person contact initiated before the start of PR, and when subsequent contact was regular, predictable, and reliable. Patients largely accepted PR-buddies as strangers and appreciated their volunteer and lay-person status, as well as the fact they also had COPD, which made them more relatable. Additionally, patients enjoyed talking to and building relationships with their PR-buddies which alleviated low mood and isolation.
**Conclusions** Patients thought the PR-buddy service was a good idea and found PR-buddy support acceptable. According to patients, the strength of PR-buddy support lies in their ability to relate to them and the use of their experience to provide confidence in attending PR.

## P34 REFLECTIONS FROM PULMONARY REHABILITATION (PR) SERVICES WHO HAVE ACHIEVED PRSAS ACCREDITATION

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10.1136/thorax-2025-BTSabstracts.216

**Background** The Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS), hosted by the Royal College of Physicians (RCP), was launched in April 2018 to improve the quality of UK pulmonary rehabilitation services. Participating services work to an accreditation pathway which involves self-assessment and quality improvement against the standards. Accredited services submit evidence annually to demonstrate maintenance to the standards and have a 5-yearly on-site assessment. The aim of this analysis was to explore the experiences of accredited services.
**Methods** A mixed method survey was sent to current accredited services (2024) to record experiences of the service accreditation journey and to explore impact, barriers and facilitators to completing the accreditation process. A descriptive analysis was undertaken with content analysis for qualitative content.
**Results** At the time of the survey 160 services were participating in PRSAS, of which 21 were accredited, 18(86%) returned the service annual survey. Most of the services (77.8% 14/18) reported measurable changes since starting and achieving accreditation, these included improvements to the exercise delivery programme (21.4% 3/14), reduced waiting times (28.6% 4/14) and improved patient outcome measures tools/data collection (42.8% 6/14). Similar numbers (77.8% 14/18) reported positive opportunities post-accreditation, including better clinical links, awareness of PR services within their organisation and/or externally (64.3% 9/14) and increased staffing (retention/recruitment/promotion, 42.8% 6/14). 88.9% (16/18) had implemented recommendations (i.e. embedding operational plans and processes [50.0% 8/16], exercise programme changes [37.5% 6/16]).

Feedback from participants highlighted a lack of standardised templates and the need for more information resources to support the process. Variation in assessor requests and as well as additional hurdles with trust requirements for data presentation were also highlighted. Several participants highlighted that the accreditation process can be 'overwhelming' and 'tricky' to coordinate alongside running clinical services. However, after completion of the process many would consider being an accreditation champion (66.7% 12/18), the

numbers participating in and completing the process successfully are increasing.

**Conclusions** These findings suggest that accreditation provides measurable impact on service Key Performance Indicators and patient outcomes.

## P35 THE IMPACT OF PULMONARY REHABILITATION SERVICES ACCREDITATION SCHEME ON KEY PERFORMANCE INDICATORS

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10.1136/thorax-2025-BTSabstracts.217

**Introduction** The Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS), hosted by the Royal College of Physicians uses a framework set out by the British Standard Institution's specification for accreditation of clinical services, to measure PR services in line with British Thoracic Society national guidance. The aim of this study is to determine the impact of accreditation on key performance indicators (KPI).

**Methods** The National Respiratory Audit Programme 2022–2023 Breathing Well report data was used. This provides service level data alongside KPI, including time to receipt of PR, completion of a discharge assessment, written discharge plan, and performing a practice 6 minute or Incremental Shuttle Walking Test (6MWT/ISWT). Services were grouped by accreditation status as of January 2025. Data was analysed using a one-way ANOVA. For accredited services, comparisons of KPI from the 2019 PR clinical audit report and 2022–2023 Breathing Well report were made using a paired t-test.

**Results** 176 services were included in the analysis (24(13%) accredited, 18(10%) undergoing assessment, 34(18%) not registered and 100(54%) registered only). There were statistically significant differences in number of patients enrolled per service in favour of those accredited (mean[SD] 143[110] compared to those not registered (71[83]) and registered only services (85[70]), $p<0.01$. The mean[SD] days from assessment to commencing PR in stable COPD was 108[82] accredited services, 126[62] undergoing assessment, 130[109] registered only and 164[118] not registered, however this was not statistically significant. There was no statistical difference for other KPI (table 1).

There was a statistically significant improvement in percentage of people enrolled from the 2019 to 2022/2023 audit in accredited services with a mean[SD] improvement of 16[31]%, $p=0.03$. Percentage of patients completing a practice 6MWT changed from 60[41] to 86[12] (n=4) and 87[26] to 80[32] in the ISWT (n=12), though this was not statistically significant.

**Conclusion** Accredited services enrol a larger number of patients into PR. There is signal in improved waiting times, written discharge plans and practice 6MWT in accredited services however this did not reach statistical significance.

**Abstract P35 Table 1** The mean[SD] percentage of patients achieving the key performance indicators. N= number of services

| % patients | Accredited n=24 | Undergoing assessment n=18 | Registered n=100 | Not registered n=34 |
|---|---|---|---|---|
| Enrolled within 90 days | 27[24] | 24[23] | 26[27] | 30[31] |
| Completing a discharge assessment | 61[22] | 63[23] | 61[21] | 65[24] |
| Receiving a written discharge plan | 91[20] | 86[24] | 81[30] | 78[36] |
| Performing a practice 6 Minute Walk Test | 63[40] (n=10) | 19[34] (n=7) | 41[38] (n=57) | 56[42] (n=16) |
| Performing a practice Incremental Shuttle Walking Test | 77[34] (n=23) | 75[34] (n=10) | 66[38] (n=59) | 83[32] (n=15) |

## P36 IMPROVING THE UPTAKE AND COMPLETION OF PULMONARY REHABILITATION (PR): DEVELOPMENT AND EVALUATION OF A TRAIN-THE-TRAINER PROGRAMME TO ESTABLISH A LAY HEALTH WORKER SERVICE

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10.1136/thorax-2025-BTSabstracts.218

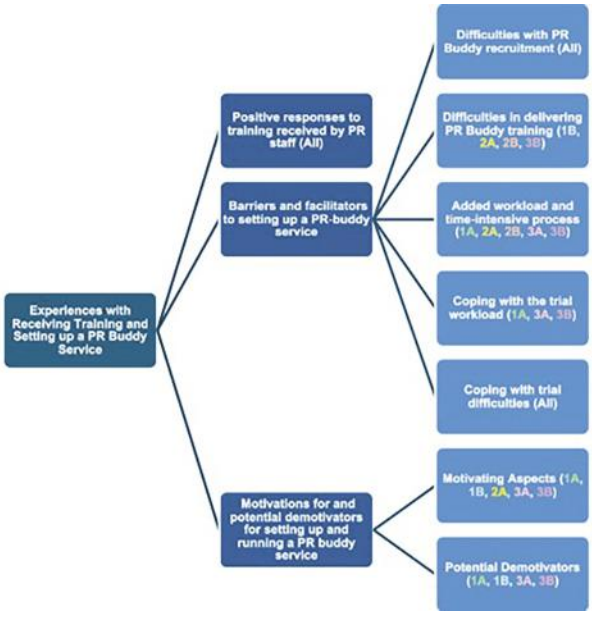
**Background** The train-the-trainer model is cost-effective and sustainable within healthcare settings but has limited evidence in pulmonary rehabilitation (PR). PR is the most effective treatment for chronic obstructive pulmonary disease (COPD), but its impact is limited by low uptake and completion.

**Objective** To design, evaluate, and refine a train-the-trainer programme for PR-staff on how to establish and manage a lay health worker (PR-buddy) service. This service aims to support the uptake and completion of PR by newly referred patients with COPD.

**Methods** A multidisciplinary team developed the training for PR-staff, to prepare them to recruit and train volunteer PR-buddies and manage a PR-buddy service. People with COPD who had successfully completed PR were invited to volunteer as PR-buddies. Semi-structured interviews with PR-staff from three pilot sites were conducted online to understand their views and experiences of receiving training and setting up the service. Interviews were completed by a team member (RD) who was not involved in training development or delivery. Reflexive thematic analysis of verbatim transcripts was conducted. Results were used to refine the training.

**Results** A 2.5-day training for PR-staff was developed, with self-directed, remote live and in-person components. It included recruitment and training of PR-buddies in communication skills, role boundaries, confidentiality, and selected behaviour change techniques. Training materials were slide decks, videos, manuals for staff and manuals for delivering training to PR-buddies. Twelve PR-staff from three trial PR services completed the initial training, and six of them participated in the interviews. Training was perceived positively by PR-staff. They found the training materials easy to use and helpful in guiding the PR-buddy training (figure 1). PR-staff reported workload challenges in the recruiting and training of PR-buddies but usually found ways to mitigate the impact of those pressures. Findings were used to modify the training given to clinicians: providing a link person from the research team for support and expanding geographical location options for in person PR-staff training.

**Conclusions** Using the train-the-trainer model in implementing a lay health worker intervention for improving uptake and completion of PR is acceptable and should be deliverable at scale.




**Abstract P36 Figure 1**

## P37 HYPERTONIC SALINE IN CHRONIC LUNG DISEASE: A LOCAL EXPERIENCE TO SUGGEST USEFULNESS?

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10.1136/thorax-2025-BTSabstracts.219

**Background** 7% Hypertonic saline (HTS) is increasingly being used as a mucolytic therapy in patients with chronic respiratory conditions, particularly those with mucus retention and impaired airway clearance. Despite its widespread use, data on the effectiveness of HTS and its potential impact on healthcare utilisation remain sparse. Understanding its role in symptom relief and hospital admission rates is critical in optimising treatment.

**Aim** The primary outcome was to observe an improvement in symptoms. The secondary outcome looked at reduction in hospital admissions comparing 12 months before HTS initiation with 12 months post-initiation.

**Method** This was a retrospective cohort study from March 2023 to February 2025. We identified patients initiated on HTS, through a logbook maintained by specialist respiratory nurses. Improvement in symptoms meant improved cough, ease of mucus expectoration and change in mucus colour, which was reported in clinic letters. We also collected data of their highest Blood eosinophil count (BEC) during this period. We used MS Excel and Vassar stats (*vassarstats.net)* for calculations.

**Results** A total of 36 patients were initiated on HTS, of which 42% (n=15) had Bronchiectasis, 8% (n=3) had COPD, 22% had Asthma (n=8), 11% had Asthma-COPD overlap (4) and 17% were labelled as others (n=6). Mean Age was 65 years (SD 10) with 56% females. 67% had BEC >0.3 $x10^9$/L.

72% (n=26) reported symptomatic improvement of which notably 42% (n=11) had Bronchiectasis, 27% had Asthma (n=7) and 15% (n= 4) had Asthma-COPD. 25% (n=9) noted no benefit and 1 patient's data was not found. Subjects showing symptom improvement had a higher mean BEC ($0.45x10^9$/L v $0.27x10^9$/L, p=0.01) when compared with those who did not show any improvement.

Our secondary outcome measure showed no effect on hospital admissions following initiation of HTS. However, 4 of 6 patients who were hospitalised over the next 12 months reported a symptomatic improvement.

**Conclusion**

1. Majority of patients with Bronchiectasis, Asthma and Asthma-COPD overlap derive symptomatic benefit from HTS treatment.

2. Improvement in patients with BEC>0.3 was statistically significant, suggesting higher degree of mucus impaction in this group.
3. HTS probably does not affect hospital admissions; but requires further validation.

## P38 PATIENT, FAMILY MEMBER, AND CLINICIAN EXPERIENCES OF PHYSIOTHERAPY AIRWAY CLEARANCE TECHNIQUES DURING HOSPITAL ADMISSION: A SCOPING REVIEW

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10.1136/thorax-2025-BTSabstracts.220

**Introduction** Effective secretion management is crucial for hospitalised patients with retained secretions due to critical illness, exacerbation of chronic respiratory diseases, or infection. Physiotherapy airway clearance techniques (ACT) facilitate secretion clearance, are safe and feasible in the hospital setting, and advocated in clinical guidelines. However, limited knowledge exists regarding the experiences of clinicians who deliver, patients who receive, and family members who are involved in ACT.

**Aim** To explore patients', family members', and clinicians' experiences with ACT in the hospital setting.

**Methods** The review protocol was registered a priori on Open Science Framework Registries https://doi.org/10.17605/OSF.IO/P4XJM. Following JBI recommendations and PRISMA-ScR Checklist, searches were conducted in CINAHL, EMBASE, MEDLINE, PsycINFO and ProQuest Dissertations & Theses Global, from inception to 27th November 2024. Qualitative studies of adult patients (≥16 years) receiving ACT, family members observing or assisting with ACT, or clinicians delivering ACT, in a hospital setting were included. Screening and data charting were performed independently and in duplicate, with findings synthesised descriptively and narratively.

**Results** Of 8980 records identified, twelve studies met the inclusion criteria. Studies were conducted across 7 countries, most frequently Australia (n=5) and ICU was the most common setting (n=8). Of the 12 studies, two explored patient experiences, one examined experiences of patients, family members, and clinicians, and nine focused on clinicians — seven of which specifically investigated physiotherapists' views. Analysis revealed five themes regarding experiences of ACT. Two themes were common across all populations: 'Knowledge, resources, and skills' and 'Relationship factors', two were shared between patients and clinicians: 'Decision-making process' and 'Perceived effects and outcomes', and one was unique to clinicians: 'Organisational factors'.

**Conclusions** Research on experiences of physiotherapy ACT in hospitals is limited, with a focus on physiotherapists in intensive care unit settings, overlooking the experiences of patients, families, and the wider multidisciplinary team. Future research should adopt a holistic, stakeholder-informed approach to fully understand the use, impact, and burden of ACT in clinical practice, and contribute meaningfully to clinical guidelines and shape future trials.

## P39 SURVEY OF NORTH CUMBRIA GP PRACTICES MANAGEMENT OF BRONCHIECTASIS PATIENTS WITH HYPERSECRETION AND PERCEIVED BARRIERS TO REFERRAL FOR RESPIRATORY PHYSIOTHERAPY

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10.1136/thorax-2025-BTSabstracts.221

**Background** Bronchiectasis is chronic lung condition. Symptoms include productive cough and mucus hypersecretion.

**Abstract P39 Table 1**

| | Oral cysteine (e.g., carbocysteine or acetylcysteine) | Education (written or online guidance for ACTs) | Referral to Respiratory Physiotherapy | Referral to Bronchiectasis clinic | Prescription of an oscillating positive expiratory pressure (OPEP) device | Fluids | Nothing |
|---|---|---|---|---|---|---|---|
| **How do you manage patients with bronchiectasis who have hypersecretions?** | 22 (85%) | 15 (58%) | 7 (27%) | 9 (35%) | 2 (8%) | 1 (4%) | 1 (4%) |
| | **No available pathway to refer into** | **No service available** | **Low understanding of service** | **Long wait times** | **No incentives** | **Other** | |
| **What are the main barriers referring bronchiectasis patients to respiratory physiotherapy?** | 12 (46%) | 0 | 9 (35%) | 1 (4%) | 0 | 4 (15%)<br>"Hard to convince patients of the benefits"<br>"Patients are referred by other staff that they see" | |

Airway clearance techniques (ACTs) are non-pharmacological interventions that support removal of mucus from the lungs. Consensus guidelines recommend ACTs are taught by specialist respiratory physiotherapists. Regional services providing specialist physiotherapy for people with bronchiectasis can be difficult to access. This can result in suboptimal management within primary care networks (PCNs), exacerbating existing health inequalities.

**Aims** To explore a local PCN's:

- Management of bronchiectasis patients with mucus hypersecretion
- Barriers to referring bronchiectasis patients to specialist respiratory physiotherapy

**Methods** A multiple choice online survey was completed by staff from GP practices within a North East and North Cumbria (NENC) primary care network (PCN). All participants treated bronchiectasis patients in their role. Snowballing recruitment was used. The survey was designed by two respiratory physiotherapists in the region. Results were collated manually using Microsoft excel.

**Results** Twenty-six health-care professionals across 14 GP practices in the NENC PCN. Results are shown in table 1. Management of patients with bronchiectasis with hypersecretion included oral cysteine 22/26 (85%), education (e.g., written instructions on ACTs) 15/26 (58%), referral to specialist bronchiectasis clinic 9/26 (35%), referral to respiratory physiotherapist 7/26 (27%) and prescription of an airway clearance oscillating positive pressure (OPEP) device 2/26 (8%). Barriers for referral to respiratory physiotherapy included lack of referral pathway 12/26 (46%), lack of knowledge on role of respiratory physiotherapy 9/26 (35%), long waiting times 1/26 (4%) and other 4/26 (15%).

**Discussion** Management of bronchiectasis patients with mucus hypersecretion in primary care is heterogenous. Oral cysteines are the most common treatment for hypersecretion in this PCN. Written information on ACTs, and prescription of ACT adjuncts (OPEP) are provided in primary care by non-specialist health professionals. Lack of pathway is the most common perceived barrier to refer patients to respiratory physiotherapy.

**Conclusion** Developing established pathways for from primary care to respiratory physiotherapy may improve access for patients. Additionally, educating primary care staff on the role of respiratory physiotherapy and how to appropriately prescribe ACTs (e.g., OPEP devices) in primary care, may support more equitable access to this essential treatment for bronchiectasis patients.

## P40 THE USE AND PERCEPTIONS OF BEHAVIOUR CHANGE INTERVENTIONS AMONG PEOPLE WITH CHRONIC RESPIRATORY DISEASE AND THOSE WHO DELIVER PHYSICAL ACTIVITY PROGRAMMES IN THE REPUBLIC OF IRELAND

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**Background** People living with chronic respiratory disease are less physically active than others, leading to poorer health outcomes.[1] While the concept of physical activity behaviour is complex, behaviour change interventions may support people with chronic respiratory disease to be physically active. The most effective interventions for long-term physical activity behaviour change in those with chronic respiratory disease remains unclear. The aim of this study was to explore the use and perceptions of behaviour change interventions among people with chronic respiratory disease and those who deliver physical activity programmes in The Republic of Ireland.

**Methods** Two anonymous online and paper-copy cross-sectional surveys were piloted and distributed via social media and relevant gate-keepers between November 2023 - April 2024.[2] Findings were summarised using descriptive statistics, including frequencies, percentages, means and medians. Relationships between variables were investigated using Chi-squared ($p=0.05$). Interventions were mapped to the COM-B model and the Theoretical Domains Framework.

**Results** The response rate to the provider survey was N=107 (107/150, 71%), and N=112 people responded to the survey of people with chronic obstructive pulmonary disease (COPD). Encouragement was perceived by providers of physical activity programmes to be the most effective intervention to influence physical activity (81/84, 96%), with education (80/84, 95%), individualized goal setting (79/84, 94%), and social support (78/84, 93%) also reported. People with COPD identified social support (76/105, 72%), which mapped to the most theoretical domains, and one-to-one interaction with a trainer (73/105, 70%) to be the most effective interventions influencing their physical activity behaviour. Using the COM-B model, motivation was identified as a common mechanism of action in influencing behaviour.

**Conclusions** Interventions for physical activity including motivational components are frequently perceived as effective influencers of physical activity behaviour by both people living with and those working with people with chronic respiratory disease in the Republic of Ireland.

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## P41 THE NUMBER AND LOCATION OF ATELECTATIC CHANGES ON CHEST CT ARE ASSOCIATED WITH INCREASED MORTALITY IN PATIENTS WITH COPD USING LONG-TERM NIV

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**Background** Long-term non-invasive ventilation (LT-NIV) may reduce the number of exacerbations and prolong survival in patients with COPD. Atelectasis is frequently reported on chest CT scan in these patients; however, the prognostic impact of this finding is not fully understood. The aim of this project to investigate the prevalence on atelectasis in patients with COPD on LT-NIV and its clinical impact.

**Methods** 347 patients with COPD participating in our prospective cohort who had a chest CT were studied.

**Results** 239 patients (69%) had atelectasis reported on their chest CT scan. Atelectasis was not associated with the number of exacerbations before or after LT-NIV setup (all $p>0.05$). In

contrast, the 5-year survival for patients with atelectasis was significantly lower compared to those who did not have atelectasis (50% vs. 65%, p<0.05). The overall mortality post-setup significantly related to the presence of left upper lobe atelectasis (n=98, p=0.03) and right middle lobe atelectasis (n=61, p=0.01) as well as the number of lobes affected by atelectasis (p=0.01).

**Conclusion** Atelectasis on chest CT scan is associated with worse clinical outcomes and may represent a treatable trait (i.e. physiotherapy for secretion management) in patients with COPD.

## P42 AN EXPERIMENTAL STUDY TO ANALYSE THE EFFECT OF RESPIRATORY PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION TECHNIQUES ALONG WITH CONVENTIONAL PHYSIOTHERAPY IN IMPROVING OXYGEN SATURATION, RESPIRATORY RATE AND HEART RATE IN COVID-19 PATIENTS ADMITTED IN ICU

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10.1136/thorax-2025-BTSabstracts.224

**Objective** Patients with COVID-19 are admitted to ICU as a result of hypoxia caused due to pneumonia. Many of these patients are ventilated using Mechanical Ventilator in order to maintain the desired oxygen Saturation. Proprioceptive neuromuscular facilitation has shown beneficial effects among patients on ventilator. Objective of this study is to compare the efficacy of Proprioceptive Neuromuscular Facilitation on respiratory rate, saturation of peripheral oxygen and heart rate.

**Methodology** 13 patients from ICU of Al Ain hospital were taken for the study who fulfilled the eligibility criteria and was systematically divided into Group A (PNF with Conventional Physiotherapy) and Group B (Conventional Physiotherapy). Patients were given the intervention according to their allocated group for 3 days and effects of these techniques on RR, SpO2 and HR parameters were observed. Data was taken at baseline and after 3 days of intervention.

**Result** Data was analyzed using Paired and Unpaired t-test. Statistically significant improvements were observed in both groups but were more pronounced in Experimental Group in terms of Respiratory Rate (t value 2.626) and Oxygen Saturation (t value 1.33) compared to Control Group. However, significant mean difference was noted in Experimental Group in terms of Respiratory Rate (8.67), Heart Rate (10.5), Oxygen Saturation (2) and Fraction of Inspired Oxygen (10.84) . Whereas in Control Group slight improvement was noted in terms of Respiratory Rate (0.43), Heart Rate (9.71), Oxygen Saturation (2.57) and Fraction of Inspired Oxygen (13.29).

**Conclusion** Study concluded that significant improvement was noticed in terms of Respiratory Rate and Oxygen Saturation in Experimental Group when compared to Control Group. However, improvement was noted in other parameters like Heart Rate and Fraction of Inspired Oxygen which was not significant statistically.

## P43 IMPACT OF INTEGRATED PULMONARY AND CARDIAC REHABILITATION: A HOLISTIC APPROACH TO PATIENT CARE IN A 10-WEEK PILOT STUDY

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10.1136/thorax-2025-BTSabstracts.225

**Background** Chronic Obstructive Pulmonary Disease (COPD) and Heart Failure (HF) share symptoms such as breathlessness and reduced exercise tolerance.

Patients with both conditions often attend separate rehabilitation services, reducing opportunities of enhanced holistic care and increasing the potential for conflicting information.

The NHS Long Term Plan promotes integrated, patient-centred models for complex multimorbidity, yet combined pulmonary and cardiac rehabilitation (PR/CR) remains underexplored.

**Aim** To evaluate the feasibility and impact of a 10-week integrated PR/CR programme, focusing on holistic outcomes, multidisciplinary collaboration, and patient involvement.

**Methods** Eight patients with confirmed dual diagnoses of COPD and HF were identified through referrals and jointly assessed.

The programme included co-developed exercise and education sessions. Patients influenced service design through questionnaires, focus groups, and weekly feedback' leading to iterative improvements.

Outcome measures included CRDQ, LINQ, physiological data, and clinical interventions. Baseline and discharge assessments were compared against national benchmarks for standalone PR and CR.

**Results** Integrated care enabled timely, holistic interventions. Cardiac patients received respiratory reviews, inhaler optimisation, COPD care planning, and bone density assessments. Respiratory patients benefited from cardiac medication titration, GTN prescribing, echocardiograms, and secondary prevention input.

CRDQ outcomes exceeded national PR averages (2018), with emotional and mastery scores more than doubling. LINQ scores fell by 4.6, indicating improved self-management.

Joint education, supported by the Recap Health App, enables personalised online education packages with videos, improved accessibility, tracking engagement, and gathering feedback, vital given the local population's average reading age of nine.

Attendance averaged 74%, with 88% completion, both surpassing national benchmarks. Patients reported increased confidence and satisfaction, with 100% recommending the service.

The programme met national PR and CR quality standards for accreditation.

**Conclusions** Integrated PR/CR is effective, feasible, and potentially scalable. It enhances access, eliminates duplication, and supports personalised care, aligning with NHS priorities.

Results demonstrate marked improvements in confidence and mental health over standalone programmes.

This innovative rehabilitation programme fosters collaboration across specialist areas to deliver a truly holistic approach

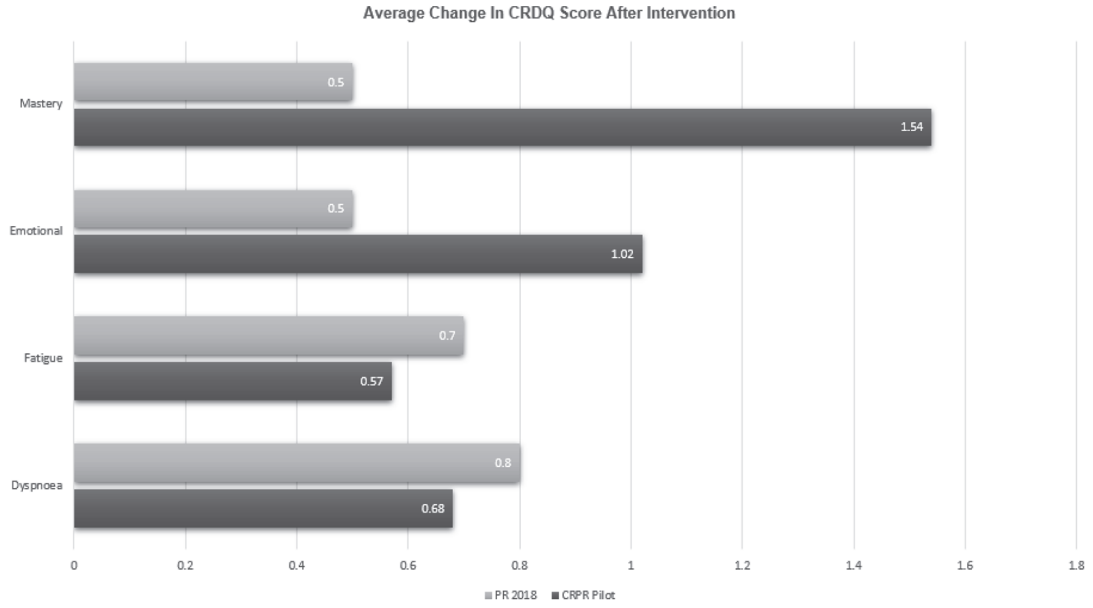



Figure 1

**Legend:**

- Light grey: PR 2018 – Average scores from the UK National Pulmonary Rehabilitation data in 2018
- Dark grey: PR/CR Pilot – Outcomes from the combined Cardiac and Pulmonary Rehabilitation pilot programme

**Note:** The CRDQ assesses the following domains: dyspnoea, fatigue, emotional function, and mastery.

- A score of 0.5 indicates a minor change
- A score of 1.0 indicates a moderate change
- A score of 1.5 indicates a substantial change
- Higher scores indicate better quality of life

**Citation: Murphy, S., 2025.** *Combined Pulmonary and Cardiac Rehabilitation Pilot Summary Report*. Derbyshire Community Health Services NHS Foundation Trust, 30 January. Figure 2

**Abstract P43 Figure 1** Comparative chart showing CRDQ outcomes for PR 2018 (light grey) versus PR/C. Pilot Programme (dark grey). The chart highlights improvements in mastery and emotional scores over the national averages

to care. Unlike anything currently offered in the UK, it responds directly to patient feedback, highlighting a clear demand for integrated support.

Further testing is needed on a larger scale.

## P44 EVALUATE THE IMPACT OF PULMONARY REHABILITATION ON STATIC KNEE EXTENSOR MUSCLE STRENGTH THROUGH A HANDHELD DYNAMOMETER IN RESPIRATORY PATIENTS – A COMMUNITY APPROACH

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10.1136/thorax-2025-BTSabstracts.226

**Introduction** Pulmonary Rehabilitation (PR) focuses on tailored physical exercise and information that helps people to better understand and manage their condition/s and symptoms, including feeling short of breath. It is a key intervention in the NHS Long Term Plan, providing PR within reach to the community.[1]

Limited literature demonstrates the impact of PR on knee extensors and the MCID of pre- and post-intervention on those muscles.

Knee extensor strength can be assessed through an isotonic or an isometric test.

It's desirable to find a time-effective and reproducible test which can be easily administered in any community rehabilitation centre.

**Aim** To evaluate the impact of PR on static knee extensor muscle strength through a hand-held dynamometer (HHD) for respiratory patients referred to Croydon Integrated Respiratory Services.

**Methods** A HHD SOP was designed and approved by the Trust Governance Committee. Participants consented. The test was performed before and after completion of a 6-week PR programme of twelve sessions. Other QoL measures obtained

**Abstract P44 Table 1**

**Demographics**

| Gender | | Ethnicity | | Conditions | |
|---|---|---|---|---|---|
| Male | 15 | White - British | 12 | COPD | |
| Female | 8 | White - Any other White background | 1 | Asthma | |
| **Age** | | Asian or Asian British - Indian | 3 | ILD | |
| <40 years old | 1 | Asian or Asian British - Pakistani | 1 | Lung Cancer | |
| 40-50 years old | 1 | Black or Black British - Any other Black background | 3 | | |
| 50-59 years old | 4 | Other Ethnic Groups - Any other ethnic group | 1 | | |
| 60-69 years old | 5 | Not stated | 2 | | |
| 70-79 years old | 7 | | | | |
| 80 years old > | 5 | | | | |

from the CRQ-SR (Chronic Respiratory Questionnaire self-reported) were compared. The programme was delivered in four sites, including one hospital and one community gymnasium, and two community halls.

**Results** A total of 258 patients were triaged, 170 attended the assessment, 158 started, thirty-five completed the programme and twenty-three had completed data.

Both legs showed improvement of +6.33 points for the right knee extensors and +8.07 for the left one.

Further analysis showed an improvement of dyspnoea of +1.14 points, Emotion +1.071, Fatigue +0.89, Mastery +1.19, CAT -5.91.

**Conclusion** HHD can easily be carried to those venues where performing an isotonic test is more difficult. It can be used safely and reliably with a vast number of patients.

The relevance of HHD in PR is supported by the current findings of this study.

Considering the small sample of participants, this lays the groundwork for future research into exploring HHD in PR services. The future of PR services can expand to the patient's home.

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## P45 DOES PULMONARY REHABILITATION (PR) LEAD TO IMPROVEMENT IN ESTIMATED 1 REPETITION (1RM) OF A LOADED SQUAT PATTERN, AND CORRELATE WITH IMPROVEMENTS IN QUADRICEPS STRENGTH MEASURED ON AN ESTIMATED 1RM KNEE EXTENSION AND INCREMENTAL SHUTTLE WALK TEST DISTANCE? A SMALL COHORT REVIEW

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10.1136/thorax-2025-BTSabstracts.227

**Background** Aerobic and resistance training are core components of a pulmonary rehabilitation (PR) programme (Man et al, 2024). A knee extension one repetition maximum (1RM) test measures the maximal strength of the quadriceps, however due to equipment available it is common in PR to measure a 1RM indirectly via an estimated 1RM (E1RM), using repetition testing at submaximal loads. Loading the lower limb muscles effectively even sub maximal levels can be a challenge without specialist gym equipment. As many PR services run in community venues, this equipment is often not available. The aim of this project was to see an E1RM squat exercise would it lead to similar improvements lower limb strength following a PR programme the commonly used knee extension E1RM does.

**Method** Patients who attended and completed a twice weekly PR programme for 8 weeks between January and May 2025 were included. Assessment as per PR guidelines were performed pre and post, including incremental shuttle walk test (ISWT) or six-minute walk test (6MET) and estimated 1RM leg extension. The Brzycki equation was then used to give an estimate 1RM (E1RM). All data was analysed to see if there were statistically significant changes and correlation between outcome measure results.

**Results** During the period 11 patients completed the PR programme table 1 outlines their demographics. There was a statistically significant change in average pre to post muscle strength score measured via the squat E1RM ($p=0.003$), with all 11 patients improving.

While was a statistically significant and strong positive correlation between pre and post E1RM squat and leg extension scores (table 1), the correlation between change in scores was weakly positive ($r=0.283$), only suggesting a possible relationship that if one improves, so does the other.

Pre to post distance in the ISWT was statistically significant ($p=0.28$), however this did not correlate with E1RM squat scores, with only a moderate correlation between the results. There was not enough data for the 6MWT.

**Abstract P45 Table 1** Patient Demographics and outcomes

| | N=11 | | |
|---|---|---|---|
| Female (%) | 7 (64%) | | |
| Age (range) | 64.9 (range 52-79) | | |
| MRC (mean±sd) | 2.72 (0.445) | | |
| COPD (%) | 5 (45%) | | |
| Interstitial lung disease (ILD) (%) | 4 (37%) | | |
| Bronchiectasis (%) | 1 (9%) | | |
| Asthma (%) | 1 (9%) | | |
| | Pre | Post | P value |
| Estimated 1RM Squat (mean±sd) | 10.64kg (1.316) | 19.27kg (2.059) | 0.003* |
| Estimated 1RM Leg extension (mean±sd) | 6.45kg (1.021) | 11.09kg (0.919) | 0.005* |
| ISWT (mean±sd) | 311.25m (31.250) | 401.43m (46.672) | 0.028* |
| 6MWT (mean±sd) | 363m (133.458) | 410m (85.049) | 0.655 |
| Correlation between estimated 1RM Squat vs knee extension | r=0.704 (p=0.016)* | r=0.759 (p=0.007)* | |
| Correlation between estimated 1RM squat vs ISWT | r=0.685 (0.090) | r=0.500 (0.253) | |
| Correlation between estimated 1RM Squat vs knee extension change in score | R=0.283 | | 0.400 |
| Correlation between estimated 1RM squat vs ISWT change in score | R=-0.613 | | 0.144 |

Key: *p value <0.05 denotes statistical significance, SD: standard deviation of the mean; COPD: Chronic obstructive pulmonary disease, MRC: medical research council breathlessness scale; ISWT: Incremental Shuttle Walk Test; 6MWT: Six-minute walk test, 1RM: One Repetition Maximum

**Conclusion** This was a small study, however the results indicate that E1RM squat test, could be a useful alternative to an E1RM leg extension in a community PR programme.

## Advances in sleep medicine

### P46 A REMOTE DIGITAL PATHWAY FOR THE MANAGEMENT OF MILD OBSTRUCTIVE SLEEP APNOEA (OSA)

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10.1136/thorax-2025-BTSabstracts.228

**Background** Obstructive sleep apnoea (OSA) affects an estimated 5% of UK adults and is increasingly prevalent. The Respiratory '*Getting it Right First Time*' report highlights the urgent need for more efficient, innovative models of care in Sleep Medicine to address rising demand with assessment of OSA often representing one third of referrals into Respiratory Medicine. Although mild asymptomatic OSA often requires no intervention, traditional referral pathways typically result in outpatient reviews for all patients. This approach places strain on services, delaying timely access to care for those with greater need and consuming significant NHS resources.

**Methods** We piloted a digital, consultant-led pathway at a large inner-city tertiary hospital for patients with mild OSA who were not excessively symptomatic. Following direct-to-test sleep studies, patients with an AHI of 5–15/hr and Epworth Sleepiness Score ≤16 were assessed for suitability. Those with self-reported English fluency and smartphone proficiency were eligible. In place of a routine appointment, patients received a diagnostic letter with detailed written advice on purchasing a semi-bespoke mandibular advancement device (MAD). SMS follow-up at 8 and 20 weeks offered options to confirm MAD purchase and request an appointment or opt-out. Patients were discharged unless engagement or clinical concern prompted further review.

**Results** Between January 2022 and April 2025, 462 patients were enrolled (baseline characteristics summarised in table 1).

**Abstract P46 Table 1** Baseline characteristics of all patients enrolled in the pathway, those who were discharged (non-responders or opt out) and those who proceeded to outpatient review. Note that 1 patient passed away

| Baseline Characteristic | All Patients (n = 462) | Discharged (n = 278) | Outpatient Review (n = 183) |
|---|---|---|---|
| Gender | | | |
| Male | 294 (63.6%) | 180 (64.7%) | 113 (61.7%) |
| Female | 168 (36.4%) | 98 (35.3%) | 70 (38.3%) |
| Age (years ± SD) | 49.6 ± 13.7 | 49.5 ± 14.0 | 49.6 ± 13.3 |
| ESS (/24): Median (IQR) | 9 (5 – 13) | 8 (5 – 13) | 9 (5 – 12) |
| AHI (events/hour): Median (IQR) | 8.00 (5.75 – 10.95) | 8.00 (5.7 – 11.0) | 8 (5.9 – 10.8) |

Of these, 221 patients (47.8%) demonstrated engagement via text response. 183 (39.6%) patients proceeded to clinic review (164 positive responders and 19 for other reasons) and 278 (60.2%) were discharged without outpatient review, either due to non-engagement (n=221, 47.8%) or opting-out (n=57, 12.3%). Only 11 patients (2.4%) were re-referred, mostly from the non-response group, suggesting a low risk of missed clinically significant disease. Avoiding 278 appointments generated an estimated cost saving of over £50,000 based on standard NHS tariffs, while reducing outpatient burden and freeing clinician time for more complex cases.

**Conclusion** This digital pathway safely triaged low-risk mild OSA patients, significantly reducing unnecessary outpatient activity. With high discharge rates, low re-referral and substantial resource savings, it represents a scalable and sustainable model for modern Sleep Medicine services.

## P47 COMPARING CPAP DELIVERY METHODS: FACE TO FACE VERSUS LOCKER COLLECTION

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10.1136/thorax-2025-BTSabstracts.229

**Introduction** CPAP treatment remains the primary treatment option for OSA.[1] Large group CPAP set up protocols have shown positive impact on waiting lists.[2] The sleep service at the Royal Berkshire NHS Foundation Trust have implemented the use of locker collection as an alternative to group or face to face (F2F) CPAP initiation.

**Method** 200 patients attended for locker CPAP collection. This involved pre appointment mask triage, assignment of individual locker code via text message, patient information leaflets and link to machine set up and usage video. 200 patients also attended for outpatient set up with a physiologist or physician associate in a 1 hour appointment. Both groups were followed up either by telephone or face to face. CPAP compliance was scored on $\geq$ 4hrs/night criteria. Non parametric independent difference testing was performed where relevant.

**Results** No significant differences in CPAP usage were found between groups. The groups were similar in age, BMI, ESS and ODI severity (table 1). Males were predominant in both F2F (64%) and locker groups (69%). There was no difference in usage hours for the compliant patients between groups (F2F: 6.1 [range 4.0–10.0] hrs/night vs Lockers: 5.9 [4.0–9.0] hrs/night).

**Conclusion** Locker CPAP collection is comparable in terms of CPAP compliance with traditional delivery methods.

**Abstract P47 Table 1** Comparison between face to face and locker collection groups. Data is mean and range where applicable

| | Age (Yrs) | BMI (kg/m²) | ESS | ODI (dips/hr) | Compliant (%) |
|---|---|---|---|---|---|
| Face to face | 57 | 38 | 10 | 35 | 54% |
| | (21-88) | (19-71) | (0-24) | (3-109) | |
| Locker | 52 | 37 | 11 | 29 | 53% |
| | (23-87) | (23-66) | (0-24) | (2-108) | |

Locker CPAP can provide a cost effective way to reduce time to treatment and a low contact alternative to group trials without little impact on treatment outcomes.

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## P48 ASSESSING THE SUSTAINABILITY OF THE CONTINUOUS POSITIVE AIRWAY PRESSURE TREATMENT PATHWAY FOR OBSTRUCTIVE SLEEP APNOEA PATIENTS


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**Introduction and Objectives** The healthcare industry is a significant contributor to the UK's carbon emissions, with respiratory medicine being a particularly high emission speciality. In alignment with the NHS's net zero 2045 goal, this study aimed to evaluate the relative carbon footprint of a typical Obstructive Sleep Apnoea (OSA) patient journey. Gold standard treatment for OSA remains Continuous Positive Airway Pressure (CPAP) therapy; however, this long-term treatment is energy intensive which raises questions about its sustainability. To assess this, we quantified the carbon footprint of CPAP treatment at a local sleep centre, to suggest opportunities for sustainability improvement.

**Methods** The annual CO2e for a one-year pathway from point of referral to post-CPAP initiation review was calculated, by mapping and evaluating the patient journey. Four categories were identified as contributing to the total carbon footprint: medical equipment, patient transport, and energy consumption from CPAP devices and hospital facility usage. Where individual component consumption was not directly quantifiable, published figures were used. Patient travel behaviours were taken from a trust-wide travel survey and used in conjunction with average travel distances for the sleep clinic cohort.

**Results** The total annual departmental carbon footprint was 222tCO2e, with a mean of 300 kgCO2e per patient. Medical equipment was the primary contributor at 67%, and within this, diagnostic studies contributed the highest percentage at 31%. Machine electricity usage contributed 17%. Full face masks bore a higher carbon footprint than nasal masks, and differences in travel habits also resulted in carbon emission variation. Although travel was a minor contributor in isolation, it represents scalable opportunity for improvement.

**Conclusion** Our results highlight the substantial environmental impact over a one-year journey for a patient with OSA undergoing CPAP treatment. Medical equipment, particularly diagnostic studies and CPAP consumables, device energy usage, and facility energy usage were the largest contributors. These emission intensive hotspots should be targeted for environmental improvement in sleep related treatment pathways. We encourage other sleep centres to use this data to review internal service pathway choices, to support the transition to environmentally responsible care.

**Abstract P48 Table 1** Final calculations of annual CO2e contributions of CPAP therapy

| Category Breakdown | Key Contributors | | Calculations (kgCO2e) | Frequency (number of patients) | Total Contribution (kgCO2e) | Percentage (%) | Percentage of Category (%) |
|---|---|---|---|---|---|---|---|
| Medical Equipment | Masks | Full Face | 52.45 | 370 | 19,407 | 8.74 | 66.79 |
| | | Nasal | 34.55 | 370 | 12,784 | 5.76 | |
| | Diagnostic Studies | | 92.23 | 740 | 68,250 | 30.74 | |
| | CPAP Consumables | | 64.14 | 740 | 47,464 | 21.38 | |
| | Appointment Disposables | | 0.50 | 740 | 368 | 0.17 | |
| Device Energy Usage | CPAP machine electricity carbon factor | | 51.10 | 740 | 37,814 | 17.03 | 17.03 |
| Patient Travel | Travel emission for 10 journeys* | Train | 2.05 | 237 | 486 | 0.22 | 1.51 |
| | | Bus | 4.34 | 129 | 560 | 0.25 | |
| | | Car | 15.88 | 145 | 2303 | 1.04 | |
| Hospital Facility Energy Usage | Hospital facility energy usage | | 44 | 740 | 32,560 | 14.67 | 14.67 |
| Total annual carbon emissions for 740 patients (kgCO2e) | | | | | 221,996 | | |
| Mean carbon emissions for an individual patient (kgCO2e) | | | | | 300 | | |

** The frequency of journeys per patient was determined through the OSA pathway; data for emissions was calculated by extrapolating results from a trust-wide travel survey.*

P49 **EVALUATING DRIVING RISK ASSESSMENT IN UNTREATED OBSTRUCTIVE SLEEP APNOEA SYNDROME (OSAS) PATIENTS AND IN CONTROLS BY USING THE LEEDS DRIVING RISK SCORE**

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**Introduction** Untreated OSAS is associated with increased road traffic accidents likely due to excessive sleepiness and advising patients about driving is a key issue in the management. Considerable variation is seen in the advice that a patient is likely to receive, and clinicians find this challenging. Screening questionnaires evaluates general sleepiness and there is a need to develop a driving specific sleepiness score which may aid in better risk assessment. We have evaluated this further in this prospective pilot study.

**Methods** Patients with an ESS of >10 and being considered for a CPAP trial either due to excessive symptoms and/or severity of OSAS following pre clinic testing, who drove regularly were recruited following an assessment in the clinic. Healthy volunteers with no symptoms of OSAS and with an ESS of <10 were recruited as controls. All subjects completed a questionnaire about driving that evaluated sleepiness specifically while driving termed the Leeds Driving Risk Score. Subjects were asked to rate on a scale ranging from 'never' (score 0), 'slight' (score 1), 'moderate' (score 2) to 'high' (score 3) their chance of dozing off or falling asleep while driving at different times of the day and on journeys of different durations (maximum possible score 30).

**Results (table 1)** OSAS patients (n=150) and controls (n=121) matched for age and driving experience were recruited. ESS and BMI were significantly worse in OSAS patients compared to controls. There was a significant difference in the Leeds Driving Risk Score between OSAS patients and controls (mean score 7±6 v/s 2±1; P= < 0.0001). No control had a total score of > 7 and 34% (n=51) of OSAS patients had a score of > 7. OSAS patients with a score of > 7 admitted to recurrent nodding in the last 12 months.

**Conclusion** Preliminary data suggests that a driving specific sleepiness score may aid in better risk assessment and may be a complementary, and less threatening way, to ask about sleepiness likely to impair safe driving. Further studies are needed to validate these findings and also in mild OSA cohorts.

**Abstract P49 Table 1**

| **Baseline Demographics** | **Controls (n=121)** | **OSAS Patients (n=150)** | **P-Value (95% CI)** |
|---|---|---|---|
| Age | 54+/-15 | 52+/- 11 | P=0.19 (-1.08 to 5.29) |
| Gender | Females=31% (n=37)<br>Males= 69% (n=84) | Females=14% (n=21)<br>Males= 86% (n=129) | |
| BMI kg·m$^{-2}$ | 28 +/-6 | 36+/-7 | P= < 0.0001 (-9.97 to -6.69) |
| Severity of OSAS | NA | Mild OSAS= 21% (n=31)<br>Moderate OSAS= 27% n=41)<br>Severe OSAS=52% (78) | |
| Driving Licence (Years) | 32 years | 31 years | 0.89 ( -3.14 to 3.59) |
| HGV Drivers | 6% (n=5) | 10% (n=15) | P= NS |
| ESS | 4 +/-2 | 14+/-5 | P= < 0.0001 (-10.88 to 8.96) |
| Annual Mileage<br>< 5000<br>5000- 10000<br>10000-15000<br>15000-20000<br>20000-50000<br>>50000 | <br>25% (n=30)<br>30% (n=34)<br>23% (n=28)<br>9% (n=11)<br>8% (n=10)<br>5% (n=7) | <br>12% (n=18)<br>40% (n=60)<br>20% (n=30)<br>12% (n=18)<br>13% (n=19)<br>4% (n=6) | |
| | | | |
| **Leeds Score** | | | |
| Score 7 or less | 100% (n=121) | 66% (n=99) | P= < 0.0001 |
| Score > 7 | 0 (n=0) | 34% (n=51) | |
| | | | |
| **OSAS Patients** | **Leeds Score < 7 ( n=99)** | **Leeds Score > 7 ( n=51)** | |
| ESS < 10 | 27% (n=27) | 22% (n=11) | |
| ESS > 10 | 73% (n=72) | 78% (n=40) | P= 0.55 |
| Nodding (> 2 times) | 1% (n=1) | 25% (n=13) | < 0.0001 |
| RTA | 7% (n=7) | 8% (n=4) | 0.73 |

## P50 A PILOT DIGITAL PATHWAY FOR REMOTE DETECTION OF SLEEP DISTURBANCE IN CARDIOMETABOLIC DISEASE

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**Introduction** Sleep disturbances/diagnoses are acknowledged complications across diverse chronic diseases but are rarely systematically assessed or treated. We set out to develop a low-impact, simple decentralised digital pathway to define the proportion of patients with multiple long-term cardiometabolic conditions affected by sleep disturbance and pilot a remote diagnostic/monitoring assessment pathway in a subset of patients with pulmonary hypertension (PH).

**Methods** Patients in an ongoing open primary-care registry with a coded diagnosis of any cardiometabolic disease (>18 yrs) were prospectively consented through the uMed AccessCMD registry and undertook a set of digital patient-related outcome measures (ePROMs) that included the Jenkins Sleep Evaluation Questionnaire (JSEQ); a short, simple 4 domain questionnaire. A cohort of patients with pulmonary hypertension in the UK National Cohort Study (UKNCS) undertook ePROMs and additional paper validation and cross-comparison with longer-form questionnaires- Insomnia Severity Index (ISI), Epworth Sleepiness Scale (ESS) and wearable assessment (GarminVenu2). Patients with a previously-defined JSEQ threshold score of >5 were put through a remote diagnostic pathway to investigate common causes of sleep disturbance with guideline-approved treatment options including insomnia, sleep apnoea (obstructive and central), restless legs syndrome (RLS) and menopause.

**Results** Provisional results from the AccessCMD registry demonstrate that 2057 (74%, n= 2758) had a JSEQ of >5, 61% in the UKNCS (n=44). JSEQ scores highly correlated with ISS (r=0.84 p=2.89e-11) and moderately with ESS (r=0.44 p=4.38e-03). Wearable data demonstrated the highest correlation between JSEQ and REM sleep variation (r=0.6 p=0.00151). To date, 24 participants (54.5%) have been diagnosed with a sleep disturbance; RLS (n=9, 20.45%), insomnia (n=9, 20.5%), menopause (n=4, 9.1%), OSA (n=4, 9.1%) and CSA (n=1, 2.3%).

**Conclusion** A high proportion of patients with cardiometabolic disease report sleep disturbance and a remote diagnostic pathway in a pilot cohort of patients with PH identified potentially treatable causes in >50% of participants.

## P51 MILD BUT NOT MINOR: INVESTIGATING THE CLINICAL BURDEN OF MAA USE IN MILD OBSTRUCTIVE SLEEP APNOEA

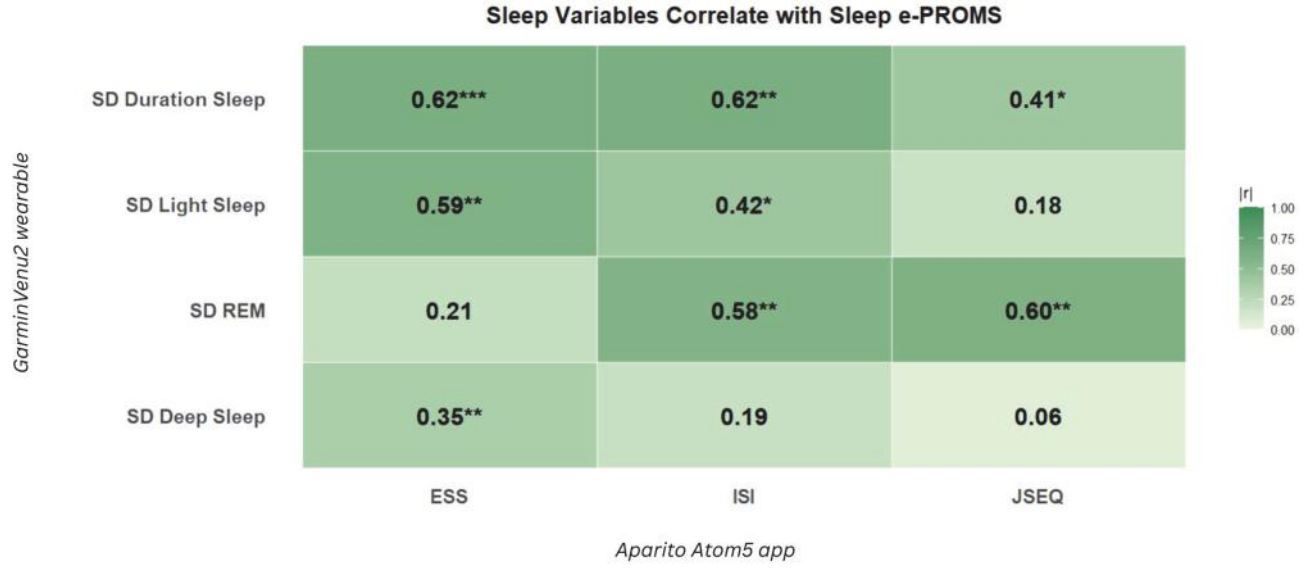
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10.1136/thorax-2025-BTSabstracts.233

**Introduction** Mild Obstructive Sleep Apnoea (OSA) is often assumed a lower symptom burden and healthcare utilisation than moderate and severe, which may overlook true patient experiences. Recent findings support a symptom-based approach to treatment over AHI-based alone.[1] This study compares the symptom and treatment burden of mild OSA with moderate and severe OSA.

**Methods** A retrospective analysis at a central London sleep centre for all patients newly diagnosed with OSA over a 5-month period. Inclusion criteria consisted of a confirmed OSA diagnosis and completion of a clinical sleep symptom questionnaire. Symptom profile, treatment modality (continuous positive airway pressure [CPAP] or mandibular advancement appliance [MAA]) and healthcare utilisation data were analysed and compared across severity groups.

**Results** 307 patients were identified. Mean age was 51 years (SD 14); median BMI was 33.5 (IQR 10.4); 68% were male (n=210). The most reported symptoms were feeling 'tired, fatigued or sleepy' during daytime (88%), snoring loudly (83%) and unrefreshed morning wakening (82%) (table 1). 42% had mild OSA (n=130), 32% moderate (n=98) and 26% severe (n=79). Symptom prevalence did not differ across severity groups (p=0.155–0.810). Excessive daytime




**Abstract P50 Figure 1**

**Abstract P51 Table 1** Sleep Symptom Distribution Across Severity Groups

| | Mild, n=130 | Moderate, n=98 | Severe, n=79 | Total, n=307 | p-value |
|---|---|---|---|---|---|
| **Do you snore loudly?** | 83% | 74% | 89% | 83% | 0.155 |
| **Do you often feel tired, fatigued or sleepy during daytime?** | 89% | 86% | 85% | 88% | 0.771 |
| **Do you wake up in the morning feeling unrefreshed?** | 85% | 76% | 81% | 82% | 0.525 |
| **Do you wake up during the night choking, gasping or with a snort?** | 66% | 52% | 65% | 62% | 0.810 |

somnolence (Epworth Sleepiness Score $\geq$11) was present in 52% of mild, 39% of moderate and 57% of severe OSA ($p>0.05$). Treatment provision varied: 88% of the severe group received CPAP, 69% of moderate and 31% of mild; 29% of the mild group received MAA versus 6% of moderate and 2% of severe. Healthcare visit burden in the first year was significantly higher in mild OSA patients than other severities (3.6 mean appointments vs 2.8 and 2.9, $p<0.001$), predominantly due to MAA treatment, shown in further analysis.

**Conclusion** Our study showed that patients with mild OSA experience a symptom burden comparable to those with moderate or severe disease. Healthcare visit burden was higher in this cohort. Our findings highlight the limitations of AHI-based definitions in estimating clinical burden and the often under-appreciated impact for patients with mild OSA.

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## P52 PREVALENCE OF SLEEP APNOEA AMONG PATIENTS WHO REQUIRED BILEVEL POSITIVE AIRWAY PRESSURE (BIPAP) THERAPY FOR ACUTE HYPERCAPNIC RESPIRATORY FAILURE – A SINGLE-CENTRE RETROSPECTIVE STUDY

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**Introduction and Objectives** Obstructive sleep apnoea (OSA) causes significant symptom burden to patients and is associated with cardiovascular and metabolic complications if it is untreated. Current evidence highlights the diagnostic challenge of OSA due to a lack of correlation between the severity of disease and its heterogeneous symptoms and associated comorbidities. This study determines the prevalence of sleep apnoea and its overlap syndromes, and investigates its association with hypertension, type 2 diabetes mellitus and obesity in patients who developed acute hypercapnic respiratory failure.

**Methods** A retrospective review was performed on patients who required BiPAP therapy for acute hypercapnic respiratory failure over a 12-month period. Patients were identified via the data analysis team and data was accessed using hospital electronic records. Eligibility criteria were summarised in table 1. Primary outcome included oxygen desaturation index (ODI) as determined by overnight pulse oximetry. Secondary outcomes included diagnoses of chronic obstructive pulmonary disease (COPD), obesity hypoventilation syndrome (OHS), hypertension, type 2 diabetes mellitus and obesity.

**Results** Out of 149 patients who had BiPAP therapy for acute hypercapnic respiratory failure, 53 patients were included in the study. 16 patients representing 30.19% of patients studied underwent screening with overnight pulse oximetry. 13 patients representing 81.25% of patients screened were found to have evidence of sleep apnoea with a mean ODI of 30.92 $\pm$ 21.31 events per hour of sleep. 10 patients representing 76.92% of patients diagnosed with sleep apnoea had overlap syndromes with COPD or OHS. There was no statistically significant correlation between the severity of sleep apnoea and the prevalence of hypertension, type 2 diabetes mellitus and obesity, $p = 0.70$.

**Conclusions** This study highlights the importance of screening patients who develop acute hypercapnic respiratory failure for evidence of sleep apnoea using the overnight pulse oximetry test. The STOP-BANG questionnaire should be used in this patient cohort to select high risk patients for the screening test. Although polysomnography remains the gold standard diagnostic test for OSA, continuous positive airway pressure

**Abstract P52 Table 1** Patient eligibility criteria presented in n (%)

| N = 149 | |
|---|---|
| **Inclusion criterion** | |
| Patients who required BiPAP therapy for acute hypercapnic respiratory failure from August 2023 to August 2024 | 53 (35.57%) |
| **Exclusion criteria** | |
| Patients who died during or shortly after hospital admission | 38 (25.50%) |
| Patients who had BiPAP therapy for the primary diagnosis of acute heart failure | 20 (13.42%) |
| Patients who had domiciliary non-invasive ventilation (NIV) therapy at the time of hospital admission | 18 (12.08%) |
| Patients who did not attend NIV clinic follow up or overnight pulse oximetry test | 6 (4.03%) |
| Patients who were started on domiciliary NIV during or shortly after hospital admission | 5 (3.36%) |
| Patients who did not live locally | 4 (2.68%) |
| Patients who received end of life care or palliative care | 3 (2.01%) |
| Patients who declined domiciliary NIV therapy | 2 (1.34%) |

(CPAP) therapy is the mainstay of treatment for central and obstructive sleep apnoea and it has been shown to improve mortality in patients with OSA.

## P53 OBSTRUCTIVE SLEEP APNEA (OSA) IN SARCOIDOSIS — A NEGLECTED ASPECT OF THE DISEASE

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**Introduction** The prevalence of OSA is 2.5 times higher in patients with sarcoidosis as compared to matched controls and can have significant impact on fatigue following the treatment.[1 2] Despite increased prevalence screening for OSA in sarcoidosis is not very robust likely due to overlapping symptoms. Through this quality improvement project (QIP), we aimed to enhance OSA screening in patients with sarcoidosis with potential impact of treatment on clinical outcome.

**Methods** As part of this QIP, we conducted retrospective review of clinical data following a baseline audit. Interventions included education of ILD team with the results of baseline audit, latest data about prevalence of OSA in sarcoidosis and to actively screen sarcoidosis patients for OSA with STOP-BANG and Epworth Sleepiness Scale (ESS), at ILD clinic. These patients were then referred to sleep clinic and underwent limited sleep study (LSS) and treatment was offered as per NICE guidelines.

**Results** Baseline audit included 34 patients, 29 (85%) of these patients had BMI of >25 kg/m2. Only 2 of these were screened for OSA and subsequently diagnosed with severe OSA requiring treatment with CPAP. Since the first intervention in January 2025, 23 patients have been screened for fatigue and OSA using STOP-BANG score. Based on

screening, 18 (78%) patients were referred for LSS, 10 (43%) patients have so far been diagnosed with OSA. Seven of these patients have moderate to severe OSA (AHI >15). Three patients have started CPAP therapy and four have been advised to make lifestyle changes.

**Conclusion** OSA is under-diagnosed in sarcoidosis. Active screening of sarcoidosis patients for OSA in ILD clinic is feasible utilizing ESS and STOP-BANG scores and results in earlier diagnosis of OSA. It may have potential impact on improving quality of life and fatigue by treatment. This strategy should be considered in a larger multicenter cohort to confirm the co-existence of OSA in sarcoidosis with implications of prompt management.

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## P54 MORE THAN A NEAR MISS: THE IMPORTANCE OF MULTI-QUESTION SCREENING FOR DRIVING IMPAIRMENT IN OBSTRUCTIVE SLEEP APNOEA

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10.1136/thorax-2025-BTSabstracts.236

**Introduction** Obstructive Sleep Apnoea (OSA) is a recognised contributor to driving impairment, yet its real-world impact remains underappreciated in clinical practice. Traditional severity measures like the Apnoea-Hypopnoea Index (AHI) and subjective sleepiness scores often fail to capture functional risk. This study sought to determine the prevalence of sleep-related driving impairment in OSA using multiple targeted questions and exploring associations with demographic and

**Abstract P54 Table 1** Patient characteristics

| | | N | % |
|---|---|---|---|
| **Sex** | Male | 130 | 80 |
| | Female | 32 | 20 |
| | | | |
| **Ethnicity** | White | 57 | 35 |
| | Black | 22 | 14 |
| | Asian | 6 | 4 |
| | Mixed | 2 | 1 |
| | Other | 3 | 2 |
| | N/A | 70 | 43 |
| **Deprivation Decile** | 1 | 3 | 2 |
| | 2 | 19 | 16 |
| | 3 | 33 | 20 |
| | 4 | 23 | 14 |
| | 5 | 22 | 14 |
| | 6 | 21 | 13 |
| | 7 | 19 | 13 |
| | 8 | 8 | 5 |
| | 9 | 7 | 4 |
| | 10 | 5 | 3 |
| | | | |
| **OSA severity** | Mild | 72 | 44 |
| | Moderate | 52 | 32 |
| | Severe | 38 | 23 |
| **Epworth Sleepiness Score (mean +/- SD)** | Driving Not Impacted: 8.67 ± 0.45<br>Driving Impacted: 13.64 ± 0.67 | | |

clinical factors, aiming to build a comprehensive understanding of how OSA affects driving.

**Methods** Retrospective analysis from patients with OSA who attended sleep clinic between May and September 2023. Inclusion criteria consisted of a confirmed OSA diagnosis, a completed sleep clinic questionnaire and being a driver. Patients completed four questions on driving impairment (near-misses, accidents, head-nodding, and needing breaks while driving due to fatigue) as part of a standard clinical symptom questionnaire. A positive response to any of the four question items was deemed indicative of driving being affected. Deprivation deciles were assessed using the Index of Multiple Deprivation (IMD), based on patients' postcodes. Subjective daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS). The relationships between reported driving impairment and clinical variables were evaluated.

**Results** 162 patients met the study inclusion criteria with cohort characteristics shown in table 1. In total, 47 (29.0%) drivers reported at least one feature of impaired driving, and 38% of this group reported more than one feature. However, the nature of driving impairment was not consistent in the responses. 30 (18.5%) patients reported head-nodding, 26 (16.0%) needing to stop for breaks, 12 (7.4%) reported near-misses and 3 (1.9%) having experienced road traffic accidents. Notably, there was no association between driving impairment and OSA severity, but ESS was found to be significant ($p<0.001$).

**Conclusion** Driving impairment is frequently self-reported among individuals with OSA, affecting 29% in this study population but with no relationship with disease severity. However, greater ESS can be used as a predictor of driving impairment. Our findings highlight the importance of screening for driving impairment in all patients and the need for multi-question tools to identify risks more comprehensively.

## P55 PERSONALIZED MAXILLO-MANDIBULAR ADVANCEMENT IN OBSTRUCTIVE SLEEP APNOEA: A MILLIMETRE-BASED INDIVIDUALIZED TREATMENT APPROACH

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**Background** A significant proportion of patients with OSA struggle to tolerate CPAP. Maxillo-mandibular advancement (MMA) represents an effective surgical intervention. However, current practice often employs standardized advancement protocols without consideration of individual anatomical requirements. This retrospective QIP evaluates outcomes from a personalized, millimetre-specific advancement strategy that tailors surgical intervention to individual patient rather than adopting a uniform approach.

**Methods** We conducted a retrospective review of patients with moderate-to-severe OSA who underwent MMA between 2023–2025. All patients had documented CPAP intolerance or treatment failure. The surgical technique involved mandibular osteotomy with initial distraction to 7 mm along with plate and screw insertion. Beginning on postoperative day 8, patients performed gradual mandibular advancement by turning distraction screws 1 mm incrementally. Follow-up sleep studies were conducted after each advancement, and patients with persistent OSA underwent further 1–2 mm advancements until therapeutic targets were achieved. Primary outcomes included surgical success rates (defined as postoperative AHI <5 events/hour or >50% reduction from baseline) and complete cure rates (AHI <5 events/hour). Secondary outcomes assessed improvement in subjective sleepiness using Epworth Sleepiness Scale (ESS).

**Results** 11 patients (9 male, 2 female) with mean age 49 years and BMI $30.3\pm5.2$ kg/m$^2$ underwent individualized MMA. Mean pre-operative AHI was 42.8/hour (range 16–110). Surgical advancement was personalized, ranging from 7 to 16 mm, (mean 11.9 mm). Post-operative AHI decreased to $6.3\pm5.3$/hour (range 1.8–20), representing an 85% reduction. Surgical success was achieved in 10/11 patients (90.9%), with complete cure in 5/11 patients (45.5%). ESS scores improved from 14.1 $\pm5.4$ to post-operative values to $5.55.5\pm4.3$. Notably, patients receiving smaller advancements (≤10 mm) achieved 100% success rates, challenging assumptions that larger advancements necessarily yield superior outcomes.

**Conclusions** Individualized approach to maxillomandibular advancement (MMA) demonstrates that personalized advancement strategies achieve excellent outcomes while potentially reducing surgical morbidity associated with excessive advancement. The wide range of effective advancement distances (7–16 mm) underscores the inadequacy of standardized protocols and supports the use of postoperative sleep studies to objectively guide patient-specific treatment, challenging the one-size-fits-all approach. These findings also highlight the importance of collaborative approach between sleep physicians and surgeons in optimizing patient outcomes.

## P56 FATIGUE, FUNCTION AND FEAR: WHAT MATTERS MOST TO PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA?

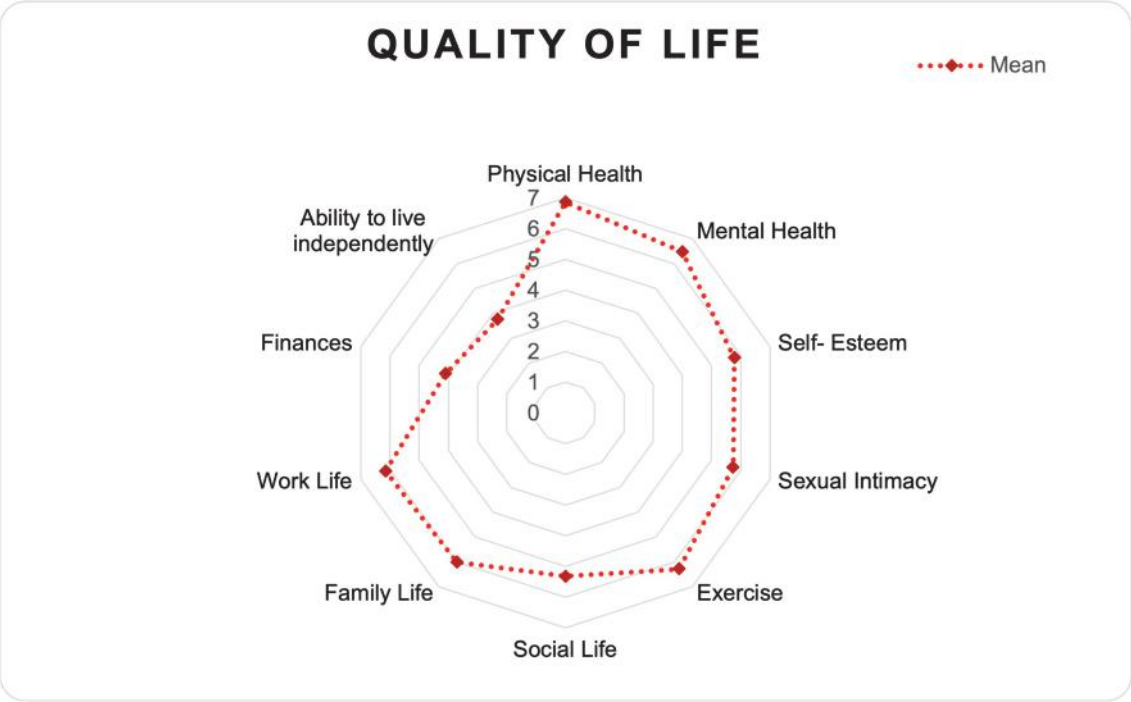
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10.1136/thorax-2025-BTSabstracts.238

**Introduction and objectives** Sleep-disordered breathing, particularly obstructive sleep apnoea (OSA), is associated with physical morbidity, psychosocial impairment and economic cost; yet patient-reported burden at diagnosis is seldom captured in routine care. We therefore surveyed adults with newly diagnosed OSA to: (i) determine the prevalence and perceived severity of core symptoms; (ii) quantify their impact across ten physical, mental and social quality-of-life domains; and (iii) establish the frequency of co-existing depressive and anxiety symptoms.

**Methods** Consecutive adult patients with newly diagnosed OSA at a single-centre sleep service were invited to complete a questionnaire prior to commencing CPAP treatment (Oct 2023-Mar 2024). Four domains were evaluated: severity of OSA symptoms; quality-of-life domain (10-point Likert scale, 10=most affected), depression (PHQ-9); and anxiety (GAD-7).

**Results** Seventy-two patients participated over a 6-month study period. They were predominantly male (58%); 49% identified as White British and 18% as Black African/Caribbean. The most prevalent symptoms of at least moderate severity were daytime low energy (81%), unrefreshing sleep (79%) and snoring (78%). The most affected life-domains were physical health ($6.9\pm2.67$), followed by mental health ($6.5\pm3.13$) and exercise ($6.3\pm3.04$). Finances ($4.1\pm3.17$) and independent




**Abstract P56 Figure 1**

living (3.8±3.32) were least affected (figure 1). Elevated depression and anxiety scores (PHQ-9 ≥ 10 and GAD-7 ≥ 10) were observed in 55% and 33% of patients, respectively. At the item level, loss of interest (75%) and low mood (66%) were reported on ≥ 'several days', and 15/72 (21%) acknowledged self-harm thoughts. Anxiety items were positive on ≥ 'several days' in 59–74% of respondents.

**Conclusions** This study shows that patients with OSA can experience marked multidimensional burden. Four in five experience moderate-to-severe fatigue or unrefreshing sleep, physical-health impact scores are highest, and exercise (mean 6.3) and family life (6.0) are similarly compromised. More than half screen positive for clinically relevant depression or anxiety. Routine assessments should incorporate validated patient-reported outcome measures for fatigue, functional limitation and mood, providing clear targets for counselling and follow-up.

## P57 ACCURACY OF STOPBANG IN IDENTIFICATION OF OSA IN DIFFICULT & SEVERE ASTHMA

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10.1136/thorax-2025-BTSabstracts.239

Obstructive sleep apnoea (OSA) is common in patients with difficult-to-control asthma and may contribute to poor disease control. The STOP-BANG questionnaire is a widely used OSA screening tool, though its validity in asthma populations remains unclear.

**Methods** We prospectively analysed 37 patients attending a difficult asthma clinic who completed STOP-BANG screening and underwent home sleep studies. Demographic data (age, sex, BMI), STOP-BANG scores (0–8), and apnoea-hypopnoea index (AHI) from sleep studies were recorded. OSA severity was defined by standard AHI thresholds: normal (<5 events/hour), mild (<15 events/hour), moderate (<30 events/hour), and severe (≥30 events/hour). We assessed the diagnostic performance of a STOP-BANG score ≥3 to detect moderate-to-severe OSA (AHI ≥15), calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).

**Results** The study cohort consisted primarily of female patients (78%), with a mean age of 51.5 years (range 27–76) and a mean body mass index (BMI) of 31.2 kg/m$^2$ (range 21.0–55.8), indicating an overweight to obese population. Using a STOP-BANG score threshold of ≥3, the tool demonstrated a sensitivity of 58.3% and a specificity of 52.0% for detecting moderate-to-severe OSA (AHI ≥15) . The positive predictive value (PPV) was 36.8%, while the negative predictive value (NPV) was 72.2%.Of the 19 patients who screened positive with STOP-BANG scoring ≥3, only 7 (37%) had moderate-to-severe OSA on diagnostic testing. In contrast, 9 (47%) had only mild OSA and 3 (16%) had no OSA, indicating a high false-positive rate. Additionally, 5 cases of moderate/severe OSA were missed by patients with STOP-BANG <3.

**Conclusion** In this female-predominant, difficult asthma cohort, STOP-BANG ≥3 showed modest sensitivity and limited specificity for identifying clinically significant OSA. While a low score may help exclude moderate-to-severe OSA, the high false-positive rate limits its standalone use in this population. These findings suggest STOP-BANG should be used cautiously in asthma cohorts and highlight the need for asthma-specific OSA screening tools.

## P58 IMPACT OF VIRTUAL CPAP SET-UP ON SLEEP SERVICE INTERACTIONS

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**Introduction** Continuous positive airway pressure (CPAP) therapy, the treatment for obstructive sleep apnoea (OSA), is conventionally set up face-to-face (F2F) but shifted to a virtual (VR) format due to the Covid-19 pandemic. This study compares the quantity and nature of patient-service interactions over a fixed period for both set-up groups to determine the impact on service provision, workflow efficiency and overall cost. The findings aim to assess the sustainability of the change and guide current practices which remain virtual within this sleep service.

**Method** In this retrospective service evaluation,100 patients were sequentially sampled for each set-up type. 195 patients met the inclusion criteria: F2F set-up (n=97) VR set-up (n=98). Data on patient-service interactions within the first year of CPAP initiation were collected from electronic patient records. Interactions were classified by total quantity, type (F2F vs VR), reason (clinical vs equipment) and professional required (specialised staff vs clinical support workers). Specialised staff includes doctors, physiotherapists, physiologists and nurses. Mann-Whitney U tests were conducted using STATA software for statistical analysis.

**Results** Table 1 outlines the recorded patient-service interactions along with their associated statistics. As per respective set-up group, there were significant differences in type of interactions with F2F patients having more in-person (57 vs 8, p-value=0.00) and VR patients more virtual (327 vs 293, p-value=0.04). No significant differences were observed between groups in their total number of interactions, professional required and reason.

**Conclusion** The transition to VR set-up methods did not increase the total number of interactions patients had with the sleep service. The service did not require more specialised staff for appointments or experience increased resource burden when providing VR set-up. Due to its equivalent impact on service provision, this is a viable alternative method for the sleep service to use without increasing workload and costs.

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## P59 USE OF MULTICHANNEL SLEEP STUDIES TO TRIAGE CPAP TREATMENT IN SYMPTOMATIC PATIENTS WITH NORMAL OVERNIGHT OXIMETRY

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10.1136/thorax-2025-BTSabstracts.241

**Introduction** Rapid assessment of overnight desaturation is key in diagnosing Obstructive Sleep Apnoea (OSA). OSA can be diagnosed with home sleep studies; using overnight oximetry with access to multichannel services if results are normal but patients are symptomatic.[1]

We sought to determine whether there were any predictive factors differentiating patients who required CPAP following completion of both overnight oximetry and multi-channel studies compared to those that were discharged.

**Method** 73 patients completed two nights of Oximetry followed by a two night Multichannel sleep study. Patients completed questionnaires to score Epworth Sleepiness Scale (ESS), measurements of age, height, weight, neck circumference and Body Mass Index (BMI) prior to initial oximetry.

**Results** 30 patients required CPAP therapy and 43 were discharged. There was no significant difference in ESS, neck circumference, age and BMI between groups. The overnight oximetry results, measured using ODI and $SpO_2$ were similar within the groups. The range of the AHI measured using Multichannel devices was similar between groups. We did find a higher average AHI in the CPAP group, this group were predominantly male, 64% in the CPAP group compared to 37% in discharged group.

**Abstract P58 Table 1** Comparison of frequencies and types of service interactions between both groups

| Service Interactions | Set-up group | | |
|---|---|---|---|
| | F2F (n=97) | Virtual (n=98) | p-value* |
| Total number | 350 | 335 | 0.47 |
| F2F | 57 (16%) | 8 (2%) | 0.00** |
| Virtual | 293 (84%) | 327 (98%) | 0.04** |
| Equipment | 67 (19%) | 45 (13%) | 0.09 |
| Clinical | 283 (81%) | 290 (87%) | 0.44 |
| Specialised staff | 235 (67%) | 224 (67%) | 0.87 |
| Clinical Support Worker | 115 (33%) | 111 (33%) | 0.83 |

*Count (Percentage)*

* *Mann-Whitney U test*

** *Significant **p**-value (<0.05)*

**Abstract P59 Table 1** Values from Overnight Oximetry (Oxim) and Multichannel (MC). Apnoea-Hypopnea Index (AHI) and Oxygen Desaturation Index (ODI)

| | No CPAP (n=43) | | CPAP (n=30) | |
|---|---|---|---|---|
| | Average | Range | Average | Range |
| **Oxim ODI** | 2.35 | 0 - 21.89 | 6.2 | 0.4 - 70.76 |
| **Oxim >3% Dips** | 8.05 | 0.31 - 30.33 | 9.52 | 1.08 - 75.56 |
| **Oxim Mean $SpO_2$** | 94.64 | 90.02 - 97.37 | 94.25 | 86.1 - 96.89 |
| **MC ODI** | 3.11 | 0.13 - 12.02 | 14.89 | 0.89 - 149.4 |
| **MC AHI** | 4.76 | 0.82 - 16.5 | 19.96 | 4.26 - 101.95 |
| **MC Mean $SpO_2$** | 95.28 | 91.95 - 97.82 | 93.5 | 70.79 - 98.02 |

**Conclusions** On reviewing both Oximetry and multichannel data, a higher average Oxim ODI and MC AHI in the CPAP group was noted in comparison to the discharged patients that were discharged with majority of patients falling into the moderate OSA category requiring CPAP. There was minimal difference in questionnaires and demographics identified between the two groups other than gender difference. Studies have shown that due to differences in sleep latency, upper airway fat distribution and difference in hormonal profile, men have a higher risk of developing OSA.[2]

Screening Oximetry remains a useful tool to identify OSA, however Multichannel studies allow more sensitive diagnosis in patients with normal oximetry if clinical suspicion of OSA is high.

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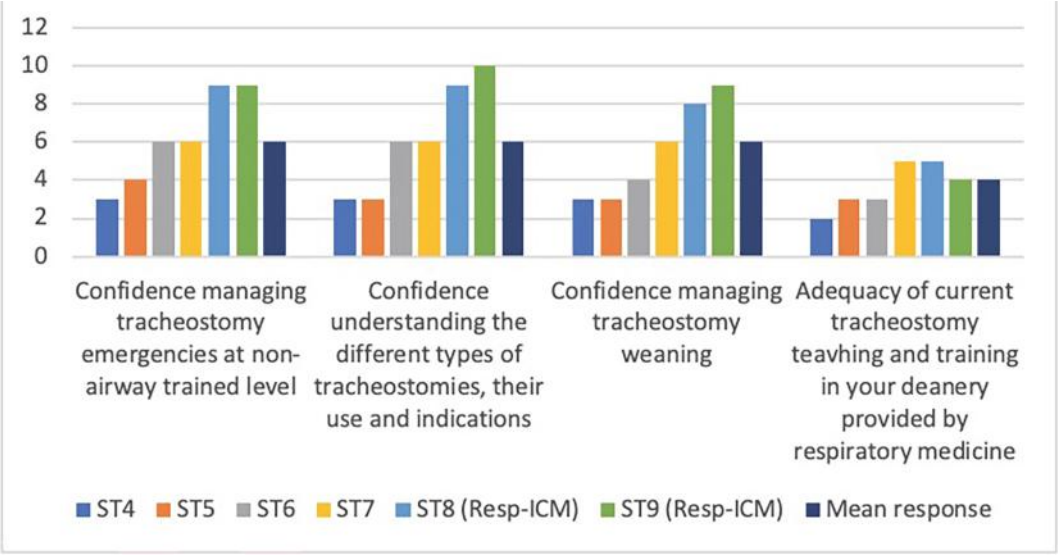

## P60 ON THE RIGHT TRACH YET? NATIONAL SURVEY OF RESPIRATORY REGISTRAR EXPERIENCE WITH TRACHEOTOMY TRAINING


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10.1136/thorax-2025-BTSabstracts.242

**Introduction** Tracheostomy care in hospitals has historically been suboptimal, contributing to increased morbidity and mortality.[1] These patients require complex, multidisciplinary care delivered by appropriately trained staff. Simulation-based training improves both knowledge and confidence in tracheostomy care. We conducted a national survey to assess training, exposure, and confidence among respiratory trainees in the UK.

**Methods** A web-based survey was designed to evaluate tracheostomy training and exposure among UK respiratory trainees.




**Abstract P60 Figure 1** Trainee Confidence Ratings

Respondents were asked about training grade, deanery, ICU rotation completion, and whether they were dual training in Intensive Care Medicine (ICM). The survey included questions on theoretical and practical training, use of simulation, confidence levels, and suggestions for improving training. It was distributed via the Dual Respiratory & ICM trainees WhatsApp group between November 2024 and February 2025.

**Results** Seventy-one trainees responded, including 12 dual ICM trainees, from 10 deaneries. Of all respondents, 32% had not received any tracheostomy training, while 41% had received both theoretical and practical instruction. Among 59 non-ICM trainees, only 31% had received both.

Confidence in tracheostomy care was associated with seniority, dual ICM training, ICU experience, and access to a tracheostomy weaning unit. Trainees with simulation experience or frequent exposure to tracheostomy patients reported higher confidence levels figure 1).

A total of 94% expressed interest in attending practical or simulation-based training. Suggested improvements included mandatory e-learning modules, annual simulation sessions, and dedicated tracheostomy teaching at regional training events.

**Conclusion** This national survey highlights a clear mismatch between the clinical expectations of respiratory trainees and their current training in tracheostomy care. Gaps in structured teaching and simulation access are common, but trainees identified practical, achievable improvements. There is strong interest in better training opportunities. It is now the responsibility of training bodies and educators to implement meaningful changes.

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# Holistically improving asthma care

## P61 BARRIER AND FACILITATORS TO PRIMARY CARE PRACTITIONER PRESCRIBING AND PATIENT ADOPTION OF ANTI-INFLAMMATORY, MAINTENANCE AND RELIEVER THERAPY (MART) FOR ADULTS WITH ASTHMA

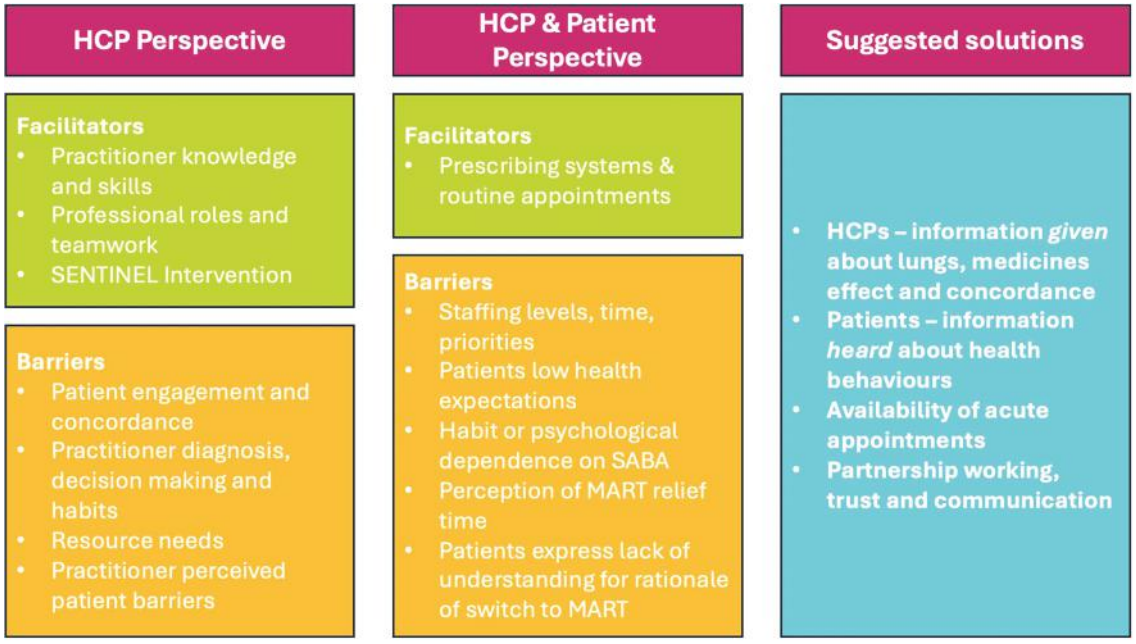
[1]J Dyson, [2]H Cummings, [1]G Williams, [3]V Raja Gopal, [3]M Watt, [3]T Morris, [4]MG Crooks. *[1]Birmingham City University, Birmingham, UK; [2]NHS Humber Health Partnership, Hull, UK; [3]AstraZeneca, London, UK; [4]Hull York Medical School, Hull, UK*

10.1136/thorax-2025-BTSabstracts.243

**Introduction** The 2024 BTS, NICE, SIGN asthma guidelines endorse anti-inflammatory-based management for everyone aged 12 years and over that are newly diagnosed or have uncontrolled asthma. This represents a paradigm shift away from approaches utilising separate ICS-containing preventer and SABA reliever inhalers. SENTINEL is a quality improvement framework that aims to identify and address SABA overuse in asthma through supported implementation of a MART-based adult asthma guideline.

**Aim** To identify the barriers and facilitators to: i) adoption and persistence of MART treatment among adult asthma patients, and ii) initiation and persistence of MART treatment among healthcare practitioners.

**Methods** This semi-structured interview study recruited primary care practitioners and adult asthma patients from a network of general practices that participated in SENTINEL. Theoretically underpinned interviews were undertaken with clinicians and patients and analysis involved deductive coding to the theoretical domains framework followed by indictive coding within domains.




**Abstract P61 Figure 1**

**Results** Thirteen asthma patients (median [range] age 34 [21–67] years, 7 female) and fourteen primary care clinicians (4 general practitioners, 2 pharmacists, 5 practice nurses, and 3 advanced nurse practitioners) took part in interviews. Fourteen themes were identified (figure 1). Facilitators included practitioner knowledge and skills (enhanced by SENTINEL), and practice prescribing and appointment systems. Barriers related to access to resources, inconsistent practice across the healthcare system (e.g. emergency departments), and managing patient-related barriers. Patient barriers predominantly related to fear of SABA discontinuation arising from psychological dependence, a lack of understanding of the rationale for MART, and concern about ability to access care in the event of loss of asthma control following a change in treatment.

**Conclusion** There are a range of barriers to adoption and persistence with anti-inflammatory-based asthma management that need to be acknowledged and addressed if the 2024 BTS, NICE, SIGN asthma guideline recommendations are to be fully implemented in practice. Ensuring clinicians have the required knowledge and skills to support patients transition to MART as part of a positive therapeutic relationship, and adoption of a system wide approach to ensure a consistent approach to asthma management, will provide the greatest chance of successful implementation of anti-inflammatory-based asthma management.

## P62 PATIENT REPORTED EFFECTS OF VAPING ON ASTHMA CONTROL IN SEVERE ASTHMA

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**Introduction** The incidence of vaping is increasing particularly amongst young adults and never-smokers, as addressed in the recent BTS statement. Further, there is mounting evidence from research particularly in children and young adults that vaping can be associated with worse asthma control and asthma exacerbations. We wanted to understand the effects of vaping and vaping exposure on patients with severe asthma.

**Methods A** questionnaire including smoking, active and passive vaping exposure and impact on asthma control was given to severe asthma patients attending a tertiary care clinic. To increase accuracy of patient report this was an anonymised questionnaire although patients could provide their name if they chose to.

**Results** A total of 125 people completed the questionnaire.80 were female and 45 were male. 17/125 had actively vaped, of these 7 (41%) reported worse asthma control due to vaping, 8 (48%) reported vaping had no effect on asthma control and 2 (12%) patients were unsure.

61/125 patients had passive vaping exposure, of these 66% (40/125) noted increased asthma symptoms after passive exposure, 12(20%) had no impact on asthma and 9 (14%) patients were not sure. Notably 15/61(25%) patients could associate sustained loss of asthma control (an exacerbation) after an episode of passive vaping exposure.

**Conclusions** There is concerning link between vaping, vaping exposure and asthma control. The majority of severe asthma patients reported worse asthma symptoms after second-hand vape exposure. The effects of vaping on airways disease should guide public health policy to restrict the locations where vaping is permitted and to clearly communicate to the risks of vaping to the public, particularly those with asthma.

## P63 BRIDGING INEQUALITY THROUGH INNOVATION: SYSTEMS-BASED PROACTIVE APPROACH TO REDUCE ASTHMA EXACERBATIONS IN A NOVEL NURSE-LED MODEL IN AREAS OF SOCIAL DEPRIVATION

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10.1136/thorax-2025-BTSabstracts.245

**Background** Acute Exacerbations in asthma (AEA) can be fatal and both NRAD (National Review of asthma deaths) and BTS have made recommendations around prevention and follow-up post-AEA, however this has been challenging in primary care and further guidance on strategically approaching this is needed. Identification of high-risk patients systematically and proactively is important to optimize care, prevent further




**Abstract P63 Figure 1** Prevalence of Acute Asthma Admissions per registered GP practice in the ICB (Integrated Care Board)

exacerbations and effectively treat difficult and severe phenotypes.

**Methods** Retrospective analyses of patients attending emergency departments (ED) with AEA using ED Dataset (fiscal year 2024–2025) was mapped to GP practices within the integrated care board (ICB) and according to socioeconomic status and index of multiple deprivation. Heat map (figure 1) shows practices with the highest number of ED attendances, where more attention is required for collaborative and integrated outward facing working by the respiratory secondary care teams. Regular monthly sessions with individual practices allows deep dive drilling down to important issues affecting the populations where AEA rates are high and ensure issues addressed like (i) education for staff and patients particularly accounting for locations of more health inequality, (ii) appropriate secondary care review or (iii) referral to other services to evaluate diagnosis and comorbidities.

**Results** 4366 patients were admitted for AEA in the past year of whom, 36% [n=1570] were of white ethnicity, 19% [n=811] black and 7% [n=303] were South Asian. 173 GP practices were reviewed and asthma attendance rate per practice showed 54% practices had rates up to 2%, and 13% of practices above 3%. Figure 1 identifies GP surgeries with highest number of patients with frequent AEA. 404 patients had 2 or more ED admissions. Areas with greatest deprivation showed higher rates of AEA admitted to hospital.

**Conclusion** With one of the worst mortality rates in Europe for asthma, a UK systems-based approach is required to personalize follow-up AEA and is key to prevention of AEA in hotspots of poor asthma control. Specific national guidance is called for to target care to the vulnerable groups in a timely manner and support on the implementation of an integrated approach to ensure delivery of focused attention to those at greatest risk.

## P64 ASSESSING THE EFFECT OF ETHNICITY AND SOCIOECONOMIC STATUS ON OUTCOMES IN ASTHMA: A SYSTEMATIC REVIEW

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10.1136/thorax-2025-BTSabstracts.246

**Background** Social determinants have been associated with adverse outcomes in asthma. The literature suggests particular roles for race/ethnicity and socioeconomic status (SES)[1] [2]. However, the vast majority of this work is from North America, which may limit its applicability in other contexts.

This systematic review aimed to summarise the literature specific to the United Kingdom (UK) with a long-term view of informing locally applicable intervention/policy development.

**Methods** Searches were conducted in July 2023 and updated in April 2024. Studies were identified from Ovid Embase, Ovid MEDLINE and Web of Science from 1998 onwards. Included studies examined objectives related to UK minority ethnicity (UKME) and/or SES in adult asthma in the UK.

Citations were managed using www.covidence.org. Screening, extraction and quality assessment were undertaken by a team of reviewers. Data were extracted using a priori criteria adapted on review of initial studies.

The study was registered in PROSPERO.

**Results** An examination of over 25 years of the UK literature found 23 studies assessing the impact of UKME and/or SES on asthma outcomes. Studies had inconsistent ways of assessing outcomes and potential mediating factors.

Of six studies assessing the impact of UKME on asthma hospitalisations, four found an increased risk of hospitalisation associated with UKME. All five studies assessing the impact of SES found an increased risk of hospitalisation associated with low SES.

**Conclusion** The UK literature suggests that UKME and SES are associated with asthma hospitalisations. A greater understanding of precisely which groups are most affected, in what contexts and through which mediating factors is required to effectively translate established knowledge into effective service and policy interventions.

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## P65 EXPLORATION OF THE PERCEPTIONS OF SOUTH ASIAN PATIENTS WITH ASTHMA: SOUTH ASIAN RESPIRATORY INTERVENTIONS (SARIS) STUDY

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10.1136/thorax-2025-BTSabstracts.247

**Introduction and Objectives** NICE and GINA guidelines recognise that ethnicity can impact asthma outcomes, with South Asian and Afro-Caribbean populations experiencing higher rates of asthma-related morbidity and hospitalizations compared to the White British population. It has been suggested that a number of culturally specific factors might explain the impact of ethnicity on health outcomes and that cultural sensitivity is paramount in the managing asthma and addressing health inqualities1,2 3.

**Method** A qualitative study was conducted in 2014 to explore the perceptions of South-Asian asthmatics, aged > 18 years on their understanding of asthma, its impact on health and to identify any cultural beliefs and barriers that may influence their management. Views were sought on the development of culturally sensitive services. Data was collected through four focus groups, in 4 community settings in Leicester City. Participants were from Moslem, Punjabi and Hindu backgrounds. The resulting data was analysed using thematic framework analysis.

**Results** The majority of participants (N=48) were aware of asthma triggers and were actively avoiding these. Although many were confident of the beneficial effects of homemade remedies, none were substituting them for prescribed medicines. Most participants understood asthma is prevalent within South-Asian communities. There was a consensus that inhalers

were not used in public due to stigmas attached to asthma within the South-Asian community. Although language was a barrier for some, participants had found effective ways of overcoming this, including using family members and translators to interpret. Barriers to the provision of information were highlighted. All participants placed their trust in healthcare professionals (HCPs), but believed time constraints restricted appropriate counselling. There was agreement that educational materials in picture format, or digital in their preferred language would support this.

**Conclusion** Trust in the advice was a key finding. HCPs need to educate and address concerns within the South-Asian community about asthma whilst taking into account patients' cultural beliefs that influence their management. This study was conducted in 2014, however, the findings still resonate with the current literature highlighting valuable insights of the importance of social and cultural factors that influence the management of asthma in the South Asian community.

## P66 IMPROVING ASTHMA OUTCOMES IN LANCASHIRE AND SOUTH CUMBRIA (L&SC) THROUGH SABA REDUCTION AND MART IMPLEMENTATION WITH SENTINEL PLUS

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10.1136/thorax-2025-BTSabstracts.248

**Introduction** There has been a concern nationally regarding the over-reliance on SABA -only reliever among asthma patients which can lead to poor asthma control, increased risk of exacerbations, and potential long-term consequences including avoidable excess carbon footprint.

**Strategy for change** L&SC ICB joint working with AstraZeneca using SENTINEL Plus, a quality improvement programme that aims to improve asthma management in patients, identifying and addressing Short-acting Beta-2 Agonist (SABA) over-use and implementing Maintenance and Reliever Therapy (MART).

**Methods** From October 2023 to September 2024, Interface Pharmacy Services analysed routinely collected NHS data to form a baseline and conducted asthma reviews aligned with SENTINEL Plus in 20 practices where 14,590 asthma patients are registered. Clinicians were provided with educational sessions on asthma management to adopt sustainable implementation of the national guidance. Changes in prescribing practice were reported using descriptive statistics.

**Results** 1,048 patient reviews were undertaken.

- 598 (57.1%) patients had their short acting bronchodilator SABA-only or short acting muscarinic antagonist (SAMA) stopped
- 753 (71.9%) patients commenced on MART
- 932 (88.9%) reviewed patients received advice on worsening asthma control and provision of a personalised asthma action plan
- 89 patients out of 170 (52.3%) patients who smoke expressed wanting to stop smoking and were signposted/ referred for smoking cessation service.
- 1,039 (99%) patients had their inhaler checked

**Conclusion** The Lancashire and South Cumbria ICB joint working approach created a collaborative framework to encourage an interdisciplinary approach and foster partnerships across multiple stakeholders in the ICB.

This resulted in a reduction in SABA-only prescribing, and an increase in MART. Implementing guideline recommended therapy is critical to improve outcomes and to support decarbonising healthcare delivery.

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## P67 WHAT PROPORTION OF PATIENTS ATTENDING A&E WITH ACUTE ASTHMA COULD BE DISCHARGED ON MART/AIR?

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10.1136/thorax-2025-BTSabstracts.249

**Introduction** New NICE/BTS/SIGN guidelines recommend patients with uncontrolled asthma switch to AIR/MART regimens except those on high-dose inhaled corticosteroids who need specialist review. Most inhaler changes are expected to occur in primary care, however, some patients rely on Emergency Departments (EDs) and Urgent Treatment Centres (UTCs) for their asthma care to the exclusion of attending their GP. Maximising this opportunity is important, however, our previous research has identified that EDs will need planning and clear guidance to undertake inhaler changes.

**Objectives** To inform and prior to implementation of NICE-compliant local guidelines we reviewed all patients attending three acute hospitals EDs and co-located UTCs with acute asthma to assess the proportion that would be suitable for switching to AIR/MART.

**Methods** An electronic search of medical records followed by clinician review of identified medical records.

**Results** An electronic search of medical records identified 1320 potential asthma ED attendances between 9th January 2024 and 9th June 2024 inclusive. On clinician review, 755 attendances were confirmed to be for an exacerbation of an established diagnosis of asthma and where the patient did not self-discharge.565 (75%) of these attendances were managed without hospital admission. Of the 565 patients, at least 6 months of prior primary care prescription records were available for 417 (74%) patients. Over that period 121 (29%) patients had had no ICS prescribed, 104 (25%) had had a high-dose ICS-LABA prescribed (49 (12%) with prescription possession ratio for ICS <75%), with the remaining 192 (46%) patients being prescribed low to medium strength ICS (20 with at least 1 AIR/MART prescription).

210 (37%) patients were discharged from ED with a SABA reliever (including 10 patients potentially on MART), 1 (<1%) patient with an anti-inflammatory reliever and 1 (<1%) patient with both SABA and anti-inflammatory relievers. Of the 190 patients discharged home after admission, 17 (9%) patients were discharged with an anti-inflammatory reliever though in 4 of those cases also with a SABA reliever.

**Conclusions** The majority (87%) of patients attending EDs and UTCs with acute asthma would be suitable to receive an anti-inflammatory reliever on discharge as part of an AIR/MART inhaler regimen.

## P68 ARE HOSPITAL ORGANISATIONAL FACTORS ASSOCIATED WITH THE DELIVERY OF THE ADULT ASTHMA DISCHARGE BUNDLE? AN ANALYSIS OF DATA FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME

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10.1136/thorax-2025-BTSabstracts.250

**Introduction** The completion of an asthma discharge bundle (DCB) is a core component of high-quality care for patients admitted with asthma exacerbations. However National Respiratory Audit Programme (NRAP) data shows that 20% of patients do not receive a DCB during their admission.[1]

**Aim** 1. Describe the variation in delivery of the asthma DCB across hospitals in England & Wales,

2. Investigate which organisational-level factors are associated with DCB completion.

**Methods** We used data from the 2023–24 NRAP to compare DCB completion in patients who had data submitted to the clinical audit for their exacerbation (n=18884 admissions). We extracted data from the 2024 NRAP snapshot *Organisational Audit* to compare key organisational factors between the top 25% performing hospitals for DCB completion vs. other hospitals.

**Results** The proportion of patients receiving a DCB varied greatly between hospitals, with a median of 80% (interquartile range 45–96%) (figure 1). There was similar variation across integrated care systems, from 6% to 100%.

The top 25% of hospitals for delivery of the asthma DCB reported having higher median respiratory nurse specialist (RNS) per 300 adult asthma admission (5.7 vs. 4.5), and higher weekend RNS availability (31% vs 20%), when compared to the other 75% of hospitals.

**Conclusion** There is concerning variation in the reported delivery of asthma discharge bundles across England & Wales. Whilst there are likely to be a number of complex factors associated with DB delivery, services should consider addressing RNS staffing and 7-day working, in efforts to improve the delivery of basic asthma care.

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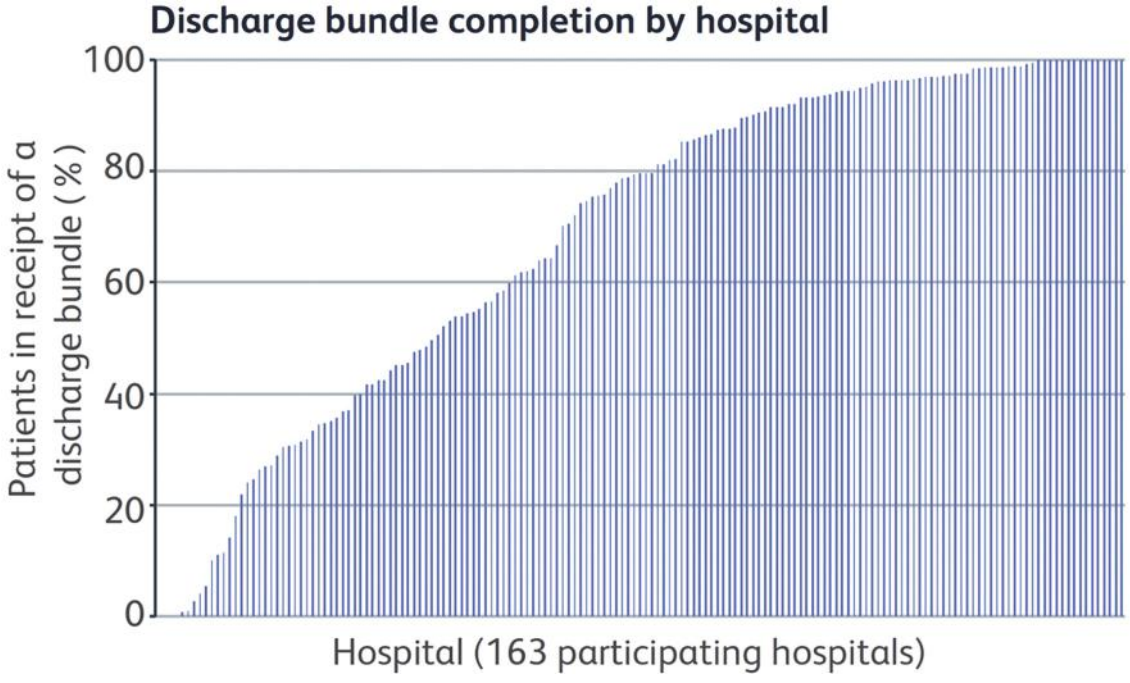
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## P69 INTENSIVE CARE ADMISSIONS FOR ASTHMA ARE CHARACTERIZED BY FREQUENT NON-ATTENDANCE TO OUTPATIENT APPOINTMENTS

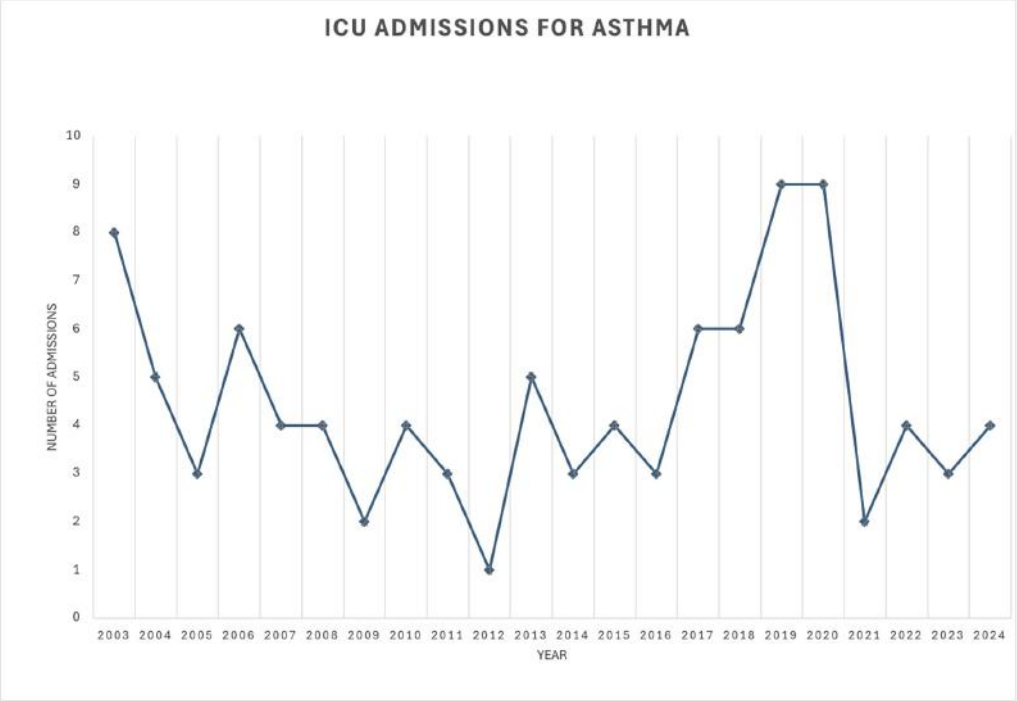
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10.1136/thorax-2025-BTSabstracts.251

**Introduction** Asthma exacerbations result in approximately 43 hospitalizations per 100,000 in Ireland.[1] They are associated with considerable morbidity, mortality, and healthcare costs. Despite improved specialist services, better ICS/LABA therapies and targeted monoclonal antibodies, it's unknown if ICU admission for asthma exacerbations have decreased.




**Abstract P68 Figure 1** The percentage of patients who received discharge bundles during admission for each of the 163 hospitals who submitted data to the NRAP clinical audit from 01/04/2023–31/03/2024




**Abstract P69 Figure 1** Demonstrates an upsurge in ICU admissions during the 2019–2020 period

**Aim** We aimed to assess any change in trends in ICU exacerbations for asthma in our hospital from 2003–2024, and investigate potential reasons for lack-of-change. We hypothesised that non-engagement in services is an important reason for ICU admission.

**Methods** A retrospective analysis of ICU admissions for asthma in our institution from 2003 onwards was undertaken. Demographics, respiratory supports, presence of T2 inflammation, length of ICU stay (LOS), mortality and lung function were analysed.

**Results** 63 patients had 99 ICU admissions for asthma over this period. Median LOS was 2 days (IQR 1.6–4 days). There was a female predominance (n= 35, 55%). Median age was 47.5 years. T2 inflammation was prevalent, with 85% having evidence of peripheral blood eosinophil (PBE) $\geq$0.3, with median maximum recorded PBE of 0.51 (IQR 0.34–0.89). There were three asthma related deaths. Figure 1 demonstrates annual ICU admissions for asthma.

PFT data were available for only 39 patients. Of those with a single ICU admission (n=23), five met ERS criteria for asthma.[2]

High rates of non-attendance to scheduled outpatients was notable, with a median of 14 missed-attendances per patient (range 0–139).

**Conclusion** Despite improvements in asthma therapies, we have not seen a reduction in ICU presentations the past 20 years. While trends from 2020–2022 may reflect the Covid-19 pandemic, lack of engagement in healthcare when well, evidenced by high DNA/cancellation rates to outpatient appointments, remains a prominent issue in this population, and may contribute to these patients' severe exacerbations. Ways to engage this cohort are imperative to improve outcomes.

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## P70 ASTHMA BIOLOGIC CASE-FINDING, AN EVALUATION OF A PRAGMATIC PHYSICIAN-LED COMMUNITY SCREENING PROGRAMME WITHIN BRISTOL COMPLEX AIRWAYS SERVICE

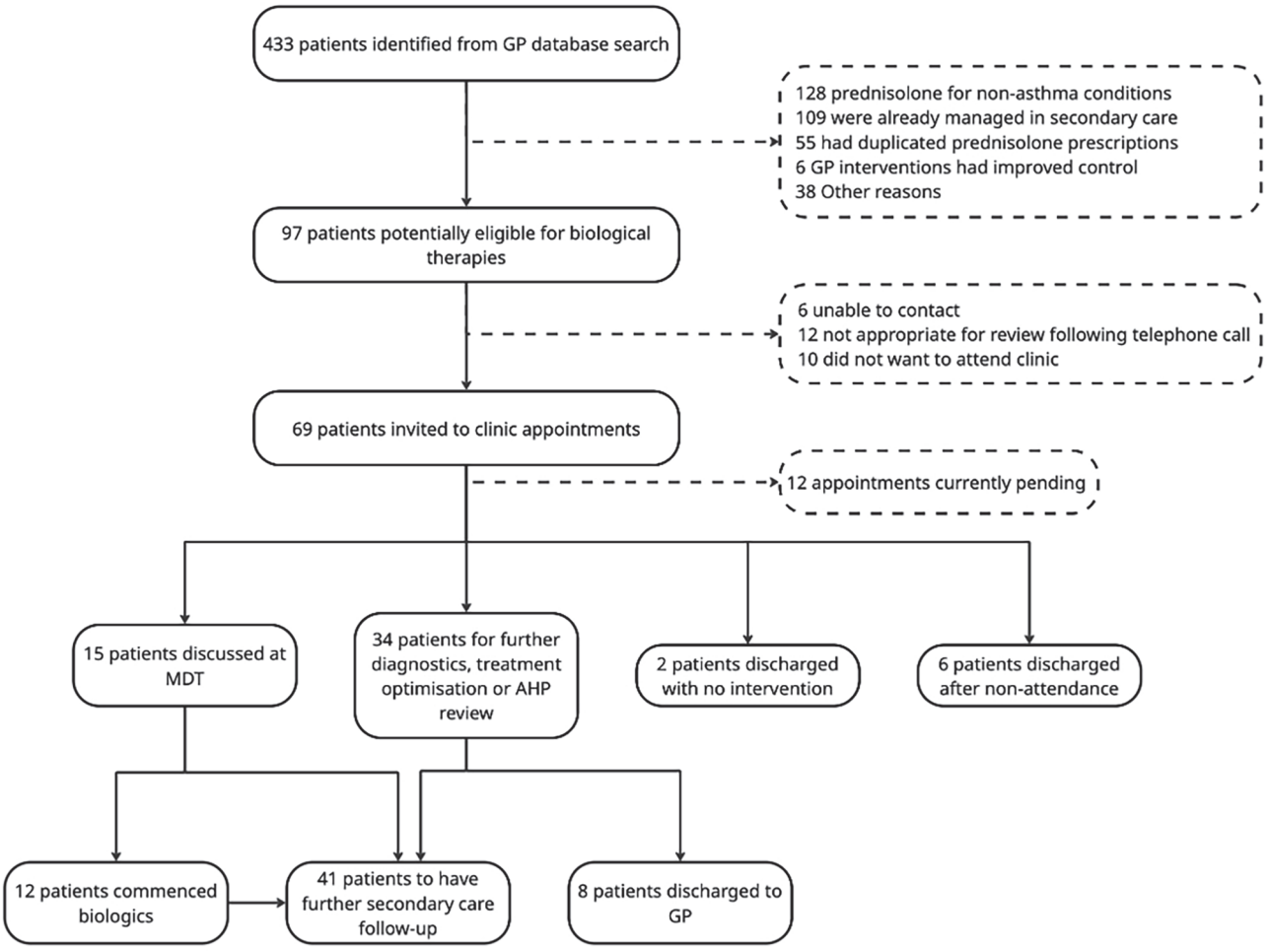
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10.1136/thorax-2025-BTSabstracts.252

**Introduction** Estimates suggest that thousands of patients across the UK, unknown to secondary care, are eligible for asthma biologic therapy.[1] This includes underserved populations who are at risk of poorer asthma outcomes. Many GP practices do not have a method of identifying such patients.[2] Case-finding programmes can be burdensome for overstretched primary care services and have previously struggled with patient engagement with secondary care.

**Aim** To assess the impact of a physician-led community asthma biologic screening programme.

**Methods** A data sharing agreement was signed with a Bristol GP collaborative who conducted a search in January 2025 across 33 practices using the criteria: 18+ years, asthma diagnosis, without prescription of asthma biologic, 3+ prednisolone prescriptions and an inhaled corticosteroid in the last year and eosinophil count $\geq$0.3 in the last two years. Electronic healthcare records were screened by a registrar or consultant to assess suitability for clinical review. Patients received a letter and telephone call to confirm suitability, invite them to the appointment, and address barriers to clinic attendance.




**Abstract P70 Figure 1** Consort diagram from patient identification to first clinic completion

**Results** 433 patients were identified from the search. 97/433 (22%) were deemed potentially eligible for asthma biologics (figure 1). 69 patients agreed to attend clinic. To date, 57 of these appointments are complete. 15/57(26%) patients were discussed at the complex airways MDT with 12 patients starting asthma biologics. 33/57(58%) patients received optimisation of asthma care, referral for diagnostics and/or allied health professional review, with 26 to have follow-up. 2 patients were discharged without intervention and 6 due to non-attendance.

**Conclusion** We demonstrate a pragmatic respiratory physician-led community screening programme for uncontrolled severe asthma. Most patients seen in clinic benefitted from treatment optimisation and 21% of patients reviewed have commenced biologic therapies - this proportion is likely to increase after follow-up clinics. An important success was the high clinic attendance rate, a barrier that we anticipated and overcame with our project design. Further work is required to expand this project to other practices and consider other asthma phenotypes.

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## P71 LIVING WELL WITH DIFFICULT ASTHMA: BELIEFS ABOUT BREATHLESSNESS MANAGEMENT THROUGH LIFESTYLE CHANGES IN A DIETITIAN-, PHYSIOTHERAPY-, AND PSYCHOLOGY-LED MULTIDISCIPLINARY GROUP

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10.1136/thorax-2025-BTSabstracts.253

**Introduction and Objectives** For many patients with severe asthma, breathlessness is the predominant symptom. Breathlessness experiences are highly individual, and there is no one resolution for all presentations. We introduced a multidisciplinary group intervention combining nutrition, exercise, and psychological support to improve beliefs about breathlessness and promote long term changes in symptom management.

**Methods** A six-week group programme was conducted for individuals with severe asthma and obesity (BMI class I or above), led by a Specialist Physiotherapist, Dietitian, and Clinical Psychologist. Each two-hour session included one hour of interactive education, 45 minutes of circuit exercise, and 15 minutes for questions and peer discussion. Ten patients were invited; four attended over 75% of sessions, and two attended 50%. Beliefs about breathlessness were assessed using a modified Beliefs about Breathlessness Questionnaire, adapted to include nutrition and psychological elements. The questionnaire was conducted at screening, week one, four, and six.

**Abstract P71 Table 1** Health belief questionnaire outcomes pre- and post- multidisciplinary group therapy

| | Pre | | | | | Post | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Strongly Disagree | Disagree | Neither Agree nor Disagree | Agree | Strongly Agree | Strongly Disagree | Disagree | Neither Agree nor Disagree | Agree | Strongly Agree |
| Feeling short of breath should not stop me from exercising | 0 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 2 | 2 |
| I can do most of the things that healthy people can do despite my asthma | 1 | 2 | 0 | 1 | 0 | 0 | 1 | 1 | 2 | 0 |
| I am confident to make healthy lifestyle changes to better manage my asthma | 0 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 2 | 2 |
| My shortness of breath would probably be better if I were to exercise more | 0 | 1 | 1 | 0 | 2 | 0 | 1 | 0 | 1 | 2 |
| Being overweight makes my breathlessness worse | 0 | 1 | 1 | 2 | 0 | 0 | 0 | 0 | 2 | 2 |
| Reducing my weight would improve my shortness of breath | 0 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 2 | 2 |

**Results** Table 1 summarises the key results from pre and post group. All patients agreed/strongly agreed feeling confident to make healthy lifestyle changes and this remained unchanged from pre- to post-group therapy. Participants consistently agreed pre- and post-group, that reducing their weight would improve their breathlessness. However, we found participants to report more positive beliefs regarding specific lifestyle changes to better manage their asthma following multidisciplinary group therapy. Beliefs about shortness of breath limiting exercise improved from disagree/neither agreed nor disagree, to agree/strongly agree. Other positive shifts were seen in beliefs relating to being able to do activities that healthy people do, that exercising more would improve their breathlessness, and that being overweight worsens breathlessness.

**Conclusions** Patients reported unexpectedly high baseline confidence in making lifestyle changes, however, post-group therapy survey feedback indicated that the majority felt their confidence had increased. The most notable improvements were observed in exercise beliefs and the role of weight, with smaller changes in general health beliefs. This multidisciplinary group approach shows promise for improving lifestyle-related breathlessness beliefs in patients living with difficult asthma.

## P72 REDUCTION IN HEALTHCARE UTILISATION AND INAPPROPRIATE PHARMACOLOGICAL THERAPY FOLLOWING MANAGEMENT OF REFRACTORY BREATHLESSNESS

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10.1136/thorax-2025-BTSabstracts.254

**Background** Inducible laryngeal obstruction (ILO) and breathing pattern disorder (BPD) often co-exist with or are misdiagnosed as asthma.

**Aim** To identify patients in the Manchester complex breathlessness service with suspected incorrect asthma diagnosis, and report subsequent change in healthcare utilisation, symptoms and medication use following nurse-led breathlessness management.

**Methods** Clinical outcomes were collected over a 12-month period following initial assessment, including: asthma control questionnaire (ACQ-6), lung function, medication use and healthcare utilisation.

**Results** Of 32 patients [30 female, median (IQR) 48 (41–58) yrs], 15 had ILO, 7 BPD and 10 both. 27 were treated with inhaled corticosteroids, and two with daily prednisolone. All had normal lung function and FeNO, and seven had a bronchial challenge (all negative). Pretreatment, median (range) 1 (0–22) ED attendances, 1 (0–7) admissions, 1 (0–12) steroid courses, 1 (0–10) antibiotic courses in previous 12 months and ACQ 2.15 (0–5.4). Patients had on average three follow-up visits with the clinical nurse specialist alongside input from speech and language therapy for ILO and physiotherapy for BPD where appropriate. Following intervention, there were significant improvements in hospital admissions, steroid/antibiotic courses and ACQ score (all $p<0.05$). 30 patients stopped their reliever completely, 18 their inhaled corticosteroids and none needed maintenance daily prednisolone.

**Conclusion** Correct diagnosis and reduction of unnecessary pharmacotherapy in patients presenting with refractory breathlessness can reduce treatment burden, healthcare utilisation and improve symptoms.

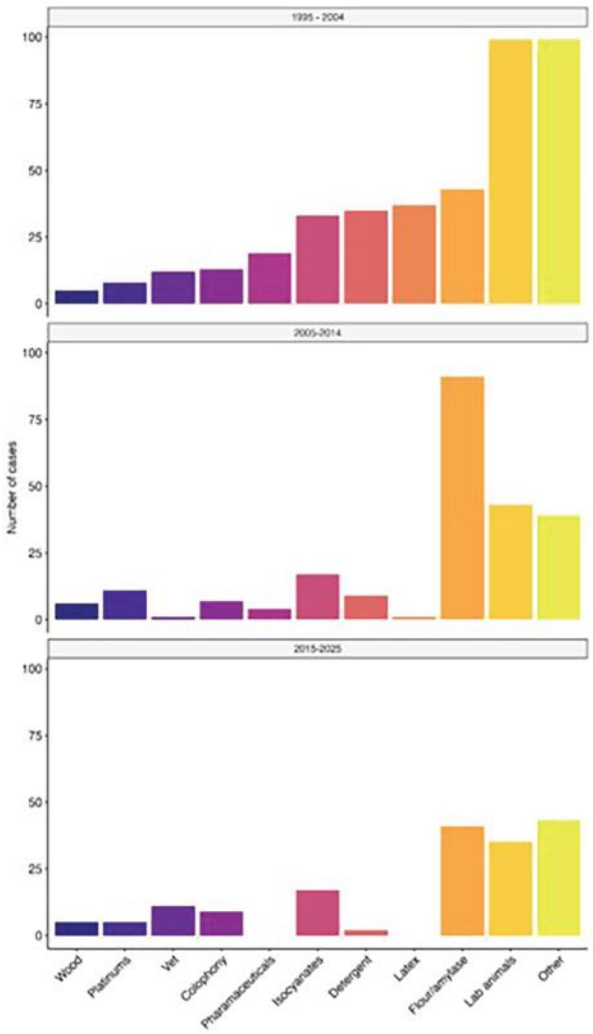
## P73 CHANGING TRENDS IN OCCUPATIONAL ASTHMA CASES IDENTIFIED AT A SINGLE SPECIALIST CENTRE OVER 30 YEARS

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10.1136/thorax-2025-BTSabstracts.255

**Background** The Occupational Lung Disease (OLD) service at Royal Brompton Hospital was established in the 1970s and is one of the largest referral centres in the UK. Around 60% of current referrals are from occupational health services following health surveillance for occupational asthma (OA). All confirmed cases of OLD identified at the clinic are recorded in a database and reported to the UK-wide SWORD (Surveillance of Work Related and Occupational Respiratory Disease) scheme. A previous publication suggested a downward trend in referrals to SWORD from 1999 to 2014 with a possible uplift from 2014–2016.[1]

**Methods** We carried out a retrospective review of all OA cases seen in our clinic over 20 years from 1995–2025 to explore trends in incidence by causal agents.




**Abstract P73 Figure 1** Causal agents for confirmed cases of OA by decade (1995–2025)

**Results** We identified a total of 800 cases with the total number of cases appearing to have declined over time: n=408 (1995–2005), n=229 (2005–2015) and n=168 (2015–2025). The most common causative allergens have consistently been bakery allergens and laboratory animals, followed by isocyanates, detergent enzymes, colophony and platinum groups salts. Incidence of OA from bakery allergens, laboratory animals and detergent enzymes has fallen; in contrast, OA due to wood, platinum group salts and colophony have remained relatively stable. Latex and pharmaceuticals are no longer frequent causes of OA in our service. (figure 1) Analyses relating to age and sex of cases are ongoing.

**Conclusion** Incident OA in our clinic has fallen the last 30 years in keeping with SWORD data.[1] This likely reflects primary prevention of disease resulting from implementation of the hierarchy of control of exposures in workplaces including removal of exposures (e.g. powdered latex gloves in the NHS) and from introduction of engineering controls (e.g. individually ventilated cages for laboratory rodents). However secondary prevention using health surveillance remains important where there is residual risk of disease. This is particularly so in the baking industry, laboratory animal research and in workers exposed to isocyanates in whom we continue to diagnose significant number of cases of OA in our clinic annually.

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## P74 IMPROVING OUTCOMES IN DIFFICULT ASTHMA: CLINICAL IMPACT OF PHYSIOTHERAPY AND SPEECH AND LANGUAGE THERAPY IN MULTIDISCIPLINARY CARE

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10.1136/thorax-2025-BTSabstracts.256

**Introduction** Asthma is a chronic respiratory condition affecting over 300 million people globally, with difficult asthma comprising 5–10% of cases. People with difficult asthma often experience persistent symptoms that are resistant to standard high-dose treatments, contributing to a significant public health and economic burden. Multidisciplinary assessments, including input from speech and language therapy (SALT) and physiotherapy, have been proposed to complement traditional pharmacological interventions by targeting symptom control and reducing exacerbations, particularly in individuals with low inflammatory biomarkers (Type 2-low). This study evaluates the clinical impact of these therapies on outcomes in people with difficult asthma.

**Methods** A retrospective study was conducted at the Leeds Difficult Asthma Clinic. Data from 50 patients treated with SALT or physiotherapy between January 2020 and December 2021 were collected. Outcomes included Asthma Control Questionnaire (ACQ) scores, spirometry parameters, exacerbation frequencies, and treatment discharge rates. Statistical analyses assessed the effectiveness of these interventions, with comparisons made pre- and post-treatment at 12 months.

**Results** The cohort included 41 females and 9 males, with a median (range) age of 47 (17–82) years. At baseline, the median (range) BMI and $FEV_1$ were 30.7 (16.5–56.0) and 2.27 (0.64–4.25) L, respectively. Median (range) eosinophil counts were low; 0.18 (0.01–23.1) $\times$ $10^9$ cells/L, consistent

with a Type 2-low asthma phenotype. A significant overall reduction in exacerbations was observed (32.7%, p=0.03), with females experiencing a greater decrease (38.6%, p<0.05). BMI, $FEV_1$, and ACQ scores did not show significant changes (p>0.05), Notably, 42% of patients were discharged from therapy, suggesting improved symptom control and meaningful clinical improvement.

**Conclusion** This study highlights the potential of SALT and physiotherapy as effective adjunctive therapies for difficult asthma. Given the substantial reduction in exacerbation rates and the significant proportion of patients achieving successful discharge, these non-pharmacological approaches represent a valuable addition to difficult asthma assessment and management. They may also offer a cost-effective alternative to biological therapies, particularly for people with Type 2-low asthma, who often respond poorly to conventional pharmacological interventions. Future research should further explore the long-term impact and broader applicability of these therapies across diverse populations.

## P75 DEVELOPMENT OF THE UK SEVERE ASTHMA REGISTRY AS A PERFORMANCE-MONITORING QUALITY IMPROVEMENT PLATFORM

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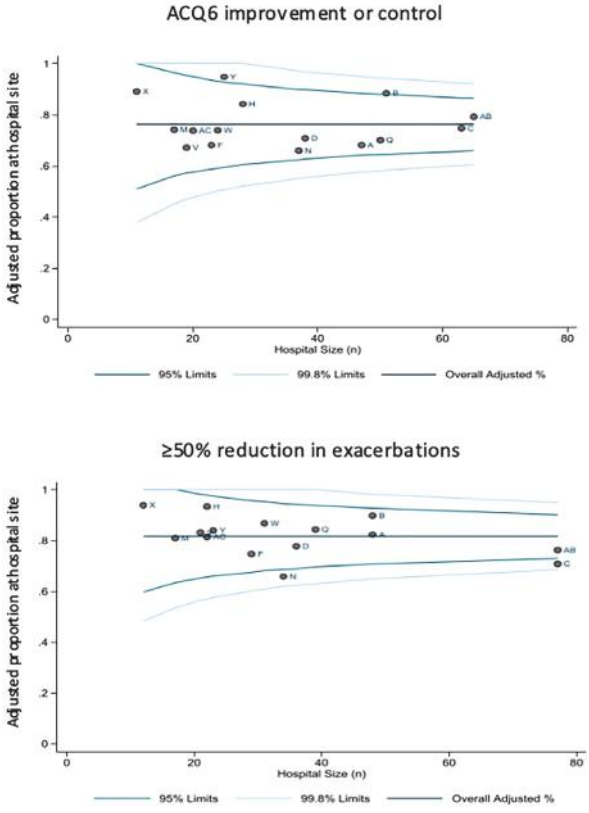
10.1136/thorax-2025-BTSabstracts.257

**Introduction and Objectives** The UK Severe Asthma Registry (UKSAR) was established to facilitate research and quality improvement including performance monitoring across UK severe asthma centres. We here describe its development and refinement for the latter of those objectives. The selection of performance outcomes measures for severe asthma clinical quality improvement from clinician and patient perspectives, data collection, and assessment of clinical variation across severe asthma centres is presented.

**Methods** After relevant stakeholder consultation (patients, commissioners and clinicians), outcomes measures relating to exacerbations, oral corticosteroid exposure, asthma-related ED attendances and hospital admissions, asthma control and quality of life were selected with appropriate case-mix adjustment to help identify unexplained clinical variation. Over 2018–2025, the number of participating centres increased from 17 to 42. To date, baseline data has been entered for 18,473 patients, with data meeting the agreed quality threshold for 12,792 patients.

**Results** Compared to 2018, in 2024 patients were older when first reviewed (54.0 vs 50.3 years), less likely to be never smokers (58.7% vs 65.8%) and to be on mOCS at baseline (25.5% vs 47.1%).

Analysis of variation in outcomes between centres, after case-mix adjustment and allowing for effect of centre size, shows relatively few examples of significant variation in outcomes between centres. Where variation was evident, for example in improvement in asthma control ACQ6 score from baseline to follow-up (see figure 1) high-performing centres could be identified to share best-practice.




**Abstract P75 Figure 1** Proportion of patients with positive outcomes for the above metrics, by hospital size, for patients with baseline records on or after 1 January 2022

**Conclusions** Regular discussion at UKSAR registry meetings, at which representatives from each severe asthma centre are present, of outcome measures and related latest research, including UKSAR research, together with sharing of best practice may explain why there is relatively little evidence of variation in clinical outcomes between centres. UKSAR shows that there are an increasing number year-on-year of patients on severe asthma biologic treatments and of hospitals both prescribing biologics and entering data for quality improvement. However, a limitation of UKSAR is that it cannot evaluate the presence of 'hidden' severe asthma not known to specialist services, a continuing issue given the harm that results from delays in appropriate diagnosis and management, and accompanying oral corticosteroid associated comorbidities.

P76 **EQUITY AND ACCESS IN SEVERE ASTHMA CARE: PATTERNS OF BIOLOGIC THERAPY INITIATION ACROSS ENGLAND**

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10.1136/thorax-2025-BTSabstracts.258

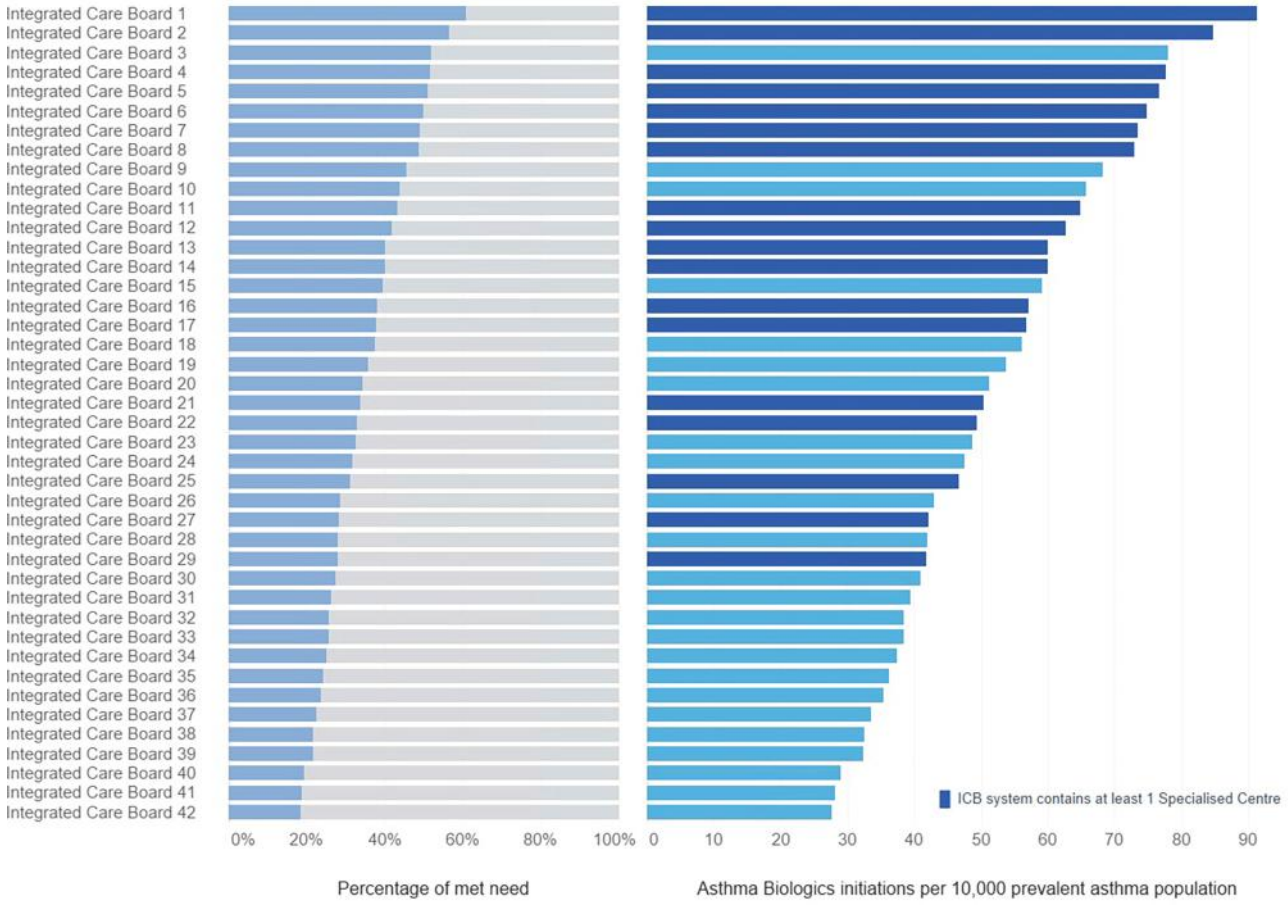
**Background** Biologic have revolutionised the management of severe asthma, reducing exacerbations, hospital admissions, and oral corticosteroid use, while improving quality of life. In England, biologics are initiated at nationally commissioned severe asthma centres for patients with uncontrolled severe asthma. We aimed to assess patterns of biologic initiation and identify potential inequities in access.

**Methods** Biologic initiation data from Blueteq (January 2016-February 2025) were linked to patient-level datasets, including Hospital Episode Statistics (HES). Initiation rates were compared to the estimated eligible population (1.5% of the QOF adult asthma population) across Integrated Care Boards (ICBs). Demographics, including ethnicity and socioeconomic status, were evaluated to explore inequities in access.

**Results** Biologic uptake increased from 738 patients in 2016 to over 21,000 by early 2025. Since 2021, the number of patients on biologics has grown by 23–27% annually. Annual initiations more than doubled from 2,008 (2021–2022) to 4,044 (2024–2025). However, an estimated 37,000 eligible patients remain untreated. Initiation rates varied markedly across ICBs- from 20% to 60% of eligible patients, with higher uptake in regions served by specialist centres (figure 1). Proximity to severe asthma centres correlated with higher initiation rates.

No significant disparities were found in initiation rates by gender, age, ethnicity, or deprivation index. Biologic initiation was associated with a 50% reduction in asthma-related non-elective hospital admissions and bed-days in the year following initiation compared to the previous year- in 2023, among 3,634 patients, hospital admissions decreased from 1,650 to 790 and bed-days from 6,500 to 3,500.

**Conclusion** While biologic use has steadily increased, a large unmet need persists. Based on our conservative estimate that 1.5% of adult asthma patients require biologic treatment, approximately 66% (~37,000 patients) of eligible individuals are not yet receiving therapy. The absence of demographic inequity is encouraging, but substantial regional variation suggests structural barriers in access. Expanding expertise and assessment capacity beyond specialist centres, alongside earlier identification of patients with uncontrolled asthma and timely referral from primary care are essential to improve equity, reach underserved populations and ensure that all eligible patients benefit from systematic assessment, multi-disciplinary team input and these transformative therapies.




**Abstract P76 Figure 1**

# Best practice in pleural procedures and pneumothorax

## P77 A NATIONAL SURVEY OF REAL-TIME ULTRASOUND GUIDANCE USE IN PLEURAL PROCEDURES IN THE UNITED KINGDOM

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10.1136/thorax-2025-BTSabstracts.259

**Introduction** Pre-procedural thoracic ultrasound is a mainstay of pleural intervention, with evidence that its use reduces complication risk. However, there are currently two forms of standard clinical practice for ultrasound use during thoracocenteses and chest drain insertions. In the first, 'real-time' ultrasound guidance is used to visualise the inserted needles and tube throughout the procedure, and anaesthetic is administered with the needle tip seen adjacent to the parietal pleura. In the second, the operator inserts needles and the tube not under 'real-time' ultrasound guidance, and anaesthetic is injected at a depth approximating that of the external pleural lining. To date, there has been limited comparison of the two techniques, however there is some evidence that real-time ultrasound guidance improves success rates and reduces the likelihood of complications.[1] The current use of real-time ultrasound guidance for pleural procedures in the United Kingdom (UK) is unclear.

**Methods** An electronic survey (Jisc Online Surveys) was circulated via the UK Pleural Society and British Society of Thoracic Imaging newsletters, and through direct communication with centres undertaking pleural procedures.

**Results** 70 responses were received, predominantly from Respiratory Consultants (33%), Radiologists (30%) and Respiratory Registrars (29%). 50% of respondents 'Never' or 'Rarely' use real-time ultrasound guidance in pleural procedures, while 37% use it 'Always' or 'Often'. Frequently highlighted reasons for not using real-time ultrasound guidance in pleural procedures included a lack of training, technical issues, increased procedure length and insufficient evidence that the technique improves outcomes. The majority of respondents (74%) indicated that they would use real-time ultrasound guidance more for thoracocentesis and chest drain insertions if the technique was shown to significantly reduce pain experienced by patients.

**Discussion** There is significant variability in the use of real-time ultrasound guidance in pleural procedures across the UK. Most respondents indicated that they would increase use of the technique if it were shown to improve outcomes for patients. Randomised studies to assess outcomes of procedures performed under real-time ultrasound guidance are essential.

### REFERENCE

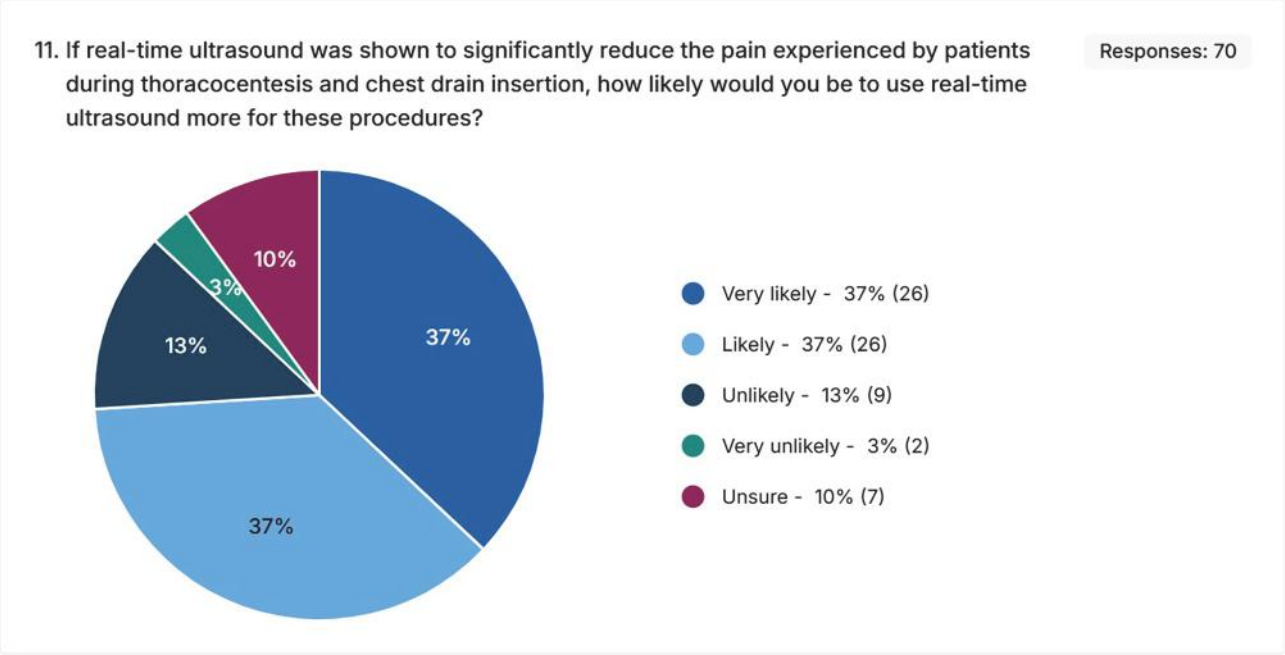
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## P78 THE ROLE OF THE GENERAL MEDICAL REGISTRAR IN PERFORMING OUT-OF-HOURS PLEURAL PROCEDURES: A UK NATIONAL SURVEY OF TRAINEE AND TRAINER PERSPECTIVES

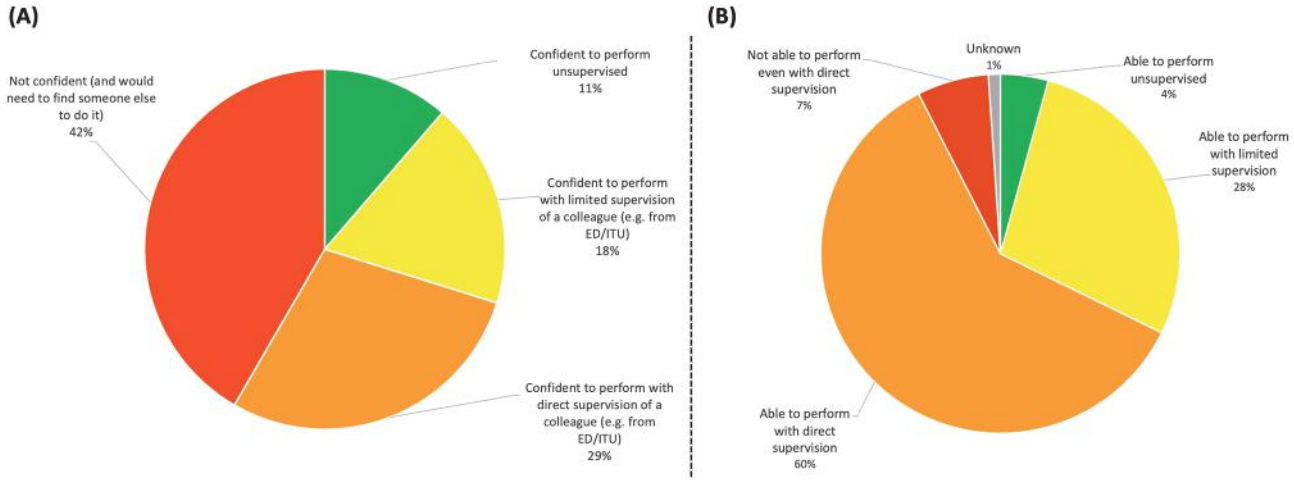
[1]D Jajbhay, [2]A Aujayeb, [1]P Denniston, [3,4]M Juniper, [1]J Liang, [3]H Shotton, [1]J Zhang, [1]O Kadwani. *[1]Guy's and St Thomas' NHS Foundation Trust, London, UK; [2]Northumbria Healthcare NHS Foundation Trust, Newcastle, UK; [3]National Confidential Enquiry into Patient Outcome and Death, London, UK; [4]Great Western Hospital, Swindon, UK*

10.1136/thorax-2025-BTSabstracts.260

**Introduction** Pleural procedural competencies are part of the UK general internal medicine (GIM) curriculum, with a widespread expectation that GIM registrars should be able to




**Abstract P77 Figure 1**




**Abstract P78 Figure 1** In the context of out-of-hours seldinger chest drain insertion for pneumothorax, **(A)** the self-rated confidence of UK non-respiratory GIM registrars (n=389) and **(B)** respiratory consultants' estimates of the pleural procedural competence of an average non-respiratory GIM registrar (n=93)

perform these procedures out-of-hours (OOH) if required. GIM registrar exposure to pleural procedures has declined, with procedures increasingly done by specialists in-hours. Pleural interventions risk significant harm. We explored GIM registrars' perceived competence and confidence in performing OOH pleural procedures, from the perspective of both GIM registrars and respiratory consultants.

**Methods** An anonymised survey was distributed to non-respiratory GIM registrars and respiratory consultants across the UK.

**Results** We received responses from 389 registrars (representing all training grades and 14 specialties) and 93 consultants (including 12 with training programme director experience). Only 37% of registrars reported being 'signed-off' as independent in inserting a chest drain for pneumothorax. 69% had not done a pleural procedure for at least a year. Regarding perceived confidence for OOH chest drain insertion for pneumothorax, 42% of registrars reported no confidence to perform the procedure even if directly supervised, with only 11% feeling confident to do it unsupervised. Respiratory consultants were asked to rate the pleural procedural competence of an average non-respiratory GIM registrar, 88% felt they would require supervision and only 4% felt they could perform the procedure unsupervised. 56% of respiratory consultants felt that pleural competencies in the internal medicine curriculum should be changed or removed. Common themes in the free text included anxiety surrounding pleural procedures, patient safety concerns, insufficient available training opportunities to maintain competency and differing opinions on the changing role of the medical registrar and who should be expected to perform OOH pleural procedures.

**Conclusions** Our findings suggest that the majority of UK non-respiratory GIM registrars lack both competence and confidence to independently perform OOH pleural procedures, according to both trainees and trainers. This raises concerns for patient safety and service delivery, highlighting the urgent need to review GIM training, curriculum, and OOH pleural procedural provisions.

## P79 PLEURAL PROCEDURES: HOW PREPARED ARE GENERAL MEDICAL AND RESPIRATORY TRAINEES?

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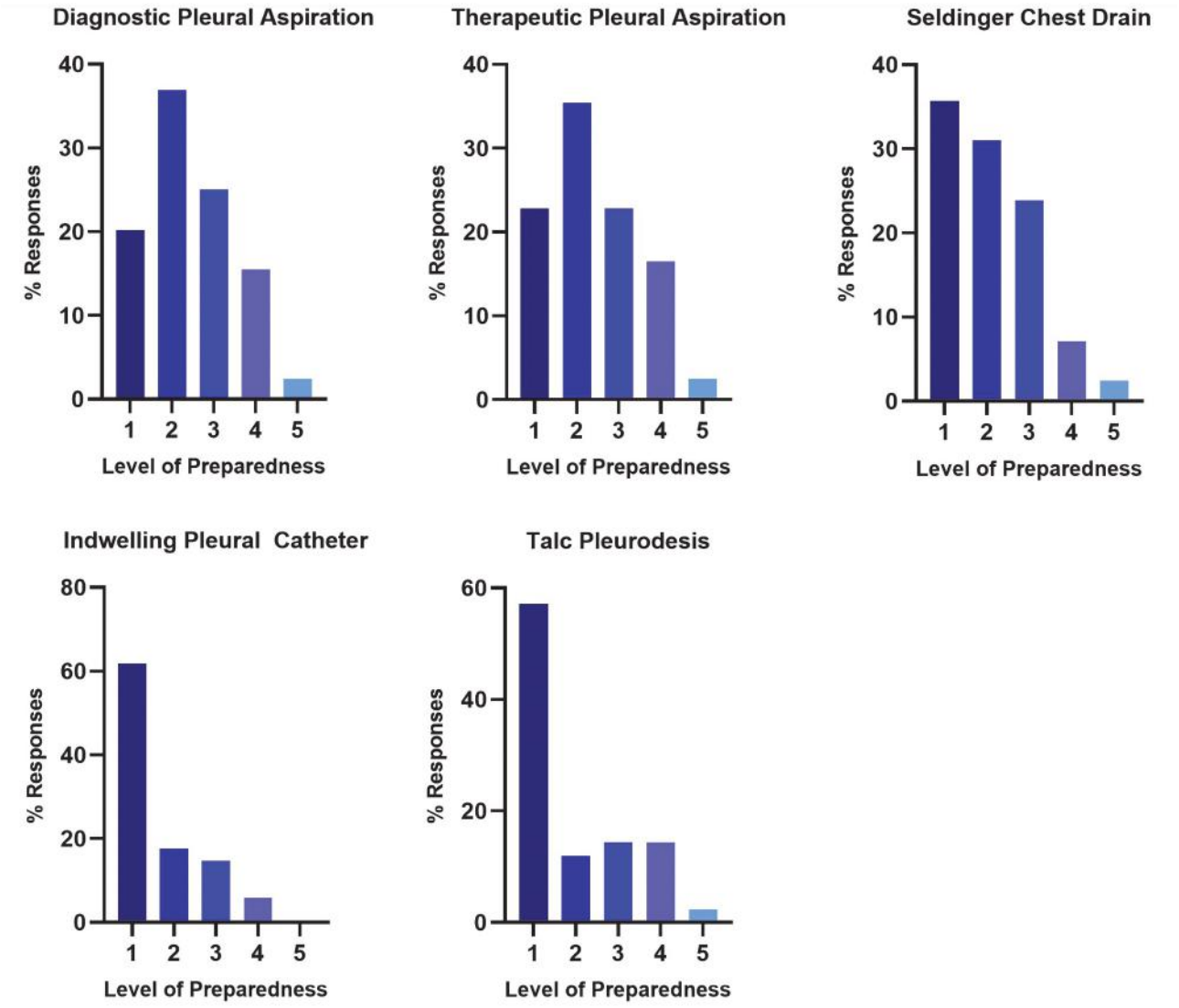
10.1136/thorax-2025-BTSabstracts.261

**Introduction** In some UK hospital Trusts, it is still expected that general internal medicine (GIM) trainees should perform pleural procedures in emergencies. Assessing the preparedness of trainees before performing their first pleural procedure is necessary to evaluate pleural training for GIM trainees.

**Methods** A cross-sectional survey was distributed to GIM and Respiratory trainees across London, focusing on their level of preparedness before performing certain pleural procedures for the first time, with only Respiratory trainees answering questions about talc pleurodesis and indwelling pleural catheter (IPC) insertion. A video of a diagnostic pleural aspiration in a real-world setting was distributed to Internal Medicine Trainees at a district general hospital, who were surveyed before and after distribution of the video to assess their preparedness for performing this procedure.

**Results** 84 responses were received for the pan-London survey. 35.7% (30/84) of trainees felt completely unprepared before performing their first seldinger chest drain, while 61.8% (21/34) and 57.1% (24/42) of respiratory trainees felt completely unprepared before performing their first IPC insertion and talc pleurodesis respectively (figure 1). Access to simulated training varied between the procedures- 75.0% (63/84) of all trainees had simulated a diagnostic pleural aspiration, compared with 22.9% (8/35) and 10.5% (4/38) of respiratory trainees for IPC insertion and talc pleurodesis respectively.

At the district general hospital where the video of the diagnostic pleural aspiration was distributed, the average score on the preparedness scale increased from 2.0/5.0 to 3.3/5.0 (P = 0.04).




**Abstract P79 Figure 1** Preparedness of respondents before doing pleural procedures for the first time (1 = not prepared at all; 5 = fully prepared)

**Conclusions** More pleural training is required early in the GIM training pathway to improve the confidence of resident doctors at performing pleural procedures for the first time. There is lack of access to simulated training for specialist pleural procedures, including IPC insertion, for respiratory trainees. Video resources for practical procedures can be used to increase confidence of trainees before performing the procedure in a real-world setting.

## P80 IMPROVED RECYCLING OF ELECTIVE PLEURAL PROCEDURAL WASTE

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10.1136/thorax-2025-BTSabstracts.262

**Introduction and Objectives** Royal Devon University Healthcare (RDUH) NHS Foundation Trust plans to reduce its carbon footprint by 80% by 2030, achieving net zero by 2040. Our respiratory team aims to help by decreasing the impact of our elective pleural procedural work at the Royal Devon and Exeter Hospital site through improved uptake of recycling. The majority of equipment for pleural procedures is single use, packaged using plastic and paper, with kits including recyclable items that are often unused. We aim to extrapolate the possible improvements across the 13 hospitals within the South West (Severn/Peninsula) Deanery that host respiratory trainees.

**Methods** We monitored weekly elective pleural clinics over 3 months; recording the number of patients, procedures performed, and weight of recycled waste collected. We included patients seen in our elective respiratory procedure space, excluding those seen for procedures in our endoscopy unit (such as those undergoing thoracoscopy). We surveyed respiratory registrars across the South West Deanery as to whether recycling is routinely collected from elective pleural procedures at each hospital site. Information regarding waste management was collected from the RDUH sustainability team.

**Results** We recorded data from 19 elective outpatient pleural clinics, reviewing a total of 95 patients, performing 40

procedures and generating 16.65 Kg recycled waste. On average we saw 5 patients and performed 2 procedures per clinic, with 0.876 kg recycled waste collected per day. Our sustainability team states that recycling is half the financial cost of disposing of clinical waste with a 0.2–1.0 kg CO2-eq/kg waste produced, versus 1.0–3.0 kg CO2-eq/kg waste for incineration. Only 1 other of the 13 hospital sites hosting respiratory trainees in the South West Deanery routinely collects recycling from their elective pleural procedures.

**Conclusions** This project highlights the small but easily achievable gains in environmental and financial impact from facilitating recycling in clinical areas used for pleural procedures. Continuing this work at our site will save up to 490 Kg CO2-eq per year. Given the low rates of similar recycling across the deanery, this would have implications for the potential recycling of up to 1567 kg of waste and saving 4389 Kg CO2-eq per year across this region.

## P81 ''KNOWING THE UNKNOWN'': A SYSTEMATIC REVIEW OF LITERATURE INVESTIGATING THE OPTIMAL MANAGEMENT OF PNEUMOTHORAX AFTER CT-GUIDED LUNG BIOPSY

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10.1136/thorax-2025-BTSabstracts.263

**Introduction** CT-guided lung biopsy is the most important test to diagnose early-stage lung cancer but it is complicated by pneumothorax in 1 in 4 patients. There are no treatment guidelines for the management of pneumothorax after lung biopsy leading to significant variability in clinical practice. We conducted a systematic review of the literature to investigate the optimal management of pneumothorax after lung biopsy.

**Methods** A literature search was conducted in 4 databases (Ovid MEDLINE, EMBASE, Cochrane CENTRAL and Cochrane database of Systematic Reviews) from inception till Dec'24 using a defined search strategy. Published Randomised controlled trials (RCTs) and case series of >200 adult patients undergoing lung biopsy were searched in English language. The main outcome was type of treatment, reason of choosing the treatment option, time to pneumothorax resolution and the length of stay in hospital. All articles were reviewed by two independent reviewers at each stage and data was extracted from eligible studies.

**Results** A total of 1950 articles were retrieved based on the search strategy. 193 articles were eligible for the full-text review and 28 were included comprising 4 RCTs and 24 case series with a total of 10, 818 patients. 2592 patients developed a pneumothorax after lung biopsy with an incidence of 23.96%. Mean age was 59 years and 5267 (48.69%) patients were male. Pneumothorax was drained in 293/2592 (11.3%) patients using a chest drain, 312/2592(12%) patients with a needle aspiration, 111/2592(4.3%) patients requiring needle aspiration or conservative care followed by chest drainage and 568/2592(21.9%) were treated with conservative care alone. The main reason of treatment was size of pneumothorax (24 studies), presence of symptoms (16 studies) and enlarging pneumothorax (9 studies). 6 studies mentioned time to resolution of pneumothorax ranging from 3.7 hours to 9 days. Length of stay in hospital was not recorded.

**Conclusion** This is the first-ever systematic review of the literature to understand the management of pneumothorax after lung biopsy. Size of pneumothorax, despite being less relevant in spontaneous pneumothorax, remains an important factor to guide management and there is a need to develop robust prospective data.

## P82 CHARACTERISTICS OF PATIENTS DEVELOPING IATROGENIC PNEUMOTHORAX DUE TO CT GUIDED LUNG BIOPSY IN A BUSY TERTIARY CARE HOSPITAL (GLENFIELD)

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10.1136/thorax-2025-BTSabstracts.264

**Introduction** CT-guided lung biopsy (CTGB) is a minimally invasive procedure that enables histological diagnosis and aids with personalized patient care. However, this procedure has its own significant risks and complications with pneumothorax being the commonest complication. We present the important characteristics of patients that developed pneumothorax in our tertiary care centre caused as a result of CTGB.

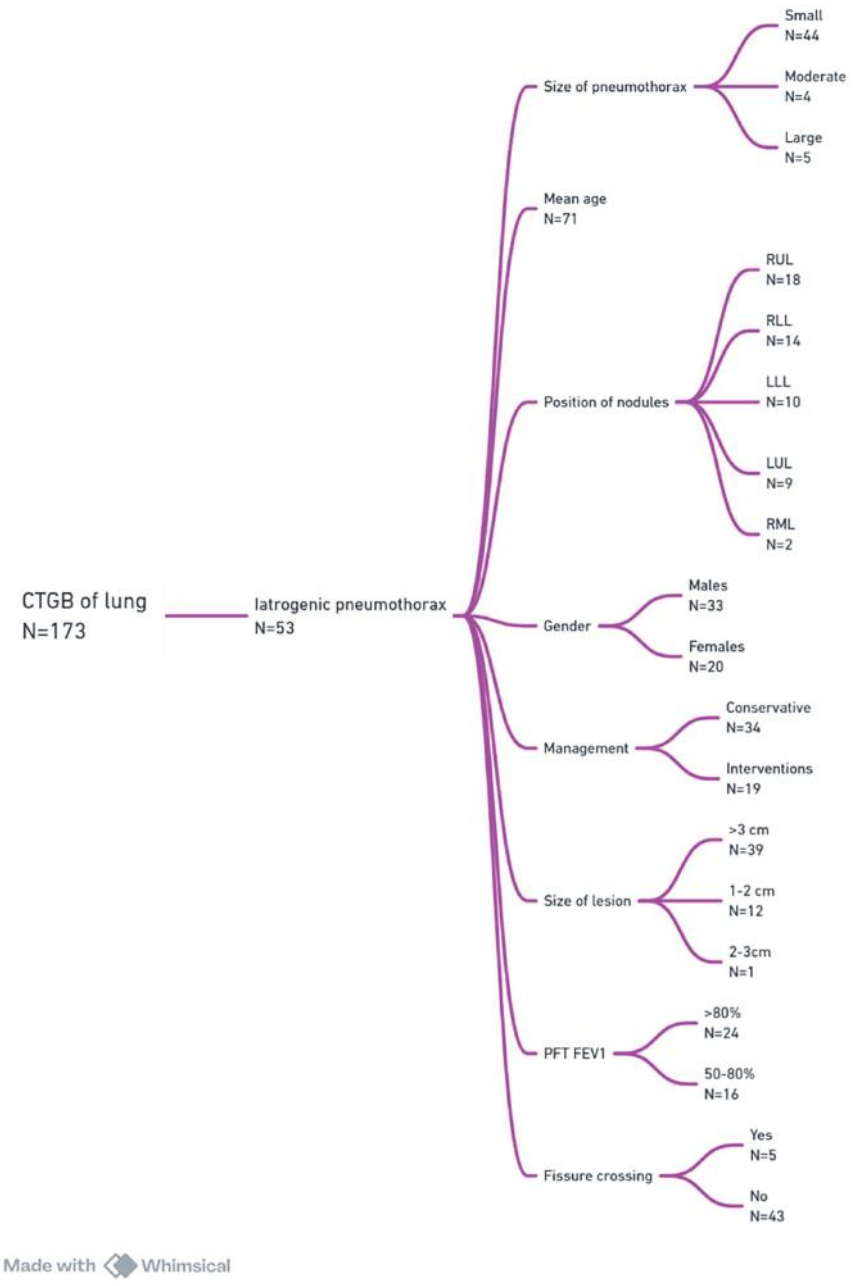
**Methods** A retrospective analysis of all requests for CTGB lung from 1/01/2023 till 31/12/2023 was conducted. This service is provided 5 days a week in our busy tertiary care hospital. Total number of patients undergoing procedure and developing pneumothorax were identified and common characteristics were recorded in patients that developed pneumothorax in detail. The management of the iatrogenic pneumothorax was also studied.

**Results** Of the 173 patients that underwent CTGB, 53 (30.63%) developed iatrogenic pneumothorax. Majority of these patients (n=34, 64.15%) were managed conservatively. However, 19 patients had interventions (35.84%). 44 patients developed small pneumothorax (83.01%), 5 had large (9.43%) with 4 moderate pneumothoraxes (7.54%) as per radiological reports and features.

23 patients who developed pneumothorax had background emphysema (43.39%) whilst 30 did not have emphysema (56.60%). The mean age of patients developing pneumothorax was 71 years. 33(62.26%) of them were male patients and 20 were females (37.73%).

The most common position for the nodules in patients developing pneumothorax was RUL (n=18) followed by RLL (n=14), LLL (N=10), LUL (N=9) and RML (N=2). Majority of lesions were >3 cm (N=39), followed by lesions 1–2 cm (N=12) and there was 1 lesion that was 2–3 cm. In 5 patients the lesions were crossing the fissure whilst it was not crossing in 43 patients. 40 (75.47%) patients with iatrogenic pneumothorax had their lung functions recorded before the CTGB. FEV1 was >80% in 24 patients and between 50–80% in 16 patients.

**Conclusion** Iatrogenic pneumothorax is a well recognised complication of CTGB which can lead to invasive interventions, the rates of invasive interventions being variable in different centres and hospital admissions. We provided an overview of




**Abstract P82 Figure 1**

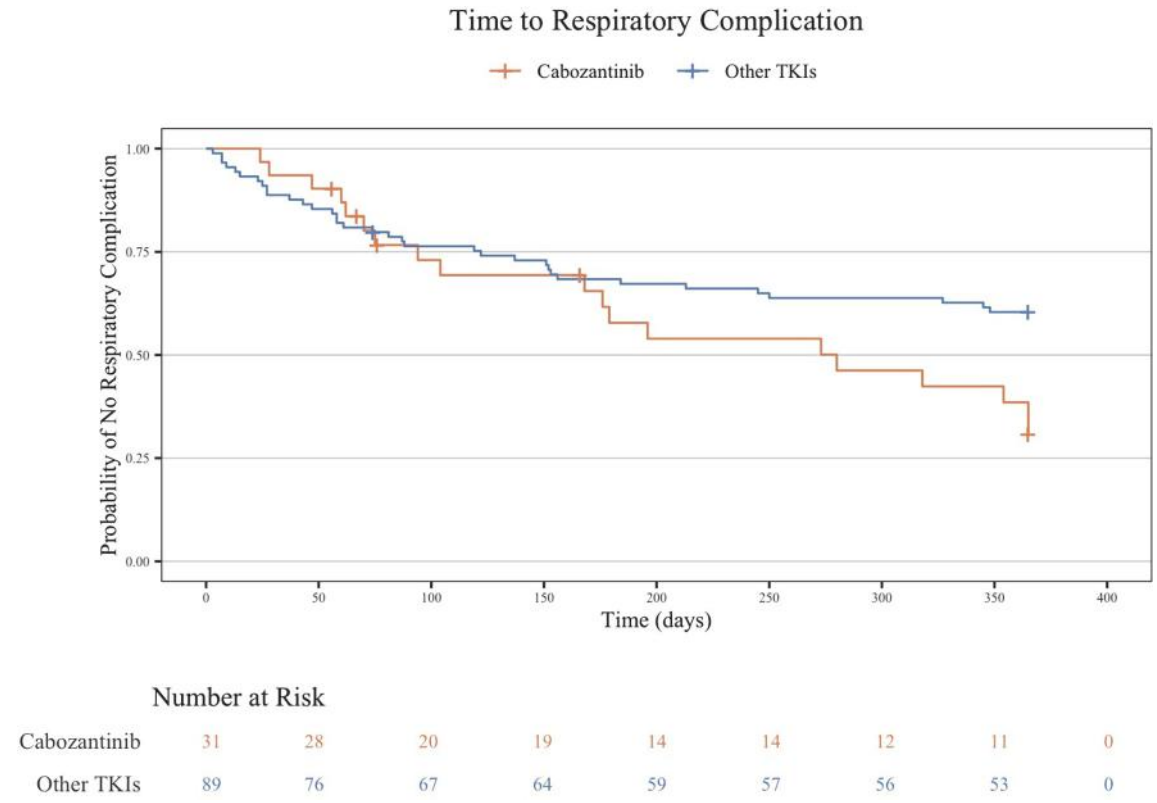
the common important risk factors and characteristics of patients that developed iatrogenic pneumothorax from our cohort.

## P83 RESPIRATORY COMPLICATIONS INCLUDING PNEUMOTHORAX IN PATIENTS RECEIVING TYROSINE KINASE INHIBITORS

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10.1136/thorax-2025-BTSabstracts.265

**Introduction** Tyrosine kinase inhibitors (TKI) are approved for the management of various malignancies. In clinical practice, some patients experience serious respiratory complications from these drugs. However, real world analysis of the adverse respiratory effects of TKIs is lacking. We set out to study clinical features predictive of respiratory complications from TKIs.
**Method** All patients aged 16 and over treated with a TKI for solid tumours between 01/01/2024 and 01/07/2024 at a large tertiary hospital were identified. Electronic patient records were screened for respiratory events that occurred within a year of commencing TKI therapy.
**Results** 120 patients (mean patient age was 65±15 years) treated with one of 11 TKIs were identified. 57% of patients (n=68) had lung metastases. The most frequently prescribed TKIs were cabozantinib (n=31), dabrafenib (n=25), and lenvatinib (n=17). 48% of patients (n=58) reported at least one respiratory complication during the follow-up period. The median time from the initiation of therapy to the development of the first complication was 79 days. The most common complications were new or worsening pleural effusion (n=21) and lower respiratory tract infection (n=19). The risk




**Abstract P83 Figure 1** Kaplan-Meier curve of the probability of no respiratory event occurring within 1 year of follow-up

of pleural effusion was significantly higher in patients who took cabozantinib compared to other TKIs (RR=3.83, 95% CI 1.79–8.20). Less common complications included pulmonary embolism (PE, n=8) and pneumothorax (n=4). Five patients who developed a PE and all patients who developed a pneumothorax had been treated with cabozantinib. For pneumothorax, 3 patients were treated for osteosarcoma. This represents a notable proportion of the 4 patients with osteosarcoma who were included in this study. Overall, the risk of respiratory complications with cabozantinib was high compared to other TKIs (figure 1; RR=1.75, 95% CI 1.25–2.46). The presence of pulmonary metastasis also increased the risk of respiratory complications (RR=1.70, 95% CI 1.11–2.60).

**Conclusion** Our study identified cabozantinib as conferring the greatest risk of developing TKI-associated respiratory complications including pneumothorax. A follow-on national study is planned.

## P84 WEATHER CHANGES AND THE INCIDENCE OF SPONTANEOUS PNEUMOTHORAX: AN OBSERVATIONAL STUDY FROM A SINGLE UK CENTRE

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10.1136/thorax-2025-BTSabstracts.266

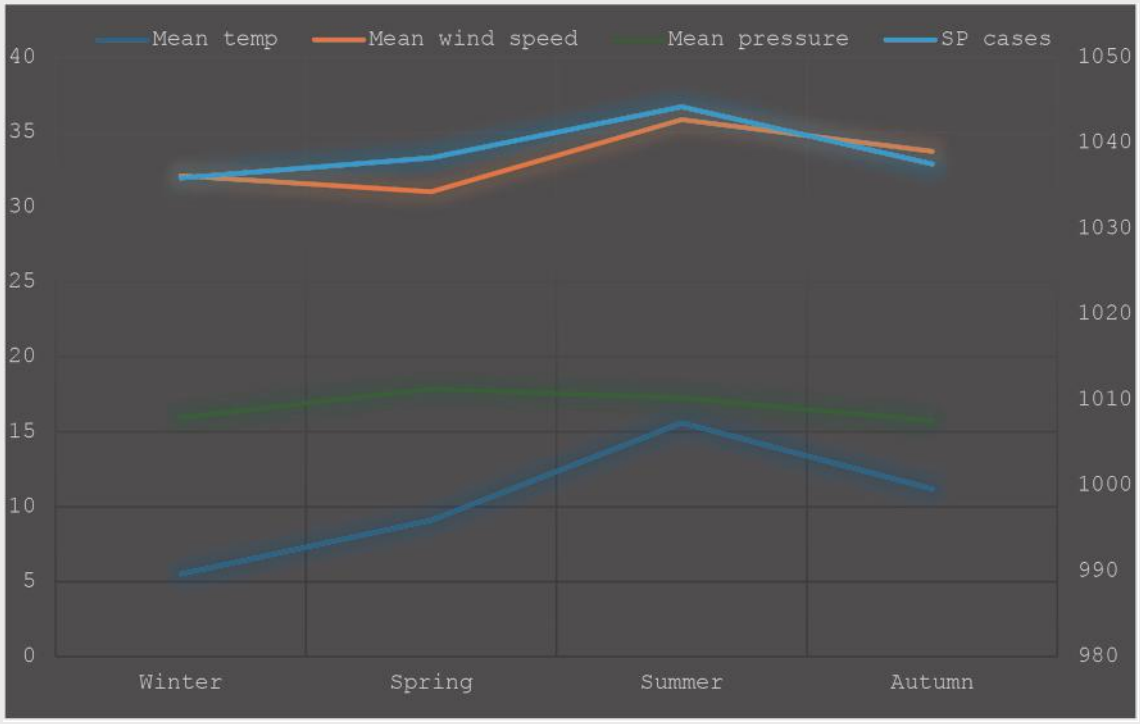
**Introduction and Objectives** Daily fluctuations in weather parameters have been suggested as possible triggers for spontaneous pneumothorax (SP).[1 2] However, there is no strong evidence to confirm this hypothesis. This study was designed to assess the correlation between daily local weather parameters and occurrence of SP.

**Methods** Retrospective observational study of 119 (Males=88, Females=31, mean age= 51.2 years) cases of primary and secondary spontaneous pneumothorax from 1st January 2020 to 31st December 2024, was conducted covering a specific geographical area. Weather data including daily values for mean, maximum, and minimum temperature, rainfall, wind speed, and atmospheric pressure, were obtained from open-meteo API, open access platform which provides historical information regarding local climate changes. Weather parameters on the days when SP were presented were compared with no SP presented, using independent sample t-tests. Seasonal variation was assessed using chi-square analysis. Logistic regression analysis performed to assess correlation between different weather parameters on a day (D-1) prior to the date of presentation and two days prior (D-2) to the date of presentation.

**Results** There were no statistically significant differences were found between SP and non-SP days for any of the weather parameters, including mean temperature (p=0.06), maximum temperature (p=0.13), minimum temperature (p=0.10), rainfall (p=0.89), wind speed (p=0.33), atmospheric pressure (p=0.47) or pressure range (p=0.64). Seasonal distribution of SP also showed no significant variation ($\chi^2$ p=0.85). There was no association found with any of the weather parameters on D-1 or D-2 (all p-values >0.05).

**Conclusion** There was no significant correlation was found between spontaneous pneumothorax and weather parameters. Limitations include the single-centre design and limited

**Abstract P84 Table 1** Seasonal variation of presentation of spontaneous pneumothorax and weather parameters (2020–2024)




number of subjects. Daily weather data may not reflect micro-climatic data of the locality which may or may not related to occurrence of spontaneous pneumothorax.

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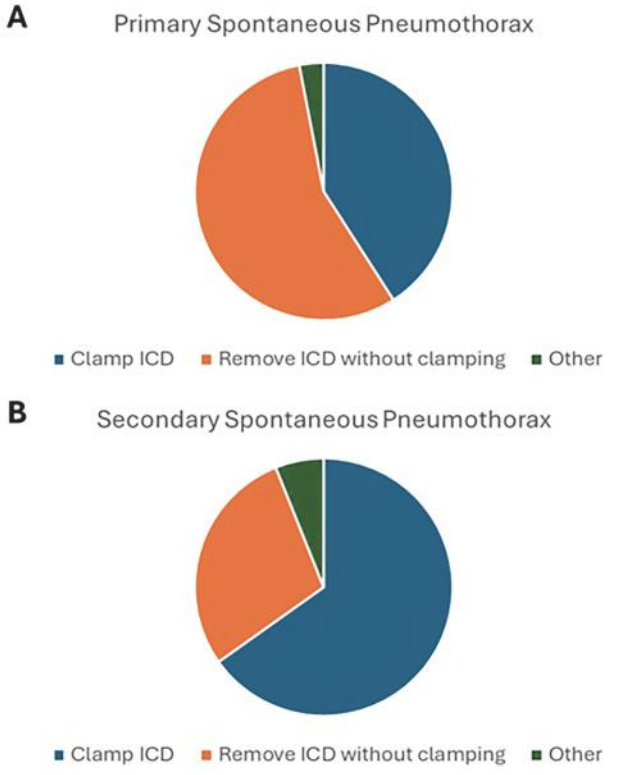
## P85 CLAMP OR REMOVE? A NATIONAL SURVEY OF INTERCOSTAL CHEST DRAIN MANAGEMENT IN SPONTANEOUS PNEUMOTHORAX

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10.1136/thorax-2025-BTSabstracts.267

**Introduction and Objectives** Recurrence of pneumothorax after intercostal chest drain (ICD) removal is a common complication, associated with the need for additional pleural procedures. In a large retrospective study, ICD clamping was not shown to be associated with a reduced risk of recurrence.[1] There is a need to determine whether ongoing variation in practice exists and whether a prospective study of this topic is feasible.

**Methods** A survey was distributed via UK-based clinical and research networks between May and June 2025. Questions focused on preferred management strategies for two hypothetical clinical scenarios: a primary spontaneous pneumothorax (PSP) and a secondary spontaneous pneumothorax (SSP), both after cessation of air leak. Questions also assessed the use of air leak monitoring devices and clinician interest in participating in a prospective trial.




**Abstract P85 Figure 1** Clinician approaches to intercostal chest drain removal. **(A, B)** Pie charts illustrating the distribution of clinician-reported approaches to ICD removal in **(A)** primary spontaneous pneumothorax and **(B)** secondary spontaneous pneumothorax scenarios (n=66)

**Results** 66 responses were received, of which 97% (n=64) were working in respiratory medicine, predominantly consultant physicians (41%, n=27) and specialist registrars (53%, n=35). Significant variation in practice was reported. For the PSP scenario, clinician preference was in favour of direct ICD removal without clamping compared with clamping (56% vs 41%) (figure 1A). For SSP, the converse was true with a preference for ICD clamping (65% vs 29%) (figure 1B). Use of air leak monitoring devices was inconsistent, with respondents split between using them routinely (17%, n=11), sometimes (33%, n=22), and never (50%, n=33). A majority of clinicians would prioritise reduction in pneumothorax recurrence over a reduced length of stay (92% vs 8%). There was enthusiasm for future research with 86% of respondents (n=57) agreeing a need to develop strategies focused on reducing early recurrence, and 97% (n=64) confirming interest in participating in a prospective trial.

**Conclusions** Variation in ICD removal practice persists across the UK, with management strategies differing between primary and secondary pneumothorax. This inconsistency in practice and overwhelming support for a future study strongly justify the need for a prospective randomised controlled trial to establish an evidence-based, standardised approach.

## REFERENCE

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## P86 THE ACCURACY OF ICD-10 DIAGNOSTIC CODES FOR THE IDENTIFICATION OF PNEUMOTHORAX AND PULMONARY EMBOLISM: A RETROSPECTIVE STUDY IN TWO UK CENTRES

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10.1136/thorax-2025-BTSabstracts.268

**Background** Trainee-led UK research collaboratives often use ICD-10 codes for their patient identification, assuming sufficient diagnostic accuracy. For effective patient identification, codes must have a reasonable positive predictive value (PPV) to minimise unnecessary chart reviews of patients without the diagnosis (i.e. false positives), and adequate sensitivity to capture a meaningful proportion of those with the diagnosis. Using local data which contributed to the INSPIRE collaborative's ERUPT and CLAMP studies, we aimed to assess the PPV of ICD-10 codes for pulmonary embolism (PE) and pneumothorax.

**Methods** We identified patients with relevant diagnostic codes at two UK hospitals over defined timeframes, for 1) PE (I26.0, I26.9), 2) PE with cor pulmonale (I26.0), and 3) Pneumothorax (J93.0, J93.1, J93.8, J93.9). There were no exclusion criteria other than eligibility time period. The PE cohort

**Abstract P86 Table 1** Positive predictive values of diagnostic codes

| Condition and disease-specific categories | With diagnostic code | Confirmed diagnosis | Not confirmed | Potentially miscoded conditions |
|---|---|---|---|---|
| ***Pneumothorax (J93.0, J93.1, J93.8, J93.9)*** *PPV: 80% (132/165)* | 165 | 132 *PSP: 21 SSP: 57 Traumatic: 12 Iatrogenic: 42* | 33 | Pleural effusions, CAP, T2RF, colonic perforation |
| ***PE with or without cor pulmonale (code: I26.0, I26.9)*** *PPV: 85.3% (515/604)* | 604 | 515 | 89 | HFpEF, pleural effusion, pulmonary oedema, LRTI, DVT, progression of malignancy, PAH, AF, HAP, COVID, IECOPD, sepsis |
| Unprovoked | 243 | 236 | 7 | |
| Provoked | 217 | 215 | 2 | |
| Unknown | 74 | 64 | 10 | |
| ***PE with cor pulmonale only (I26.0)*** *PPV: 93.4% (155/166)* | 166 | 155 | 11 | Cor pulmonale secondary to COPD, chronic PAH |
| Clinical exam only | 9 | 8 | 1 | |
| Documented ECG findings only (RAD, RVH) | 3 | 3 | 0 | |
| Radiological investigations only (CTPA or ECHO) | 129 | 125 | 4 | |
| Clinical exam + ECG | 0 | 0 | 0 | |
| Clinical exam + radiology | 11 | 7 | 4 | |
| ECG + radiology | 12 | 11 | 1 | |
| Clinical exam + ECG + radiology | 2 | 1 | 1 | |

(RAD: right axis deviation, RVH: right ventricular hypertrophy, PSP: primary spontaneous pneumothorax, SSP: secondary spontaneous pneumothorax, PE: pulmonary embolism, ECG: electrocardiogram, CTPA: Computed Tomography Pulmonary Angiogram, ECHO: echocardiogram, COPD: chronic obstructive pulmonary disease, PAH: pulmonary artery hypertension, HFpEF: heart failure with preserved ejection fraction, LRTI: lower respiratory tract infection, CAP: community acquired pneumonia, T2RF: type 2 respiratory failure, DVT: deep vein thrombosis, AF: atrial fibrillation, HAP: hospital acquitted pneumonia, IECOPD: infective exacerbation of COPD)

spanned a 12-month period, and the pneumothorax cohort 30 months. Two clinicians extracted demographics and disease-specific variables, using a standardised template, blinded to the other's assessments. We collected patient demographics (age, sex, index of multiple deprivation of home postcode), diagnoses, and disease-specific aspects (PE: provoked/unprovoked, malignancy, deep vein thrombosis. Pneumothorax: type, respiratory disease, smoking history, malignancy, pleural procedures). Conflicts were resolved by consensus.

**Results** We reviewed 165 pneumothorax-coded and 604 PE-coded patients. There was a male predominance in pneumothorax group (pneumothorax: 36.4% female; PE: 53%), with similar ages (pneumothorax: mean 66 (SD 20); PE: 71 (SD = 17)). PPVs were: pneumothorax 80% (132/165), PE 85.3% (515/604), and PE with cor pulmonale 93.4% (155/166). Inter-reviewer reliability for decisions regarding diagnostic confirmation was generally high (Cohen's kappa coefficients (κ): PE = 1, Presence of cor pulmonale = 0.53).

**Conclusion** ICD-10 codes for PE and pneumothorax showed moderate-high PPVs, supporting their use if accompanied by diagnostic confirmation. We recommend collaborative studies using ICD-10 codes should examine and report PPVs given the minimal associated workload and importance in establishing their validity.

## P87 SURGICALLY MANAGED PNEUMOTHORAX: A 5 YEAR INSTITUTIONAL APPRAISAL OF HISTOPATHOLOGICAL FINDINGS AND THEIR CLINICAL IMPLICATIONS

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10.1136/thorax-2025-BTSabstracts.269

**Background** Surgically managed pneumothorax cases are at increased risk of underlying lung pathologies, with 10% of patients believed to have a genetic predisposition. Histopathological features of pneumothorax lung tissue can be divided into non-specific and specific findings, with the latter suggestive of a pre-existing lung condition. Microscopic evaluation plays a crucial role in identifying clinically unsuspected pathogenesis which may require follow-up.

**Aims and Objectives** To evaluate the institutional incidence and indications of surgically treated pneumothorax cases, as well as incidental histological findings of pulmonary pathologies in this cohort.

**Methods** A 'CORVU' search of the Laboratory Information Management System at the Department of Pathology from was performed from January 2019 to December 2024, utilizing SNOMED codes for Pneumothorax and Bulla. Study data was collected and analysed utilising Microsoft Excel.

**Results** 7%(n=89) of the 1302 surgical lung specimens retrieved over the study period were surgically treated pneumothorax cases, with an age range of 14–69, and median age of 30. The male: female ratio was 3:1. Recurrent pneumothorax was the most common indication for surgery in 52% (n=46). 89%(n=80) of cases were spontaneous, 11%(n=9) were secondary. 25%(n=20) of spontaneous cases had emphysematous change, of which 55%(n=11) were <40 years old. Of these, 45%(n=5) were smokers. All secondary cases had emphysematous change. Nonspecific histological features in the spontaneous cohort were predominantly pleuritis (75%, n=60), bullae and fibrosis (73%, n=58). Of the spontaneous cases, 20 were smokers, 11 were ex-smokers, 10 were non-smokers, the remainder had unknown smoking status. Assessment of the nonsmoking group revealed 50%(n=5) had abnormal airspace enlargement.

Atypical adenomatous hyperplasia (AAH) was identified in 10%(n=8) of spontaneous cases, of which 75%(n=6) were <40 years old. 50% were smokers, 50% had unknown smoking status, however those with unknown smoking status had microscopic smoking-related changes of respiratory bronchiolitis (n=3) and emphysematous change (n=2). One of these AAH cases was also diagnosed with Langerhans cell histiocytosis. No malignancies were identified.

**Conclusion** Histopathological examination of lung tissue can yield important incidental findings in surgically treated pneumothorax patients, which require appropriate follow-up. Clinical outcomes and follow-up data for this patient cohort is currently being analysed.

# Insights and advances in ILD care

## P88 DOES ROUTINE LIVER TEST MONITORING FOR PATIENTS ESTABLISHED ON NINTEDANIB ADD CLINICAL VALUE? – EXPERIENCE OF AN UK TERTIARY ILD CENTRE

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10.1136/thorax-2025-BTSabstracts.270

**Background and Aims** Our tertiary ILD centre routinely monitors liver function tests (LFTs) 3 monthly for patients established on Nintedanib. However, beyond the initial 3 months the requirement for monitoring is 'periodically thereafter or as clinically indicated'.[1] We aimed to identify the prevalence of clinically relevant abnormal LFTs identified on routine scheduled testing, and the value of routine monitoring (cost £4.34 per LFT).

**Methods** We retrospectively reviewed all local patients who initiated Nintedanib for idiopathic pulmonary fibrosis (IPF) or progressive pulmonary fibrosis (PFILD) at our ILD centre between February 2014 and October 2024. We reviewed all LFT results taken after 3 months of initiating medication, categorising tests into routine or acute, identified all abnormal results, their circumstances and outcomes. Fisher's exact test was used for group comparisons.

**Results** 404 patients initiated Nintedanib (313 IPF, 91 PFILD), with a mean 543 days on treatment (range 1 - 4,901 days). The cumulative exposure to Nintedanib was 175,124 patient-days, requiring 1,918 routine tests.

Routine testing after 3 months identified 41 patients with abnormal LFTs (1 abnormal result every 46.8 tests), of which 36(88%) were <3x upper limit of normal (ULN), with none >5x ULN (table 1). 30(73%) had asymptomatic rises in ALT, 29 requiring no action, and one patient requiring a treatment break then restarted medication (this test was likely haemolysed). 11 patients had abnormal ALTs associated with gastrointestinal symptoms or new medications, of which 7(64%) patients required treatment breaks or stopped treatment. Overall, we had 1 abnormal routine test resulting in changed management every 239.8 tests (cost per clinically relevant abnormal test £1040.52).

**Abstract P88 Table 1** Abnormal routine ALT test results

| | Asymptomatic (n=30) | Other associations (n=11) | p-value |
|---|---|---|---|
| **ALT ULN** | | | |
| <3x | 29(96.7%) | 7(63.6%) | **0.014** |
| 3-5x | 1(3.3%) | 4(36.4%) | |
| >5x | 0 | 0 | |
| **Other Associations** | | | |
| Gastrointestinal symptoms | | 6(54.5%) | |
| Medication changes | | 3(27.3%) | |
| Other* | | 2(18.2%) | |
| **Outcomes** | | | |
| No dose change | 29(96.7%) | 4(36.4%) | **<0.001** |
| Break then restarted treatment | 1(3.3%) | 3(27.3%) | 0.052 |
| Permanent stop | 0 | 4(36.4%) | 0.211 |

**Extensive bruising following fall, heavy alcohol use for short period, p<0.05 significant*

A further 26 patients had abnormal LFTs on acute testing outside of routine schedules either related or unrelated to Nintedanib treatment, necessitating treatment breaks or stopping treatment in just 4(15%) patients.

**Discussion** Routine LFT monitoring after 3 months rarely identified clinically relevant rises in ALT and came at a significant time and monetary cost for our ILD service. We are developing an optional patient-initiated follow up (PIFU) pathway for monitoring LFTs in patients who are stable on treatment.

**REFERENCE**

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## P89 A 'CALL TO ACTION' IN DETERMINING AND ESTIMATING A MINIMUM IMPORTANT DIFFERENCE (MID) IN 6-MINUTE WALKING DISTANCE (6MWD) IN PH-ILD

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10.1136/thorax-2025-BTSabstracts.271

**Objectives** This study aimed to determine an minimally important difference (MID) in 6MWD for pulmonary hypertension associated with interstitial lung disease (PH-ILD) using clinical trial data (INCREASE NCT02630316), using different analytical methods, and structured expert elicitation (SEE).

**Methods** A distributional approach was needed to be used to determine the MID. Distributional approaches commonly involve a fraction of the standard deviation (SD), typically 0.5SD. The 0.5SD can either be defined using the baseline SD or the SD of the change from baseline, also known as the effect size (ES) and standardised response mean (SRM), respectively. The trial comprises of both a treatment (Treprostinil, TRT) and placebo (PLB) arm. The TRT arm was selected for the MID using the SRM, $0.5SD_{change}$ with a change from baseline at 16 weeks. A second analysis was undertaken focusing on all patients whose 6MWD improved over 16 weeks, irrespective of the study arm. The study also determined clinical expert MCID values through SEE using STEER and SHELF methods.

**Results** In the trial the SD for placebo was thirty-percent greater than for the TRT arm suggesting a greater degree of uncertainty associated with placebo relative to treatment. The MID estimate derived using the first analysis was 25.8 m for TRT. The MID also corresponds to the 10% improvement from baseline. The second approach demonstrated a consistent score of 19 m as indicative of a MID. The SEE methodology provided a MCID of 31 m however, when an expert response was excluded based on task comprehension concerns

and lack of PH experience, an MCID of 28.6 m was established.

**Conclusion** This study has established an interim 6MWD MID for PH-ILD that ranges between 19 m to 31 m. Due to the variability of the MID for PH-ILD, this study is a 'call-to-action' for more research to be conducted.

## P90 SEEING WHAT'S IMPORTANT: ELICITING EXPERT JUDGEMENTS TO ESTIMATE THE MINIMALLY CLINICALLY IMPORTANT DIFFERENCE FOR SIX-MINUTE WALKING DISTANCE IN PH-ILD'

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10.1136/thorax-2025-BTSabstracts.272

**Objectives** A challenge of examining patient outcomes in clinical research is determining if the observed differences represent a statistically significant change and, if so, whether this constitutes a clinically meaningful change for patients. There is currently no estimate for minimally clinically important difference (MCID) for the six-minute walking distance (6MWD) for populations with pulmonary hypertension associated with interstitial lung disease (PH-ILD). This study sought to employ a novel approach to MCID estimation, employing validated techniques to elicit clinical experts' judgement.

**Methods** The study was piloted with two clinical experts, and five clinical experts from the UK and Germany with experience in PH-ILD or ILD participated in the workshop. They were asked to construct a histogram using the 'chips and bins 'method. The experts were asked to complete two MCID elicitation exercises. The MCID tasks were interspersed with a facilitated group discussion to examine between-expert variability.

**Results** The first MCID estimates ranged from 17.5 m (SD = 1.4 m) to 47.1 m (SD = 11.8 m) with a pooled distribution of 31.9 m (SD = 12.4). The MCID pooled distribution for the second elicitation was 31 m (SD = 8.7 m). The individual responses ranged from 24.8 m (SD = 2.0 m) to 40.6 m (SD = 8.7 m). One expert expressed they were uncertain in their estimate being valid explained by (1) their experience with only ILD patients and (2) the observed variability in patient performance on the 6MWD test. The expert made contradictory comments, indicating they did not fully understand the task. If this expert's response is excluded, the MCID would be 28.6 m (SD 6.9).

**Conclusion** According to the study, the MCID lies between 24.8 m to 40.6 m. All experts discussed uncertainty due to the severity of PH was unspecified which could explain the range provided by the experts.

## P91 REAL-WORLD HEALTHCARE RESOURCE UTILIZATION IN PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS INITIATING ANTIFIBROTIC THERAPY IN THE MEDICARE FEE-FOR-SERVICE POPULATION

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10.1136/thorax-2025-BTSabstracts.273

**Objectives** To characterize treatment patterns and healthcare resource utilization in patients with idiopathic pulmonary fibrosis initiating antifibrotic therapy.

**Methods** A retrospective cohort study was conducted using the 100% Medicare Fee-For-Service data to identify patients who initiated antifibrotic therapy (nintendanib or pirfenidone) between 01/01/17 and 12/31/22. Eligible patients were aged ≥18, had ≥1 inpatient or ≥2 outpatient IPF diagnosis claims pre-index, continuously enrolled for 6-months prior and had no prior antifibrotic therapy or lung transplant. Discontinuation, using a 45-day gap definition, per-person per-month (PPPM) healthcare resource utilization and mortality were examined. Patients were censored at the earliest occurrence of switching to the other antifibrotic, lung transplant, death, end of enrollment, or end of data. Unadjusted time-to-event was estimated using Kaplan-Meier Cox-proportional hazards model identified characteristics associated with discontinuation.

**Results** 5,397 patients met selection criteria, 3,555 initiating nintendanib and/or 2,496 initiating pirfenidone. For all patients, mean (SD) age at initiation was 75.6(7.3), with 38% female, 87% white race and mean (SD) Deyo Charlson comorbidity index score of 2.6(2.4). 25% of patients discontinued antifibrotic therapy at 3 months and 50% at 11 months. Mean (SD) PPPM inpatient hospitalizations increased from 0.07(0.13) in the pre-index to 0.10(0.26) in the post-index. 25% and 50% of patients died at 18 and 41 months after initiation, respectively. Patients that were aged ≥75 were more likely to discontinue (HR 1.23; 95%CI: 1.14–1.33) therapy relative to those aged <75. Men (HR:0.76; 95%CI:0.71–0.81) and patients that experienced a hospitalization prior to initiation (HR:0.85; 95%CI: 0.78–0.94) were less likely to discontinue therapy, after adjustment.

**Conclusions** Patients with IPF have high treatment discontinuation to antifibrotic therapy, hospitalizations and mortality. A large unmet need remains in patients with idiopathic pulmonary fibrosis.

## P92 A NOVEL CT-CHEST SCORING SYSTEM TO PREDICT NODAL SARCOIDOSIS IN PATIENTS WITH ISOLATED HILAR AND MEDIASTINAL LYMPHADENOPATHY

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10.1136/thorax-2025-BTSabstracts.274

**Introduction and Objectives** Isolated hilar and mediastinal lymphadenopathy (IHML) on Computed Tomography (CT) chest is a frequent incidental finding requiring further evaluation. While benign causes such as sarcoidosis and reactive lymphadenopathy are among the common differentials, concerns remain regarding more serious pathologies such as lymphoma, occult nodal metastases and tuberculosis (TB). Nodal sampling is performed using endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) to confirm the diagnosis. We aimed to assess the feasibility of a novel CT-based scoring system based on defined radiological features to predict the likelihood of sarcoidosis in IHML.

**Methods** We conducted a retrospective analysis of 81 patients who underwent EBUS-TBNA for IHML in 2022. A thoracic radiologist, blinded to histopathology results, reviewed CT scans for the following features: nodal distribution (2 points

for typical distribution and 1 for atypical), nodal density (1 for high, 0 for normal), peri-lymphatic lung nodules (1 if present, 0 if absent), and features suggestive of other pathologies such as infection or malignancy (0 if present, 1 if absent). A total score of 1 was considered low, and 5 high probability for sarcoidosis. Scores were compared against nodal histology as a form of preliminary validation.

**Results** Of the 81 patients, mean age was 64 years (range 22–90), males 50 (61.7%). 6 patients had a score of 5, of which 4 had confirmed granulomatous lymphadenitis on nodal histology, one was diagnosed with sarcoidosis on wedge lung biopsy after inconclusive EBUS-TBNA, and one patient was treated as sarcoidosis after a multidisciplinary review. 22 patients had a score of 4, of which 20 had granulomatous lymphadenitis. 21 patients scored 3, of which only 3 had granulomatous lymphadenitis, with one treated as sarcoidosis and one diagnosed with TB. In 32 patients with scores of 1 and 2, only 3 had granulomatous lymphadenitis, of which one was confirmed TB. None of the patients had malignancy.

**Conclusions** Our study supports a novel CT scoring system to predict pre-test probability of sarcoidosis in IHML, with preliminary validation in a retrospective cohort. Further prospective studies are required to validate its clinical utility, along with interobserver variability assessment between radiologists.

## P93 CONCOMITANT PRESENCE OF ALLERGIC RHINITIS (AR) IN CHRONIC/FIBROTIC HYPERSENSITIVITY PNEUMONITIS (C/F-HP) PATIENTS: A SIGNIFICANT CORRELATION!

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10.1136/thorax-2025-BTSabstracts.275

**Background** Chronic/Fibrotic Hypersensitivity Pneumonitis (C/F-HP), inspite of its high prevalence, is often underdiagnosed due to the complexity of its diagnostic process, which typically involves lung biopsies. These invasive procedures carry significant risks, especially for patients who are severely ill. This study explores the potential of Allergic Rhinitis as a non-invasive clinical indicator for diagnosing C/F-HP, aiming to simplify the diagnostic approach and reduce the need for biopsies.

**Objective** The objective of this study was to assess the occurrence of AR in patients with C/F-HP and evaluate its utility as a clinical marker for non-invasive diagnosis.

**Method** This cross-sectional descriptive study was conducted at BAM&DC Multan between July 2021 and December 2022. A total of 107 patients diagnosed with C/F-HP, aged 18 to 60, were enrolled using a consecutive sampling technique. The exclusion criteria included patients with known causes of Interstitial Lung Disease and comorbid conditions. Allergic Rhinitis was diagnosed based on clinical history and serum IgE levels, following AAFP guidelines.

**Results** The study comprised 107 C/F-HP patients, with a mean age of 46.73 ± 9.36 years. Male patients accounted for 59.8% of the cohort. AR was diagnosed in 99 out of 107 patients (92.5%), with an average serum IgE level of 542.65 ± 182.93. A small percentage (3.7%) of AR patients had normal IgE levels, possibly due to prior corticosteroid use. Among AR-positive patients, males outnumbered females (62:37), reflecting a similar ratio in the overall C/F-HP population.

**Conclusion** This study highlights a significant correlation between AR and C/F-HP, suggesting that AR could serve as a reliable clinical indicator for C/F-HP diagnosis, potentially reducing the need for invasive lung biopsies. *It seems that our Hypothesis 'in an uncertain ILD patient, if AR is*

**Abstract P93 Table 1** Demographic characteristics of patients with chronic hypersensitivity pneumonitis (N=107)

| | | |
|---|---|---|
| Age (year), | Mean | ± SD |
| | 46.73 | ± 9.369 |
| Gender | N | % |
| Male | 64 | 59.8% |
| Female | 43 | 40.2% |
| Occupation | N | % |
| Paint factory | 21 | 19.6% |
| Pigeon breeder | 55 | 51.4% |
| Power loom | 31 | 29.0% |
| Serum IgE level (IU/ml) mean ± SD | Mean | ± SD |
| | 542.65 | ± 182.930 |
| Allergic Rhinitis | N | % |
| Yes | 99 | 92.5% |
| No | 08 | 7.5% |

*concomitantly present, C/F-HP should be sought out', stands validated.* Further research is recommended to solidify these findings.

## P94 DEVELOPMENT AND VALIDATION OF THE HYPERSENSITIVITY PNEUMONITIS SOUTH ASIAN QUESTIONNAIRE FOR EXPOSURE (HP-SAQE)

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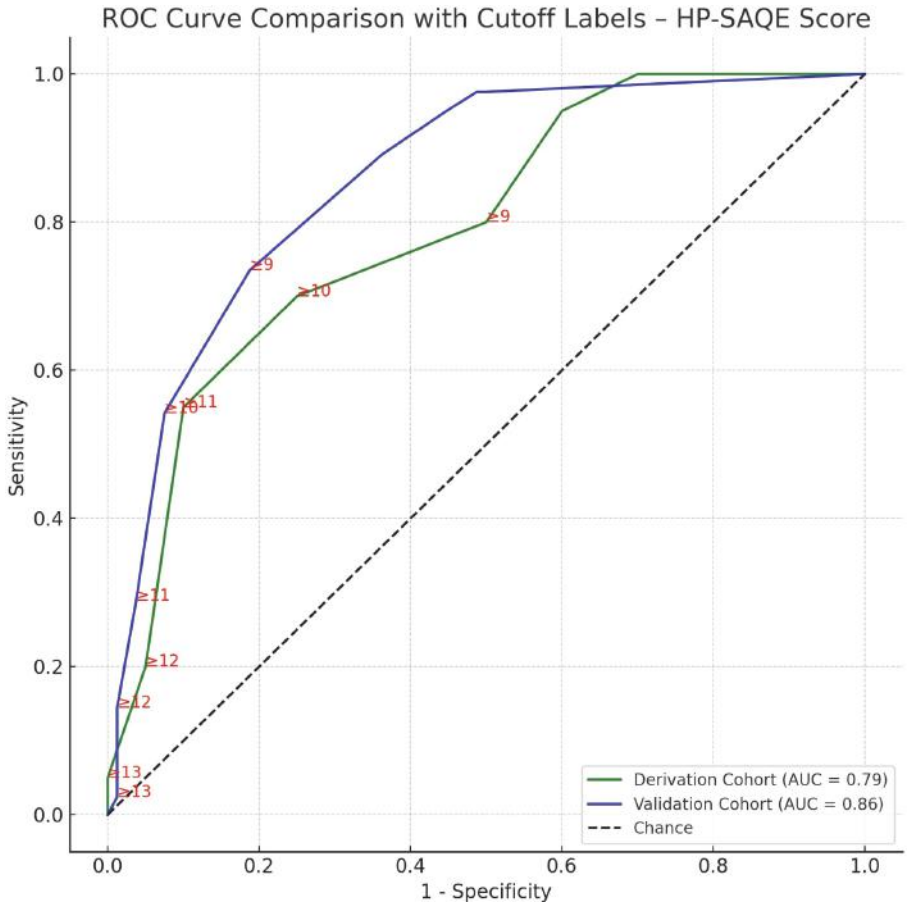
10.1136/thorax-2025-BTSabstracts.276

**Introduction** There is an unmet need for a validated qualitative and quantitative tool to assess environmental exposures in patients with interstitial lung disease (ILD), particularly hypersensitivity pneumonitis (HP). This study aimed to develop and validate a comprehensive tool to systematically identify exposures in ILD patients across South Asia.

**Methods** A systematic literature review identified exposures associated with ≥5 documented HP cases, which were evaluated through a two-round Delphi survey involving 39 pulmonologists from India, Sri Lanka, Pakistan, and Nepal. The preliminary questionnaire was pilot-tested (N=30) to ensure clarity and usability. Following refinement, the final questionnaire was prospectively validated in 202 ILD patients across six centres in India and Sri Lanka. Each patient underwent centralized, protocolized online multidisciplinary discussion. The questionnaire had a qualitative part and a quantitative score ranging from 0 to 14.

**Results** The systematic review screened 5,170 records and included 407 studies, identifying 24 exposure categories. A 50-item Delphi survey achieved expert consensus (>70%) on 17 qualitative and 4 quantitative items. The questionnaire was translated into Hindi, Tamil, and Sinhala. In the derivation cohort (n=40), the HP-SAQE score was significantly higher in HP vs. non-HP patients (median: 11.0 vs. 8.5; p=0.0015). This finding was validated in a separate cohort (n=162; median: 10.0 vs. 2.5; p<0.001). ROC analysis showed good diagnostic performance (AUC: 0.79 derivation; 0.86 validation). In the derivation cohort, the exposure score demonstrated an AUC of 0.79, with a cutoff of ≥9 yielding 80% sensitivity, 50% specificity (Youden's Index 0.30). A higher cutoff of ≥11 improved specificity to 90% with a moderate sensitivity of 55% (Youden's Index 0.45). In the validation cohort, the AUC was 0.88, indicating excellent discriminative performance. A cutoff of ≥9 provided the optimal balance with 73% sensitivity, 81% specificity (Youden's Index 0.54).

**Conclusion** The HP-SAQE is the first regionally adapted, qualitative and quantitative exposure assessment tool for ILD patients, developed through systematic review, Delphi consensus, and multicentre prospective validation. Its high specificity




**Abstract P94 Figure 1**

and consistent centre-wise performance support its role in structured exposure assessment, particularly in resource-limited or high-burden settings. Lower score cutoffs may enhance its utility as a clinical screening tool.

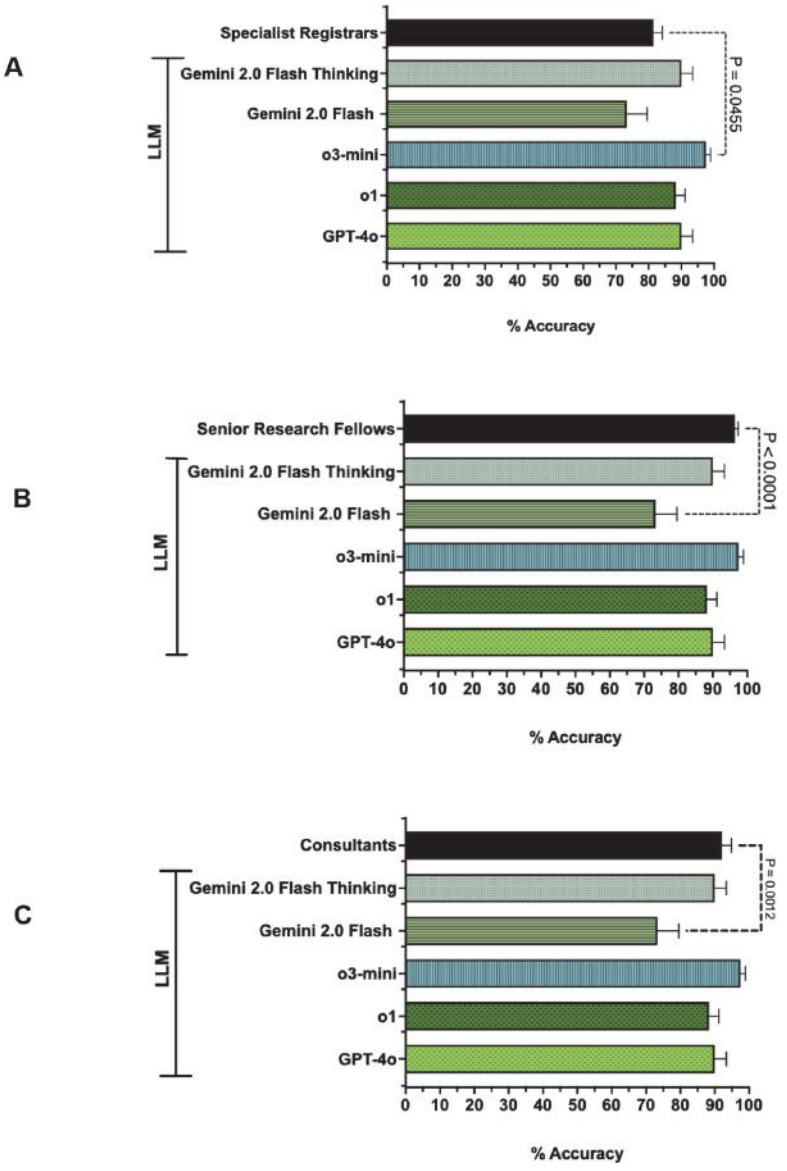
## P95 EVALUATION OF CHATGPT AND GEMINI IN GENERATING DIFFERENTIAL DIAGNOSES IN DIFFUSE INTERSTITIAL LUNG DISEASES (DILDS) FROM TRANSCRIBED RADIOLOGICAL REPORTS

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10.1136/thorax-2025-BTSabstracts.277

**Background** Diagnosis of Diffuse interstitial lung diseases (DILDs) is a complex task requiring observer experience. This study analyses the accuracy of Large Language Models (LLMs) in making DILD diagnoses from transcribed radiological reports and compares their accuracy to that of thoracic radiologists.

**Methods** LLMs analysed included ChatGPT (GPT-4, o1 and o3-mini) and Gemini (Gemini 2.0 Flash and Gemini 2.0 Flash Thinking). Radiologists of differing seniority (specialist registrars, senior research fellows, consultants [n=11]) from a tertiary referral centre were invited to participate. No patient data were used. Instead, 40 'Textbook' descriptors based on HRCT patterns/signs of common DILDs were created by a consultant radiologist. To mimic varying radiologists' reporting styles, four descriptors were generated for each DILD: narrative, telegraphic, with/without relevant negative findings. LLMs and radiologists were presented with the same HRCT descriptors in random order. Different prompting methods were used: 'unrestricted', 'restricted' and 'engineered' prompts. For the




**Abstract P95 Figure 1** Comparisons of diagnostic accuracy of LLMs and radiologists when given unrestricted prompts. The diagnostic accuracy of five LLMs (*GPT-4o, o1, o3-mini, Gemini 2.0 Flash and Gemini 2.0 Flash Thinking*) was compared to the diagnostic accuracy of **A)** Specialist registrars (SpRs), **B)** Senior research fellows, and **C)** Consultants. Accuracy of 100% = all first-choice diagnoses were correct. Tukey's multiple comparisons test was used to compare differences between LLMs. Significant P values are labelled on the graph. Statistical significance was assumed at P<0.05. Error bars signify mean ±SEM

'unrestricted' prompt, the groups were asked to provide their differential diagnoses in decreasing order of likelihood. For the 'restricted' prompt (LLMs only), LLMs were restricted to three differential diagnoses. Few-shot prompting techniques were also used to make an 'engineered' prompt, with the aim of improving diagnostic accuracy of LLMs.

**Results** Overall accuracy of the LLMs (unrestricted and restricted prompts): GPT-4o:90%, 93.3%; 01:84.2%, 74.2%; o3-mini: 96.7%, 100%; Gemini 2.0 Flash: 73.3%, 73.3%; Gemini 2.0 Flash Thinking:90%, 90%. Prompt Engineering did not improve diagnostic accuracy of LLMs. Overall accuracy of radiologists (unrestricted prompt): specialist registrars: 81.5%; senior research fellows: 96.5%; consultants: 92.1%.

**Conclusion** LLMs executed excellent diagnostic capabilities comparable to senior research fellows and consultants. LLMs could be used as adjunctive tools to improve the diagnostic accuracy of less-experienced radiologists. The results of prompt engineering techniques are potentially attributable to imperfect prompting methods. Future work should be targeted at establishing the diagnostic accuracy of LLMs designed for medical use, across a wider variety of DILDs, whilst ensuring safe diagnosis of patients.

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## P96 THE FIRST THREE YEARS OF THE R421 PULMONARY FIBROSIS GENETIC TESTING PANEL: OUTCOMES FROM A REGIONAL FAMILIAL ILD CLINIC

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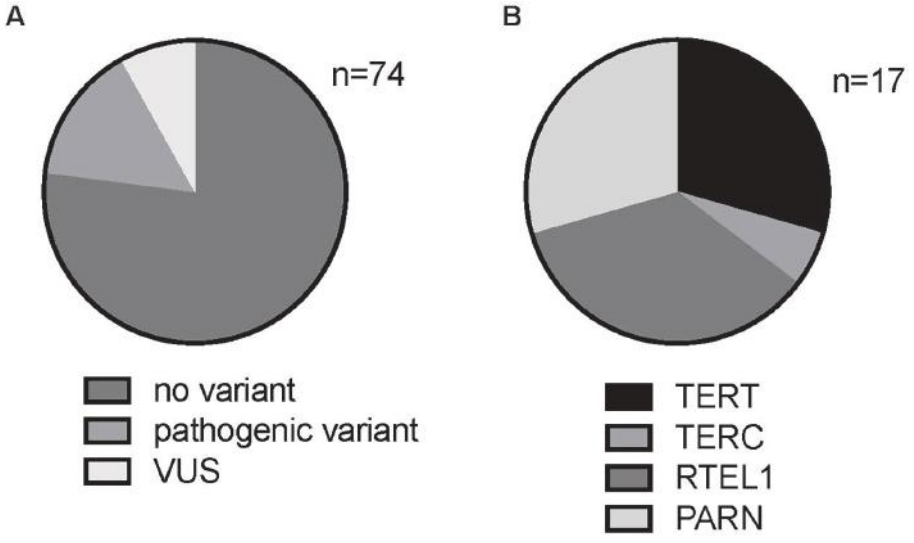
10.1136/thorax-2025-BTSabstracts.278

**Background** Familial pulmonary fibrosis (FPF) is defined as the presence of at least two affected first-degree relatives and accounts for ~10% of fibrotic ILD. Identifying an underlying monogenic cause is important for optimal patient management e.g. of extra-pulmonary features of telomere biology disorders (TBDs) or cancer surveillance (surfactant biology disorders) and for family screening to identify pre-clinical disease. The national test directory R421 panel, available from 2022, comprises 27 ILD-associated genes. We wished to review the outcomes of R421 testing from the regional FPF clinic at Royal Papworth to determine whether we can identify those likely to have positive findings through their clinical features, and to better inform patients about testing outcomes.

**Methods** We reviewed records of all patients within the FPF clinic at Royal Papworth who had undergone R421 genetic testing. We recorded genetic test results, demographics, family history, clinical data (FBC/LFT abnormalities, premature greying, history of malignancy) and radiology.

**Results** Seventy-four individuals had the R421 panel sent. Sixty-seven had at least one affected first-degree relative; others had early onset disease (n=5) or clinical suspicion of a TBD (n=2). Seventeen (23%) had a pathogenic variant or likely pathogenic VUS (figure 1A). All 17 were variants in telomere-related genes (RTEL1 (n=6), PARN (n=5), TERT (n=5) and TERC (n=1), (figure 1B)) with no surfactant biology-related variants found. Those with pathogenic variants were a similar age to those without (64.4 vs 61 yrs, $p>0.05$) and radiological pattern did not predict genetic result, with UIP being commonest (41% variant found; 36% no variant found). Those with pathogenic variants were more likely to have an FBC abnormality (47% vs 16%, $p<0.05$); no other clinical features associated with genetic diagnosis.

**Conclusions** Even within a cohort of individuals with a high pretest probability of a monogenic inherited ILD, <1/4 had a causal variant identified. Demographics and radiology were unable to predict a genetic result but those with an incidentally found FBC abnormality (macrocytosis/anaemia/thrombocytopenia) were more likely to have a TBD. These data highlight the challenges identifying those with monogenic ILDs before genetic testing. Studying larger cohorts may help us better understand predictors of genetic testing outcomes.




**Abstract P96 Figure 1** Results of R421 testing. **(A)** Proportion of results which identified no variant, a pathogenic variant or a likely pathogenic variant of uncertain significance (VUS). **(B)** Genes in which pathogenic or likely pathogenic variants were located

## P97 COMPARING INFLAMMATORY AND FIBROTIC HYPERSENSITIVITY PNEUMONITIS: INSIGHTS FROM A UK TERTIARY CENTRE

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10.1136/thorax-2025-BTSabstracts.279

**Background** Hypersensitivity pneumonitis (HP) is an immune-mediated interstitial lung disease characterised by lymphocytic infiltration of the airways and, in more advanced stages, fibrotic destruction of lung parenchyma, which worsens prognosis. Avoidance of the antigen (Ag) is crucial for modifying disease progression, yet it remains undetectable in over 50% of cases. The heterogeneity of HP and the lack of robust diagnostic and treatment guidelines complicate patient management.

**Aim** To assess differences between fibrotic HP (fHP) and inflammatory HP (iHP) phenotypes.

**Methods** We conducted a retrospective analysis of 160 HP patients from an ILD cohort at a large UK centre. Patients were categorised as fHP or iHP based on radiological reviews by the ILD multidisciplinary team. We analysed demographic, clinical, and radiological data and applied Chi-square tests for categorical variables and t-tests or Mann-Whitney U tests for continuous variables.

**Results** Amongst 2666 ILD cohort patients, HP prevalence was 6.2%, with 111 (69%) diagnosed with fHP (table 1). Patients with fHP were older (72.1 vs. 61.1 years, $p<0.001$) and predominantly male (42.3% vs. 14.2% $p\leq0.001$).

**Abstract P97 Table 1** Characteristics of inflammatory and fibrotic HP

| Characteristics | Inflammatory HP (n= 49 ) | Fibrotic HP (n=111) | p value |
|---|---|---|---|
| **Demographics** | | | |
| Age in years mean (±SD) | 64.1 (10.7) | 72.1 (10.2) | <0.001 |
| Female n(%) | 42 (85.7) | 64 (57.6) | <0.001 |
| BMI kg/m$^2$ mean (±SD) | 28 | 31.7 | 0.57 |
| **Pulmonary function test** | | | |
| FVC percentage predicted mean (±SD) | 84.3 (18.4 | 76.1 (21.0) | 0.032 |
| TLCO percentage predicted mean (±SD) | 65.8(15.1) | 50.0(13.76) | <0.001 |
| Obstructive spirometry yes(%) | 17.9 | 6.5 | 0.04 |
| **Smoking history** | | | |
| Lifelong non smoker n(%) | 28(57%) | 49 (44%) | 0.213 |
| <10 pack year history n(%) | 14(28%) | 13(11%) | |
| >10 pack year history n(%) | 14(28.5%) | 48(43.2%) | |
| **Exposures** | | | |
| Antigen identified (%) | 77.50 | 68.40 | 0.242 |
| Avian (%) | 89.40 | 59.00 | <0.001 |
| Feather bedding (%) | 7.80 | 15.70 | |
| Mould (%) | 0.00 | 2.60 | |
| Textiles (%) | 0.00 | 5.20 | |
| Other (%) | 2.60 | 21.00 | |
| Avian precipitins positive n(%) | 34 (79.1) | 53 (53.5) | 0.004 |
| **Radiology** | | | |
| Baseline radiological distribution | | | 0.501 |
| Upper zone predominant n(%) | 7(14.3) | 24(21.6) | |
| Lower zone predominant n(%) | 6 (12.2) | 10 (9) | |
| Diffuse n(%) | 36(73.5) | 77(69.4) | |
| **Bronchoalveolar lavage DCC** | | | |
| Bronchoscopy performed n (%) | 14(28.7) | 27(24.3) | n/s |
| Lymphocyte mean (±SD) | 29.1 (5.07) | 22.4 (4.4) | 0.252 |
| Neutrophil mean (±SD) | 14.6 (3.9) | 16.3 (2.4) | 0.872 |
| Eosinophil mean (±SD) | 1.89 (0.68) | 3.3 (0.61) | 0.115 |
| BAL lymphocyte distribution | | | 0.435 |
| Lymphocytes 1-15 % n(%) | 4(30.8%) | 14(51.9%) | |
| Lymphocytes 16-30 % n(%) | 4(30.8%) | 5(18.5%) | |
| lymphocytes > 30% n(%) | 5(38.5%) | 8(29.6%) | |
| BAL neutrophil distribution | | | 1 |
| Neutrophils <5% | 2(20%) | 5(20%) | |
| Neutrophils ≥5% | 8(80%) | 20(80%) | |

HP-hypersensitivity pneumonitis,BMI-body mass index;FVC-forced vital capacity; TLCO-transfer factor of carbon monoxide;Diffuse-all zones in both lungs; BAL-bronchoalveolar lavage; DCC-Differential cell count;Other-feather bedding,textiles,mould,vapors,gases,dust,fumes,chemicals

Notably, obstructive spirometry was more frequent in iHP (17.0% vs. 6.5%, p<0.04). Reported Ag exposure was similar in both groups (77% iHP vs. 68.4% fHP, p=0.242), with avian exposure being commonest (89.4% in iHP vs. 59% in fHP). Positive serum avian precipitins were more frequently detected in iHP (79.1% vs. 53.5%, p=0.004). Both groups showed diffuse lung involvement, and persistent groundglass (88.2%) and mosaicism (77.4%) was still seen in fHP. There was no significant difference in the cellular composition of bronchoalveolar lavage fluid (BALF) between the groups. Importantly, a BALF lymphocytosis of >30%[1] was seen in both fHP and iHP.

**Conclusion** Older age and male gender may represent risk factors for fHP. Positive serum precipitins may suggest persistent immune activation and represent an opportunity for immunomodulation in iHP to delay or prevent fibrotic complications. Prospective, longitudinal studies are required to further understand the evolution of iHP and role of serum precipitins and BALF lymphocytosis in disease behaviour.

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## P98 THE BURDEN OF COMORBIDITY IN FIBROTIC INTERSTITIAL LUNG DISEASE (FILD): A PROSPECTIVE STUDY IN A REGIONAL ILD CENTRE

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10.1136/thorax-2025-BTSabstracts.280

**Introduction** The pathogenesis of fibrotic interstitial lung disease (fILD) is poorly understood. We sought to understand whether there is an increased burden of comorbidities in patients diagnosed with fILD.

**Abstract P98 Table 1** Demographics and baseline characteristics of study participants expressed as N (%) unless otherwise specified. Abbreviations: FVC% (percent of predicted forced vital capacity), DLCO% (percent of predicted diffusing capacity for carbon monoxide), FEV1% (percent of predicted volume exhaled in one second), BMI (body mass index), ILD (interstitial lung disease), IPF (idiopathic pulmonary fibrosis), fILD (fibrotic interstitial lung disease), UIP (usual interstitial pneumonia)

| | Patients | Controls |
|---|---|---|
| **Age** (mean, range) | 73.7 (49-89) | 73.7 (49-89) |
| **Sex** | | |
| Male | 80 (78.4%) | 80 (78.4%) |
| Female | 22 (21.6%) | 22 (21.6%) |
| **Ethnicity** | | |
| White European | 102 (100%) | 102 (100%) |
| **Smoking status** | | |
| Current smoker | 0 | 2 (2%) |
| Former smoker | 60 (58.8%) | 48 (47%) |
| Never smoked | 42 (41.2%) | 52 (51%) |
| **BMI (kg/m$^2$)** (range, SD) | 28.3 (20.4-44.0, 4.7) | 27.5 (18.8-40.3, 3.92) |
| **FVC% predicted** (range, SD) | 71.6 (41-33, 16.8) | |
| **FEV1% predicted** (range, SD) | 75.5 (27-116, 17.3) | |
| **DLCO% predicted** (range, SD) | 45 (16-106, 15.2) | |
| **Disease duration in years** (mean, range) | 4.11 (0-16) | |
| **ILD diagnosis** | | |
| IPF | 61 (59.8%) | |
| Non-IPF fILD | 35 (34.3%) | |
| Indeterminate for UIP fibrosis | 6 (5.9%) | |

**Methods** 102 patients with a diagnosis of fILD were recruited from outpatient clinics at a regional ILD centre between March 2022 and March 2023. Data was obtained through semi-structured interviews and examination of medical records. Age and sex-matched controls were sourced from the Exeter 10,000 (EXTEND) cohort. Demographic and clinical characteristics are described in table 1.

Odds ratios (OR) and Fischer's exact test were used to compare the rates of each comorbidity in the fILD group with controls. The two-tailed paired t-test was used to assess the mean difference in number of prescribed medications between the two groups.

**Results** There was an increased likelihood of a diagnosis of diabetes (OR 4.75, 95% CI 1.72 – 13.1 p=0.003), connective tissue disease (OR 16.5, 95% CI 2.14 – 127.4 p=0.007) and hypertension (OR 45.38, 95% CI 6.06 – 339.48, p<0.0001) in the fILD group compared to controls.

It was not possible to calculate OR for liver disease or chronic kidney disease (CKD) as these were not identified in the control population. Using Fischer's exact test, there was an increased likelihood of CKD in the fILD population (p=0.029), but no increase in the likelihood of liver disease (p=0.12).

There was no difference in the likelihood of a diagnosis of heart disease, encompassing coronary artery disease, valvular heart disease and heart failure, (OR 1.58, 95% CI 0.77 – 3.23 p=0.21) or cancer (OR 0.80, 95% CI 0.41 – 1.57, p=0.52).

The mean number of prescribed medications per individual was 5.45 in the fILD group compared to 3.06 in controls (mean difference 2.39, p<0.0001). 36 patients and 9 controls were prescribed oral or inhaled corticosteroids.

**Conclusions** In our cohort, fILD patients were more likely than age-matched controls to have diabetes, connective tissue disease, hypertension and CKD. fILD patients had a higher medication burden than age-matched controls. These preliminary data lead us to consider whether fILD co-exists as part of a wider, multisystem disorder in some patients.

## P99 ABSTRACT WITHDRAWN

## P100 PLACE OF DEATH IN PEOPLE WITH PULMONARY FIBROSIS: A COHORT STUDY USING ELECTRONIC HEALTHCARE RECORDS IN ENGLAND

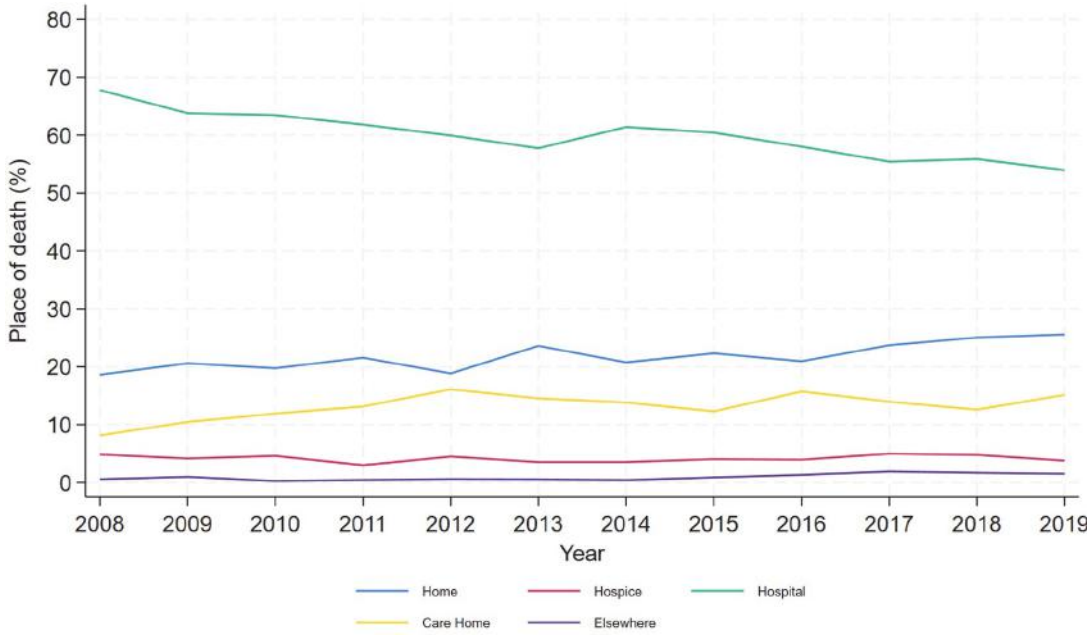
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**Background** Pulmonary Fibrosis (PF) describe a group of fibrotic lung diseases which are life-limiting. Despite inclusion in guidelines, only 20% of people with idiopathic pulmonary fibrosis are referred to palliative care services in England. Furthermore, large epidemiological studies on place of death in people with PF are lacking. The aim of this study was to estimate the proportion of people with PF who die in hospital and investigate factors associated with death in hospital.

**Methods** We used data from the Clinical Practice Research Datalink (CPRD) Aurum, linked with Hospital Episode Statistics Admitted Patient Care and Office of National Statistics mortality data to estimate the proportion of people with PF who die in hospital (vs community), and if this had changed over time. Multivariate logistic regression was used to examine factors associated with death in hospital.

**Results** The study population consisted of 13,706 people with at least one record of PF between 1/1/2008 and 31/12/2019. Mean age at death was 79.2 years (SD 9.6), median time between first record of PF to death was 2.5 years (IQR 11.5 months - 5.1 years) and 61.2% were male. The proportion of




**Abstract P100 Figure 1**

people with PF dying in hospital decreased from 67.8% in 2008 to 54.0% in 2019, whilst the proportion dying at home increased from 18.6% to 25.6% (figure 1). Factors associated with death in hospital included male sex (OR 1.12, 95% CI 1.02 – 1.22), younger age at death (OR for 18–69 years 1.58, 95% CI 1.35–1.82), South Asian ethnicity (OR 1.87, 95% CI 1.46–2.39), hospitalization from PF within 30 days of death (OR 4.41, 95% CI 3.85–5.04), comorbid ischaemic heart disease (OR 1.15, 95% CI 1.05–1.25) and heart failure (OR 1.23, 95% 1.10–1.25). Referral to palliative care strongly reduced the likelihood of death in hospital (OR 0.20, 95% 0.18–0.22).

**Conclusion** The proportion of people with PF dying at home has increased, but over half still die in hospital. Proportion of deaths in hospice beds remain unchanged, reflecting capacity and funding restraints. Our findings re-iterate the importance of palliative care in advanced care planning and choice of place in death in these patients.

## P101 CASE SERIES: THE ROLE OF BRONCHOALVEOLAR LAVAGE IN INTERSTITIAL LUNG DISEASE MANAGEMENT AND PATIENT OUTCOMES

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10.1136/thorax-2025-BTSabstracts.282

**Introduction** Bronchoalveolar lavage (BAL) is increasingly utilised in the diagnostic workup of interstitial lung disease (ILD) patients, often reducing the need for more invasive procedures like surgical biopsy. However, definitive data on how BAL results should consistently guide diagnostic and therapeutic decision-making in ILD management remain limited.

**Objective** To evaluate the utility of BAL cellularity, alongside other clinical and investigative findings, in the diagnosis and management of ILD patients within our hospital, and to assess its correlation with the overall disease course.

**Method** We conducted a retrospective review of patients who underwent BAL for ILD at Hull University Teaching Hospital from October 2021 to January 2024. Baseline demographics, pulmonary function tests (PFTs), high-resolution computed tomography (HRCT) findings, preliminary diagnoses, predominant BAL cellularity, administered treatments, and overall patient outcomes were analysed.

**Results** A total of 19 patients with non-idiopathic pulmonary fibrosis (IPF) ILD underwent BAL. The key findings are summarized in table 1.

**Conclusion** In this case series of 19 patients with non-IPF ILD who underwent BAL, cellular analysis contributed to the confirmation or exclusion of diagnoses amenable to steroid-responsive treatment. Although BAL cellularity aided early decision making regarding antifibrotic versus immunosuppressive therapy, the rate of FVC decline and mortality remains comparable to Idiopathic pulmonary fibrosis. This highlights the complex nature of non-IPF ILDs. A prospective, multicentre study with extended follow-up is warranted to definitively establish the comprehensive role of BAL in the long-term management and prognostic assessment of ILD.

**Abstract P101 Table 1** Demographic, clinical, radiological, BAL, and outcome data for ILD patients who underwent BAL. Continuous data are presented as mena (range)

| Parameter | | Information available on |
|---|---|---|
| **Age (years)** | 64 (36-84) | 19 |
| **Sex (male/female)** | 8/11 | 19 |
| **Smoking status** (Never/Ex/Current) | 9/7/3 | 19 |
| **Family history of ILD** | 8 | 8 |
| **Triggers identified** | Occupational 2, Pets 3 | 19 |
| **CT findings** | | |
| Ground glass | 15 | |
| Mosaic pattern | 8 | |
| Reticulation | 8 | |
| Bronchiectasis | 9 | |
| Interlobular septal thickening | 4 | |
| Honeycombing | 3 | |
| **BAL Findings** | | |
| Normal | 7 | 19 |
| Neutrophilia | 7 | |
| Lymphocytosis | 4 | |
| Eosinophilia | 0 | |
| Others/Non-Specific | 1 | |
| **Preliminary diagnosis (Before BAL)** | | 19 |
| Hypersensitivity pneumonitis | 9 | |
| NSIP | 3 | |
| Probable IPF | 3 | |
| CTD ILD | 1 | |
| Unclassifiable | 1 | |
| Others | 2 | |
| FVC (ml) | 2.67 (1.27-4.34) | 18 |
| FVC (%) | 75 (39-115) | 18 |
| TLCO (%) | 61.6 (36-91) | 16 |
| KCO (%) | 76.8 (45-104) | 16 |
| 6MWD (metre) | 330 (209-440) | 12 |
| Annual rate of FVC decline (ml) | -306 (-7, -770) | 6 |
| Annual rate of FVC decline (%) | -6.3 (-1, -17) | 6 |
| **Treatment given** | | |
| Anti-inflammatory | 7 | 19 |
| Anti-fibrotic | 6 | 19 |
| Others | 4 | 19 |
| Nil | 4 | 19 |
| Length of follow-up (days) | 625 (80 to 2446) | 19 |
| 1-year progression (%) | 6 (60% of 10 patients with 1-year FU data) | 10 |
| 1-year mortality (%) | 4 (21% of 19 patients) | 19 |

## P102 STRENGTH IN PARTNERSHIP: ADVANCING ILD CARE THROUGH COLLABORATION

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10.1136/thorax-2025-BTSabstracts.283

**Introduction** The Interstitial Lung Disease Interdisciplinary Network (ILD-IN) is a collaborative network for healthcare professionals of all disciplines caring for people with interstitial lung disease (ILD). Collaboration and partnership of organisations, including patient advocate charity, professional healthcare organisations and industries such as pharmaceutical, technology and legal, is key to achieving best patient outcomes in ILD. In January 2024, the ILD-IN set up quarterly meetings with partner organisations to share initiatives and work together, e.g. on co-producing patient resources.

**Aims** To evaluate ILD-IN partners' perceptions on the impact of collaboration with the ILD-IN on the following domains: influence and practice in ILD care, knowledge and skills sharing and improvements in ILD service provision and pathways.

**Methods** Data was collected via anonymous surveys and analysed in themes via descriptive statistics and free text thematic analysis.

**Results** Nine responses were received. Eight partners (88.8%) reported satisfaction from partnership with ILD-IN. All partners reported a positive impact on patient resource development. Key areas to align goals included education for healthcare professionals (8/9, 88.8%), education for patients and carers (7/9, 77.7%). Areas for improvement included opportunities for partner organisations to participate in activities, e.g. public involvement, teaching and policymaking.

**Conclusion** The ILD partners collaboration and partnership has the potential to promote best practice, support service and guideline development, support policy-making and research collaboration for people with ILD.

# The road to change in chronic cough

## P103 PATIENTS' PERSPECTIVE OF CONTROL IN CHRONIC COUGH

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10.1136/thorax-2025-BTSabstracts.284

**Introduction** Chronic cough (duration >8 weeks) is a common and burdensome medical problem; however, little is known about the concept of control of disease in chronic cough. In turn, existing patient reported outcome measures in cough focus almost exclusively on severity or health status. We aimed to investigate the concepts of control of cough from the patients' perspective.

**Methods** Consecutive patients with chronic cough were recruited from a specialist cough clinic for focus groups. Focus groups (max 4 participants) were semi-structured with open-to-closed questioning following an iterative process, and recruitment continued until thematic saturation. Interviews were recorded verbatim and analysed through open coding for thematic analysis.

**Results** A total of 14 participants were recruited; median (IQR) age 65 (57–70) years, 10 (71%) female, cough duration 15 (4–25) years, and 13 (93%) with a diagnosis of refractory chronic cough. A total of nine themes were identified, and broadly referred to frequency, urge, sequelae and suppression of cough.

Patients were burdened by cough frequency and fits or bouts.

'*…more than anything else…the frequency…of the coughing*'




**Abstract P103 Figure 1** A word cloud to demonstrate words and phrases most prominent from patient focus group transcript coding in relation to cough control. Larger font denotes greater frequency

'…a bad coughing fit…I feel as if my throat is completely closing up'

Patients were also affected by urge to cough, triggers of cough and the unpredictability of coughing.

'It's a very tickly sensation in my throat…'

'…*the smell of very strong aftershaves or detergent will trigger the cough…*'

'…that's the (unpredictable) cough that's worse because you don't know why (it happens)…'

Interference to activities of daily living, and physical, emotional and social impacts of cough, were significant sequalae.

'…we're asking you (patient) to leave because the cough is upsetting other patrons…'

'Sometimes I pee on myself (when I cough)…'

'I burst into tears…I was just so sad.'

Finally, patients valued the ability to suppress cough.

'I've got water…I'm sucking sweets (to suppress my cough).'

**Conclusion** Themes were identified with patients to facilitate the construction of a preliminary conceptual framework on control of chronic cough as a disease. Further work will investigate the concepts of control of cough with experts, and integrate both the patients' and experts' perspectives to construct a holistic conceptual framework of cough control.

## P104 THE VALIDITY OF A COUGH SEVERITY NUMERICAL RATING SCALE

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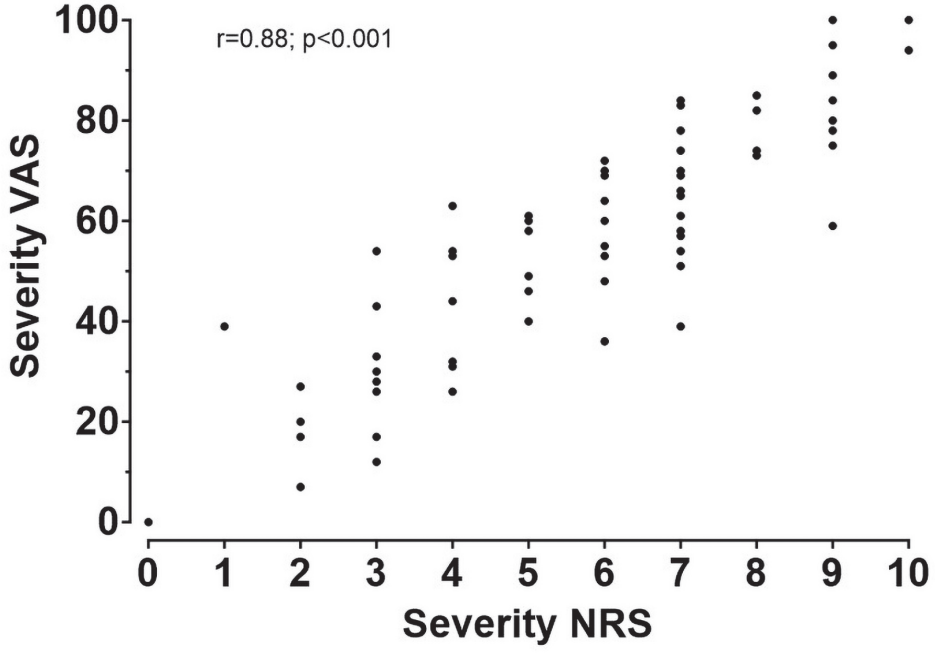
10.1136/thorax-2025-BTSabstracts.285

**Introduction** The visual analogue scale (VAS) is commonly used to assess cough severity in chronic cough patients. However, its main limitation is the difficulty patients have in marking the scale accurately, due to lack of gradings, which can lead to variability in repeated scores of stable patients. The numerical rating scale (NRS) is a graded and numbered solution. We aimed to investigate the performance of NRS against validated patient reported outcome measures and objective cough frequency monitoring.

**Methods** Consecutive patients with chronic cough from a specialist cough clinic completed cough severity NRS and VAS (0–10 or 0–100 mm respectively; higher scores = worse severity), patient global impression of severity (PGI-S) (0 (mild) -5 (very severe)) and cough-specific health status Leicester Cough Questionnaire (LCQ) (3–21; higher scores = better quality of life). Patients were invited to undergo 24-hour objective cough frequency monitoring with the Leicester Cough Monitor (LCM).

**Results** A total of 72 patients completed the assessments; mean (SD) age 62 (11.7) years, 55 (76%) female and 46 (64%) with a diagnosis of refractory chronic cough. Mean (SD) severity NRS, VAS, PGI-S and LCQ were 6 (2), 55 (24) mm, (3 (1) and 11.1 (3.4) respectively. The NRS scores were associated with VAS ($r=0.88$; $p<0.001$), PGI-S ($r=0.75$; $p<0.001$) and LCQ ($r=0.50$; $p<0.001$), and similar to VAS associations with PGI-S ($r=0.80$; $p<0.001$) and LCQ ($r=0.51$; $p<0.001$) (figure 1). 18 (25%) patients also completed objective cough monitoring; mean (SD) age 64 (12) years, 15 (83%) female. Geometric mean (SD) 24-hour total cough counts were 685 (2) coughs. NRS scores were associated with 24-hour total cough count ($r=0.54$; $p=0.020$).

**Discussion** The numerical rating scale is an intuitive tool, and our data support validity of the NRS in the assessment of chronic cough. NRS is highly associated with VAS, and has similar validity. Further larger-scale work should investigate the repeatability and responsiveness of the NRS in chronic cough.




**Abstract P104 Figure 1** Relationship of numerical rating scale (NRS) with visual analogue scale (VAS)

## P105 AN AUDIO TRIMMING ALGORITHM FOR COUGH ANALYSIS IN AMBULATORY RECORDINGS: A PROOF-OF-CONCEPT STUDY

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**Background** Cough frequency, typically reported as 24-hour cough frequency, is used as an objective measure of cough. It may be derived by manually annotating coughs in ambulatory audio recordings collected using established cough monitoring systems such as the VitaloJAK®. However, this can be a time-consuming process. Here we present an AI-based audio trimming algorithm that removes non-cough activity to provide reduced-length (trimmed) audio recordings to assist manual cough annotation. The aim of this study was to assess its performance in sensitivity (cough retention) and reduction of audio recording length, for more efficient cough analysis.
**Methods** A test data set was employed comprising 24-hour audio recordings from 20 adult chronic cough subjects (4M/16F) from the RaDAR database (Manchester University NHS Foundation Trust), collected using the VitaloJAK. Coughs were annotated by trained analysts totalling 14,931 coughs (range: 17–3,164; median: 469). Recordings were then trimmed by the algorithm and independent blinded analysts annotated the coughs on the trimmed recordings. Sensitivity (retention of original cough annotations) and percentage reduction in recording length post-trimming were computed. Agreement between 24-hour cough frequency derived from untrimmed versus trimmed recordings was assessed using intra-class correlation (ICC), linear regression and Bland-Altman analyses.
**Results** The algorithm achieved a median sensitivity of 100% (95% CI: 99.9–100; range: 97.6–100) and reduction in audio recording length of 95.4% (95% CI: 93.8–96.7; range: 75.5–97.7). 24-hour cough frequency derived from untrimmed versus trimmed recordings showed excellent agreement (ICC=0.99; $R^2$=0.99; p<0.001), with a unity slope of 1 (95% CI: 0.99–1.01) and minimal bias (intercept=11). Median difference bias was 8 coughs (p=0.8).
**Conclusions** The audio trimming algorithm may greatly reduce audio recording length with minimal impact on 24-hour cough frequency, showing strong potential to support more efficient cough analysis.

## P106 COMPARISON OF TWO DIFFERENT NEBULISATION SYSTEMS FOR CITRIC ACID COUGH CHALLENGE TESTING

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**Introduction** Cough challenge testing is used to study cough mechanisms. Cough responses are quantified by counting the maximum number of coughs evoked by increasing doses of inhaled tussive agents (Emax), the concentration of tussive agent evoking 50% of the maximum response or the concentration evoking 2 or 5 coughs (C2, C5). In recent years, the equipment used for cough challenge, Koko dosimeter (nSpire Health Ltd, UK) and characterised DeVilbiss 646 nebuliser (DeVilbiss Health Care Inc., US), have become irreparable and obsolete. The Provo.X (Ganshorn Medizin Electronic GmbH, Germany) is a potential replacement but requires comparison with established equipment. This study compared cough responses to citric acid using both devices and supplementing previous work for capsaicin cough challenge.
**Methods** Healthy adults attended two visits, were naïve to performing cough challenge and had normal spirometry. At visit 1, consented participants performed spirometry and citric acid cough challenge delivered by either the KoKo or Provo.X system (order randomised). Four single inhalations of doubling concentrations of citric acid (0.03–4.0M) were administered every 30 seconds, and the number of coughs evoked within 15 seconds recorded. The challenge ended when the maximum tolerated dose was reached or all concentrations inhaled. At visit 2, the alternative nebuliser system was used. A flow regulator valve (modified duckbill valve, Vernay Laboratories) was incorporated into the Provo.X nebuliser to limit inspiratory flow to 0.5L/s.
**Results** Twelve healthy volunteers were studied; 6(50%) males, median age 39(range 22–84), median(IQR) BMI 23.4(21.4–25.1), FEV1 and FVC%predicted 113%(100–122) and 120% (103–129) respectively.

Cough responses for the Provo X vs. KoKo were median (IQR) Emax 12.0(8.3–14.8) vs. 10.5(5.3–17.8) coughs, ED50 1.0M(0.625–2.0) vs. 1.0M(0.3125–2.0), C2 0.5M(0.25–1.0) vs 0.25M(0.156–1.75). In 16/24 challenges C5 was not reached.

Agreement between cough responses for the devices was Emax (intraclass correlation coefficient, ICC=0.87, p=0.001), ED50 (logED50 ICC=0.554, p=0.098) and C2 (logC2 ICC=0.51, p=0.135). There were no significant differences between cough responses across the range of concentrations from the two devices using GEE modelling, (p=0.393).
**Conclusions** Based on the minimal differences observed between the devices, the Provo.X appears to be a suitable and comparable system for performing citric cough challenge.

## P107 EVALUATING THE LOSS OF COUGH FREQUENCY RESOLUTION WHEN USING COUGH SECONDS INSTEAD OF EXPLOSIVE COUGHS

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10.1136/thorax-2025-BTSabstracts.288

**Background** Objective cough frequency is widely accepted as a measure of cough. It is commonly derived from the number of explosive cough sounds occurring in ambulatory audio recordings collected using cough monitoring systems such as VitaloJAK®. Cough second frequency has been proposed as an alternative measure whereby a cough second is any second of audio containing at least one explosive cough sound.[1] Here we compare the performance of cough frequency in 24 hours (C/24H) and cough second frequency in 24 hours (CS/24H) to assess the impact on derived measures.
**Methods** A total of 380 24-hour ambulatory audio recordings were obtained using the VitaloJAK from 77 chronic cough subjects (60F/17M) participating in a clinical study. Trained analysts annotated all coughs totalling 236,738 coughs (median 506) and 185,743 cough seconds (median 389) across all recordings. The relationship between C/24H and CS/24H was

assessed using linear regression analysis. The number of coughs per cough second was also computed. Furthermore, the percentage decrease in cough frequency resolution between C/24H and CS/24H was computed per recording.

**Results** A statistically significant linear correlation was observed between C/24H and CS/24H ($R^2$=0.98; p<0.0001; slope=0.76 (95% CI: 0.75–0.77); intercept=17.28). A 21.5% decrease in cough frequency resolution was observed between C/24H and CS/24H across all recordings (median 21.4; range: 0–38.3). It was also observed that 26.2% of cough seconds contained two or more coughs.

**Conclusions** While C/24H and CS/24H are linearly correlated, cough frequency resolution decreased by >20% when using cough seconds. Furthermore, multiple coughs occurred within cough seconds, making cough seconds less sensitive to changes in other measures of interest e.g. time-based cough bouts, which require individual time locations of coughs. Based on our findings, cough seconds are a less useful unit for measurement of cough than explosive cough sounds due to a loss of resolution impacting on measurement of cough frequency and bouts.

## REFERENCE

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## P108 PROVOCATION LARYNGOSCOPY IN THE MANAGEMENT OF CHRONIC COUGH; WHAT IS THE ADDED VALUE?

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10.1136/thorax-2025-BTSabstracts.289

**Aims and objectives** Chronic cough is an important condition with significant morbidity. Speech therapy has been recognised as a treatment option. The aim of this study was to review procedure outcomes from laryngoscopy and to review the utility of this procedure in a chronic cough cohort. Speech and Language Therapists (SLTs) conduct diagnostic provocation laryngoscopy as part of routine care for upper airway disorders (RCSLT 2021).

**Methods** Medical records were reviewed over 12 months in patients who had been seen in a chronic cough service. Demographics and assessment outcomes were collated for patients who had a diagnostic laryngoscopy.

**Results** 49 patients underwent a diagnostic laryngoscopy (of whom 76% female, mean age 59.8, SD = 15.37). For procedure outcomes, see table 1. Six patients were discharged. Referrals for Physiotherapy were for assessment of breathing pattern disorder. Of those commenced on sinus treatment, five (50%) had described symptoms of nasal congestion or post-nasal drip at initial consultation. Of those diagnosed with ILO, five (45.5%) did not report any breathlessness at initial consultation. No adverse events occurred in laryngoscopy.

**Conclusion** The benefits of laryngoscopy include: Confirmation of upper airway diagnoses and comorbidities, informing onward referrals and medical treatment, trialling and identifying beneficial therapy techniques for speech and language intervention. Images can also be reviewed for comparison at later date, to give reassurance to the patient and for bio visual feedback.

**Abstract P108 Table 1**

| Laryngoscopy outcomes | |
|---|---|
| SLT treatment | 42 (86%) |
| Diagnosis of Inducible laryngeal obstruction (ILO) | 11 (22%) |
| Further Respiratory investigation | 9 (18%) |
| Sinus treatment | 10 (20%) |
| Referral to ENT | 3 (6%) |
| Referral to Physiotherapy | 3 (6%) |

## P109 TREATABLE TRAITS IN COUGH: PATIENTS WITH REFRACTORY CHRONIC COUGH UNDERGOING LARYNGOSCOPY AND COUGH CONTROL THERAPY

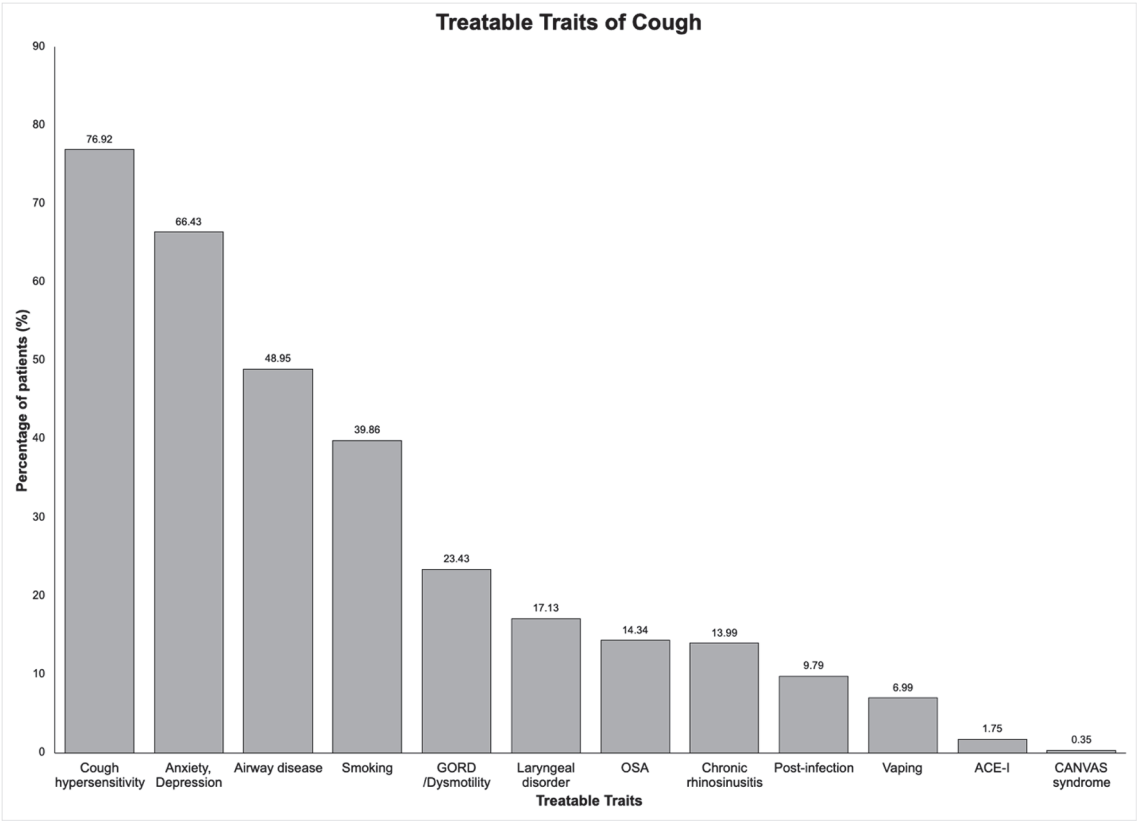
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10.1136/thorax-2025-BTSabstracts.290

**Introduction** Treatable traits have fundamentally changed chronic cough management. The BTS clinical statement[1] outlined 12 key traits, but there is limited research in this area. Patients with refractory chronic cough (RCC) may exhibit features of cough hypersensitivity but are otherwise poorly characterized. Speech therapy treatment has been shown to be an effective treatment and assessment with laryngeal endoscopy and provocation enables us to better elucidate traits in this group of patients.

**Methods** Retrospective review of electronic health records for 286 patients with RCC referred to speech and language therapy and phone triaged pre assessment between December 2022 to December 2024.

**Results** 196/286 (68%) patients were female, mean duration cough 34 months. 84.27% were overweight/obese, mean BMI = 31. Mean number of symptoms reported was 4 (1–8) including dry cough (80.07%), throat clearing (60.84%), laryngeal paraesthesia (72.38%), dysphagia (45%), dysphonia (51%), throat tightness (31%), and breathlessness (43%). Most (52%) reported 4 or more traits, mean number traits = 3.8 (0–9), including smoker/ex-smoker (39.86%), coexisting respiratory disease (48.95%) (asthma, COPD, ILD, bronchiectasis, sarcoidosis), poor inhaler hygiene (52.80%), poor fluid intake




**Abstract P109 Figure 1**

(45.10%), rhinitis, (38.46%), reflux (53.15%), post infection (9.79%), anxiety (44.06%), and depression (34.62%). Many cases of reflux and rhinitis were questionable. 129/286 (45.10%) had subsequent SLT-led laryngoscopy. Laryngoscopy with provocation revealed further traits including hypersensitivity (88.37%), laryngeal muscle imbalance (63.57%), dry/viscous secretions (51.16%), as well as inflammatory (15.50%), structural (13.18%) and neuromuscular impairment (4.65%) of the laryngeal and supra-laryngeal regions. Co-morbidity was common (mean number of comorbidities = 5 (0–17)).

**Conclusion** Patients with RCC exhibit a high symptom burden, multiple co morbidities and a high number of treatable traits. Obesity is common. Laryngoscopy allows extended phenotyping, identifying traits that are not usually considered, and allowing us to better manage this difficult disease.

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## P110 UPPER AIRWAY SPEECH AND LANGUAGE THERAPY-LED LARYNGOSCOPY CLINICS ARE SAFE AND ENABLE BESPOKE THERAPY PROGRAMMES TO BE DELIVERED

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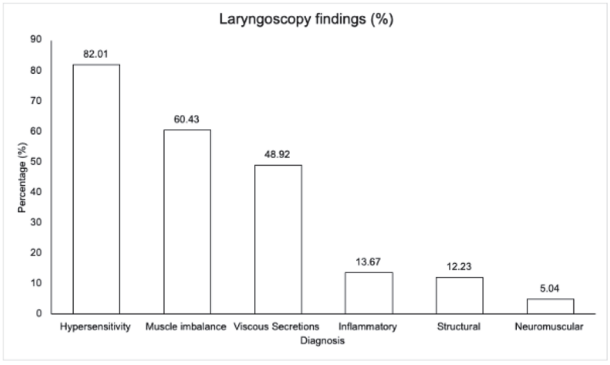
10.1136/thorax-2025-BTSabstracts.291

**Background** Cough hypersensitivity is associated with poor quality of life and high healthcare use. Cough can co-present with dysphonia, inducible laryngeal obstruction (ILO) and dysphagia. Speech and Language Therapists (SLTs) support assessment and management of these disorders.

This study reports on data from a specialist upper airway SLT led- laryngoscopy clinic, with access to on-call respiratory physicians and virtual otolaryngology image reviews.

**Method** A retrospective data review was conducted of 139 patients with suspected cough hypersensitivity, who attended the SLT-led laryngoscopy clinic between December 2022 and December 2024. Lidocaine was used for 2 patients. Provocation trials were informed by patient-reported triggers: vocal tasks (n=109, 78.42%), external irritants e.g. aerosol spray (n=79, 56.83%), oral trials including diet and fluids (n=32, 23.02%), and exertion (n=15, 10.79%). Data was collected using electronic health records. Abnormalities were reviewed by an Otolaryngologist.

**Results** No medical emergencies occurred.




**Abstract P110 Figure 1**

Diagnoses included cough hypersensitivity (n=114, 82.01%), laryngeal muscle imbalance (n=84, 60.43%), viscous secretions (n=68, 48.92%), laryngeal inflammation (n=19, 13.67%), structural impairment e.g. polyps (n=17, 12.23%), neuromuscular impairment e.g. vocal cord paresis (n=7, 5.04%) and ILO (n=22, 15.82%).

Exercise trials included: a) *Breath control*: Sniff and cheek puff (n=139, 55.40%), Sniff and blow (n=78, 56.12%), Tongue protrusion (n=29, 20.86%), Straw breathing (n=14, 10.07%), Pursed lips breathing (n=10, 7.19%) and others (n=9, 6.47%).

b) *Muscle tension release*: Tongue protrusion (n=72, 51.80%), Yawn (n=91, 65.47%), Inside smile (n=1, 0.72%), other (n=2, 1.44%).

c) *Voice quality*: Humming (n=23, 16.55%) Siren (n=3, 2.16%); Whimper (n=6, 4.32%); Sob (n=6, 4.32%); and others (n=15, 10.79%).

98 (70.50%) found breathing exercises to be the most effective, specifically sniff and puff (45/98, 45.92%) and sniff and blow (49/98, 50%).

Biofeedback was provided to 135 (97%) patients to support understanding of diagnosis and therapy.

**Conclusion** SLT-led laryngoscopy clinics are safe and do not require medical supervision. They enable co-morbidities to be identified, bespoke management programmes to be developed, and biofeedback to be provided. Further research is needed to look at the longer-term outcomes of these clinics, and patient satisfaction.

## P111 REDUCED COUGH SEVERITY AND IMPROVED CONFIDENCE IN SELF-MANAGEMENT FOLLOWING COMPLETION OF A VIRTUAL PHYSIOTHERAPY GROUP INTERVENTION FOR CHRONIC COUGH (VIP-COUGH)

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10.1136/thorax-2025-BTSabstracts.292

**Introduction** Prolonged waiting times significantly limited access to our respiratory physiotherapy service for cough suppression therapy. In response, we developed a **vi**rtual physiotherapy group intervention for individuals with chronic **cough** (ViP-Cough) to improve accessibility. The aim of this study is to describe both the uptake and potential benefits of this novel approach.

**Methods** A retrospective observational cohort study, involving patients with chronic cough who were referred to respiratory physiotherapy for cough control therapy at a single centre. All referrals were assessed by a specialist physiotherapist and offered access to the ViP-Cough. ViP-Cough comprised a single group therapy session which included: (1) education and lifestyle advice; (2) cough control therapy; and (3) breathing pattern retraining and vocal hygiene.

Patients completed outcome measures at baseline and four weeks following the group therapy session. These included: (1) a Numerical Rating Scale (NRS) for cough severity (range 0–10; higher scores indicating worse outcomes); and (2) an NRS for confidence in cough self-management (range 0–10; higher scores indicating better outcomes), both based on a seven-day recall period.

Descriptive statistics [mean (standard deviation)] were used to describe those referred and opted into ViP-Cough. Paired samples t-tests were used to compare pre-post treatment scores.

**Results** 194 individuals (153 female), aged between 25–87 years (median 61), were referred for physiotherapy for the treatment of refractory chronic cough. Of these, 70% reported experiencing cough symptoms for more than five years. Of those screened, 155 (80%) opted for ViP-Cough; however, 39 (25%) did not attend. Of the 116 who attended, 106 patients completed all assessments and were included in the final analysis.

Patients that completed ViP-Cough demonstrated a significant reduction in their NRS cough severity scores [ pre 6.84 (1.93) vs post 5.58 (2.25); mean difference (95% CI) -1.26 (-0.82, -1.70), p<0.001]. Accompanied by a statistically significant improvement in confidence in cough self-management [pre 3.85 (2.34) vs post 6.01(2.16); mean difference (95% CI) 2.16 (1.64, 2.68), p<0.001].

**Conclusion** ViP-Cough offers a promising approach for delivering non - pharmacological cough control therapy at scale, and lower cost. These preliminary positive findings warrant further testing in a randomised controlled trial.

## P112 DIFFERENCES IN AX-8 AND PLACEBO EFFECTS ON COUGH BOUTS IN REFRACTORY OR UNEXPLAINED CHRONIC COUGH: A NEW COUNT- AND DURATION-BASED ANALYSIS

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10.1136/thorax-2025-BTSabstracts.293

**Background** Previous analyses from the AX8–003 clinical study indicated that in patients with refractory or unexplained chronic cough (RCC/UCC) and throat discomfort (baseline visual analogue scale [VAS] score ≥55 mm), the TRPM8 agonist AX-8 reduced bout frequency (cough bouts are defined as clusters of ≥2 coughs separated by ≤2 seconds),[1] more effectively than cough frequency. This study further explores how AX-8 preferentially targets bouts, examining detailed count- and duration-based metrics.

**Methods** This was a randomized, double-blind, placebo-controlled crossover study of RCC/UCC patients (≥1 year cough duration, cough severity VAS score ≥40 mm, and 24h cough frequency ≥10 coughs/h at screening). In the throat discomfort subgroup (n=24), we compared the effect of AX-8 (40 mg orally disintegrating tablet BID) versus placebo on cough and bout frequencies, as previously, as well as on single and bout cough frequencies, mean number of coughs per bout, and total and mean bout durations. Analyses below focus on the 4-hour period following the first dose on day 1.

**Results** AX-8 showed a non-significant decrease in single cough frequency (15.5%, p>0.2) but significantly reduced bout cough frequency (50.4%, p=0.001) without affecting the number of coughs per bout (p>0.2). Placebo, conversely, moderately reduced the number of coughs per bout. In terms of cough duration, AX-8 significantly lowered the total bout duration (51.4%, p<0.01), primarily by reducing bout frequency (p<0.001) but not the mean bout duration (p>0.1). In contrast, placebo's small benefit arose from shortening the mean bout duration.

**Conclusion** These data demonstrate that AX-8 selectively targets the initiation of bouts rather than single coughs, which are less important for RCC/UCC patients. The different effects of AX-8 and placebo on bout characteristics may reflect distinct underlying neurophysiological mechanisms.
**Clinical Trial Registry** NCT04866563

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## P113 EFFICACY AND SAFETY OF NALBUPHINE EXTENDED-RELEASE IN REFRACTORY CHRONIC COUGH: RESULTS FROM THE PHASE 2A RIVER TRIAL

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10.1136/thorax-2025-BTSabstracts.294

**Introduction and Objectives** Refractory chronic cough (RCC) persists despite guideline-based treatment for any comorbid cough-associated conditions and carries a high burden of disease and impact on patients' lives. Nalbuphine extended-release (NAL ER, oral tablet form) is a kappa receptor agonist and mu receptor antagonist which acts on the cough reflex arc centrally and peripherally, targeting opioid receptors involved in chronic cough. The RIVER trial (NCT05962151) evaluated NAL ER in patients with refractory chronic cough (RCC).
**Methods** This double-blind, placebo (PBO)-controlled, crossover study enrolled patients with RCC stratified into 2 subgroups based on 24-h cough frequency at screening (10–19 coughs/h [moderate] and ≥20 coughs/h [severe]) measured by a cough monitor. Patients were randomly assigned to one of two 21-day treatment sequences: NAL ER to PBO or PBO to NAL ER, with a 21-d washout between treatment periods. NAL ER was titrated every 7 d (27 mg, 54 mg, and 108 mg, all BID). The primary endpoint was relative change from baseline in 24-h objective cough frequency at day 21 in all patients.
**Results** Sixty-six patients were randomized (mean age, 60.2 y; mean cough frequency, 34.7 coughs/h). There was a significant difference in the relative change from baseline in 24-hour cough frequency with NAL ER 108 mg compared to PBO (-65% and -9%, respectively with a PBO-adjusted improvement of 57%; $P<.0001$; figure 1). Cough frequency subgroups of 10–19 and ≥ 20 coughs/hr achieved PBO-adjusted improvements of 75% and 51%, respectively (both $P<.0001$), with NAL ER at day 21 (108 mg). Significant reductions in 24-h cough frequency occurred at day 7 with 27-mg NAL ER BID ($P<.0001$). No serious treatment-emergent adverse events (TEAEs) were reported; 10 patients discontinued treatment due to TEAEs. The most common (≥10%) TEAEs were constipation, somnolence, nausea, dizziness, headache, and fatigue.
**Conclusions** The significant reduction in 24-h cough frequency supports further investigation of NAL ER for RCC.

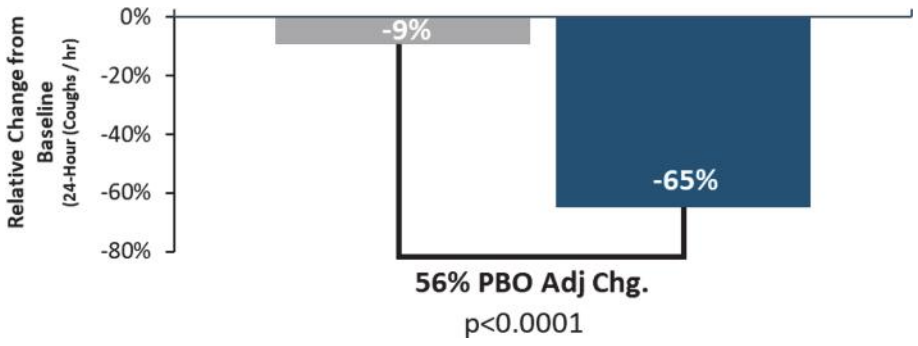
## P114 DEVELOPMENT OF ACCESSIBLE DIGITAL RESOURCES TO ENHANCE UPPER AIRWAY CONTROL THERAPY

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10.1136/thorax-2025-BTSabstracts.295

**Background** Speech and Language Therapists (SLTs) support management of upper airway disorders including chronic cough and inducible laryngeal obstruction (ILO). Interventions are complex, often including psychoeducation, breath/cough control exercises, and lifestyle changes. In a specialist UK upper airways service, patient and staff engagement identified demand for digital resources to promote self-management.
**Methods** A website was developed in collaboration with patients, the wider multidisciplinary team and the Communications Team. Video content included a patient story, and breathing exercises. Written and pictorial information was presented on differential diagnoses and management. Accessibility tools enabled information to be accessed audibly, and via different languages, font sizes and colour contrasts. Paper versions of leaflets remained in use to minimise digital exclusion.

Relevant links were shared directly with patients via SMS upon receipt of referral, shared during clinics, and added to emails, appointment letters, reports, and leaflets in both text and QR code form. The website was also shared via social media and clinical networks. The communications team collected activity and satisfaction data.
**Results** Over two years, the webpages have been accessed a total of 94,426 times, from across the UK. The most frequently viewed pages were ILO (38,748 views), laryngeal hypersensitivity (16,810 views), breathing exercises (16,728




**Abstract P113 Figure 1** Relative Change from Baseline at Day 21 (108 mg BID)

views) and chronic cough (11,144 views). The least viewed page was throat care (2,709 views). The most frequently watched videos related to breathing exercises (11,857 views).

In addition to direct links, webpages were accessed via search engines, notably google (n= 49,086). Mobile devices (57%) were used more than desktop devices.

The average satisfaction rating was 9.5/10 (Range 9.3–9.8). The ILO, chronic cough and breathing exercise pages were ranked in the top 3 spots for highest satisfaction across 2024 for the host NHS Trust.

**Conclusion** There is demand for digital information on upper airway disorders, notably ILO and breathing exercises. Websites enable people to access information regardless of local service provision, promoting more equitable and consistent access to healthcare resources. There is a need for more formal intervention development, and evaluation of impact, including cost effectiveness.

## P115 ASSOCIATION BETWEEN BASELINE COUGH BURDEN AND GEFAPIXANT DISCONTINUATIONS DUE TO TASTE-RELATED ADVERSE EVENTS

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10.1136/thorax-2025-BTSabstracts.296

**Introduction and Objectives** The phase 3 COUGH-1/-2 trials (NCT03449134, NCT03449147) demonstrated that gefapixant 45 mg twice daily (BID) is effective for treatment of refractory or unexplained chronic cough but may be associated with taste-related adverse events (taste AEs), potentially affecting treatment continuation. Here, we evaluate whether there is an association between baseline cough burden and gefapixant discontinuations due to taste AEs.

**Methods** COUGH-1/-2 methods have previously been reported (McGarvey et al. *Lancet*. 2022;399:909–923). This post hoc analysis evaluated odds of discontinuing treatment with gefapixant 45 mg BID due to taste AEs based on baseline cough burden variables (24-hour cough frequency, cough severity visual analog scale, Cough Severity Diary, and Leicester Cough Questionnaire).

**Results** Of 683 participants receiving gefapixant 45 mg BID, 95 (13.9%) discontinued treatment due to taste AEs. Subgroups with categorically lower baseline cough burden had higher odds of discontinuation rates due to taste AEs (figure 1).

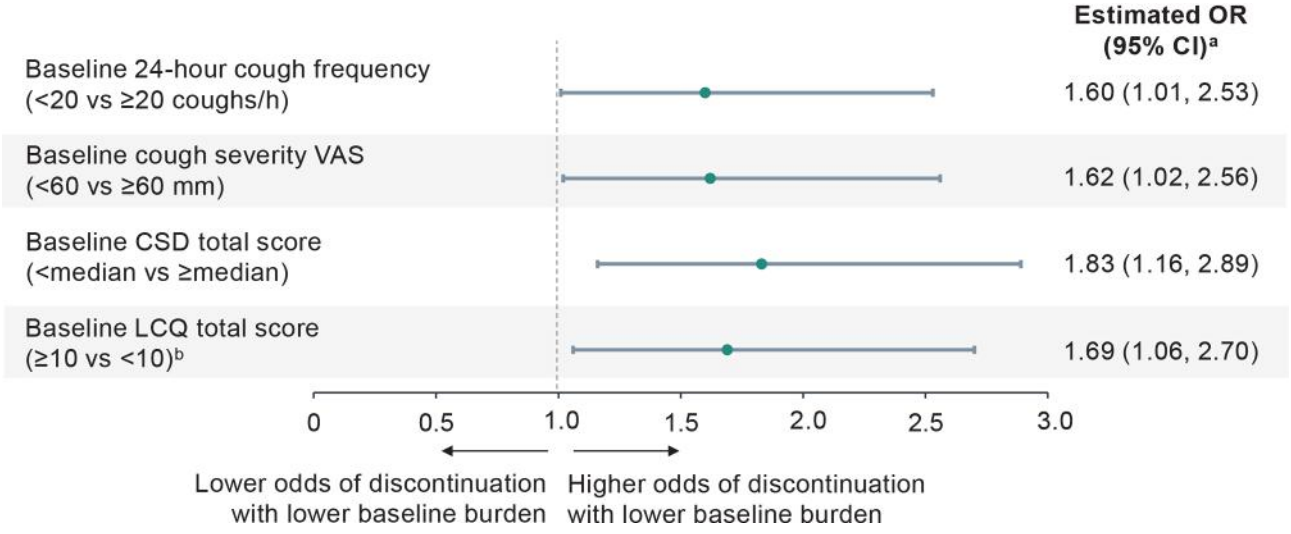
**Conclusions** These findings suggest that patients with higher baseline cough burden may be more willing to continue treatment regardless of taste AEs. Because results of this post hoc analysis are observational, they require further confirmation.

## P116 CLINICALLY RELEVANT ATTENUATION OF COUGH WITH GEFAPIXANT: A POST HOC ANALYSIS

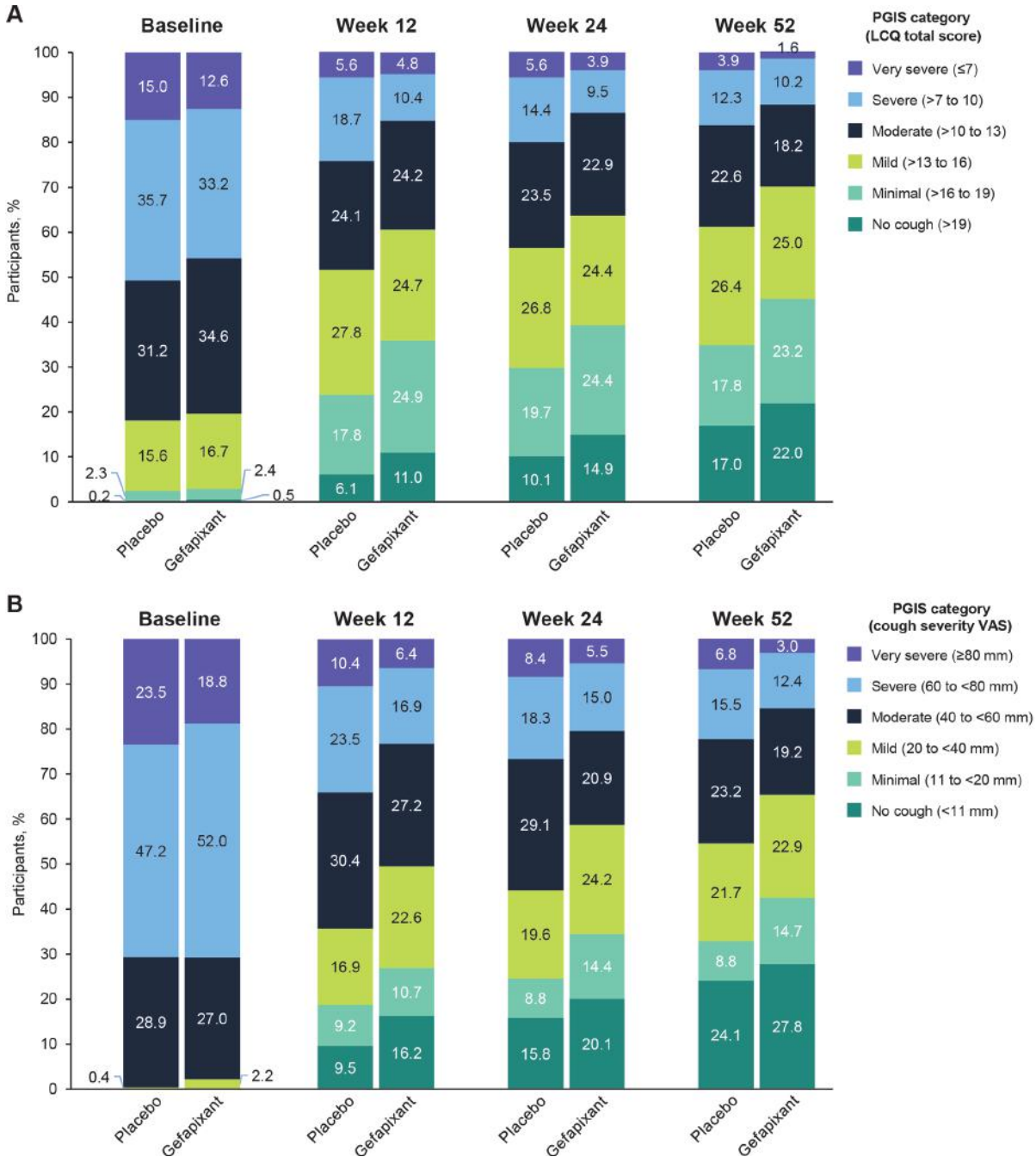
[1]S Birring, [2]P Dicpinigaitis, [3]L Dupont, [4]L McGarvey, [5]A Morice, [6,7]J Smith, [8]J Xu, [8]A Martin Nguyen, [8]G Philip, [8]C La Rosa. *[1]Centre for Human and Applied Physiological Sciences, School of Basic and Medical Biosciences, Faculty of Life Sciences and Medicine, King's College London, London, UK; [2]Albert Einstein College of Medicine and Montefiore Medical Center, Bronx, New York, USA; [3]University Hospital Leuven, Katholieke Universiteit Leuven, Leuven, Belgium; [4]Wellcome-Wolfson Institute for Experimental Medicine, School of Medicine, Dentistry and Biomedical Science, Queen's University Belfast, Belfast, UK; [5]Hull York Medical School, Cottingham, UK; [6]Division of Infection, Immunity and Respiratory Medicine, University of Manchester, Manchester, UK; [7]Manchester University NHS Trust, Manchester, UK; [8]Merck and Co., Inc., Rahway, USA*

10.1136/thorax-2025-BTSabstracts.297

**Introduction and Objectives** Patient-reported outcomes (PROs) to assess refractory or unexplained chronic cough in phase 3 trials of gefapixant (COUGH-1/-2 [NCT03449134, NCT03449147]) included the Leicester Cough Questionnaire




**Abstract P115 Figure 1** Probability of discontinuing gefapixant 45 mg BID due to taste-related AEs in baseline cough burden groups based on 24-hour cough frequency, cough severity VAS, CSD total score, and LCQ total score. AE, adverse event; BID, twice daily; CSD, Cough Severity Diary; LCQ, Leicester Cough Questionnaire; OR, odds ratio; VAS, visual analog scale. [a] Estimated ORs based on logistic regression with the following covariates: age, sex, region, study, baseline cough duration. [b]For the LCQ, a lower total score indicates a higher cough burden




**Abstract P116 Figure 1** Percentage of participants by PGIS categories for **(A)** LCQ and **(B)** cough severity VAS scores at baseline and over time of treatment with gefapixant 45 mg twice daily vs placebo. LCQ, Leicester Cough Questionnaire; PGIS, Patient Global Impression of Severity; VAS, visual analog scale

(LCQ) and cough severity visual analog scale (VAS). Severity categories for these PROs anchored against the Patient Global Impression of Severity (PGIS) scale were recently defined (Rhatigan et al. *J Allery Clin Immunol Pract*. 2023;11:3706–3712). Here, we evaluate categories of cough severity in LCQ and VAS scores over time in COUGH-1/-2.

**Methods** COUGH-1/-2 methods have previously been reported (McGarvey et al. *Lancet*. 2022;399:909–923). Post hoc analyses evaluated LCQ and cough severity VAS scores with gefapixant 45 mg twice daily and placebo based on PGIS cough severity thresholds. As the PGIS was not used in COUGH-1/-2, LCQ and VAS score ranges corresponding to the 6 PGIS categories (shown in figure 1) were based on Rhatigan et al.

**Results** By Week 12, a higher percentage of participants treated with gefapixant vs placebo (35.9% vs 23.9%) had LCQ scores corresponding to the 2 lowest PGIS categories of *minimal* or *no cough* (figure 1A). By Week 52, respective percentages were 45.2% vs 34.8%. Cough severity VAS results were similar (Week 12, 26.9% vs 18.7%; Week 52, 42.5% vs 32.9%; figure 1B).

**Conclusions** A sizeable and higher percentage of participants treated with gefapixant vs placebo experienced clinically relevant attenuation of cough, which increased over time.

## P117 PATIENT GLOBAL IMPRESSION OF CHANGE: CONTEXTUALIZING PATIENT-REPORTED OUTCOMES IN GEFAPIXANT PHASE 3 TRIALS

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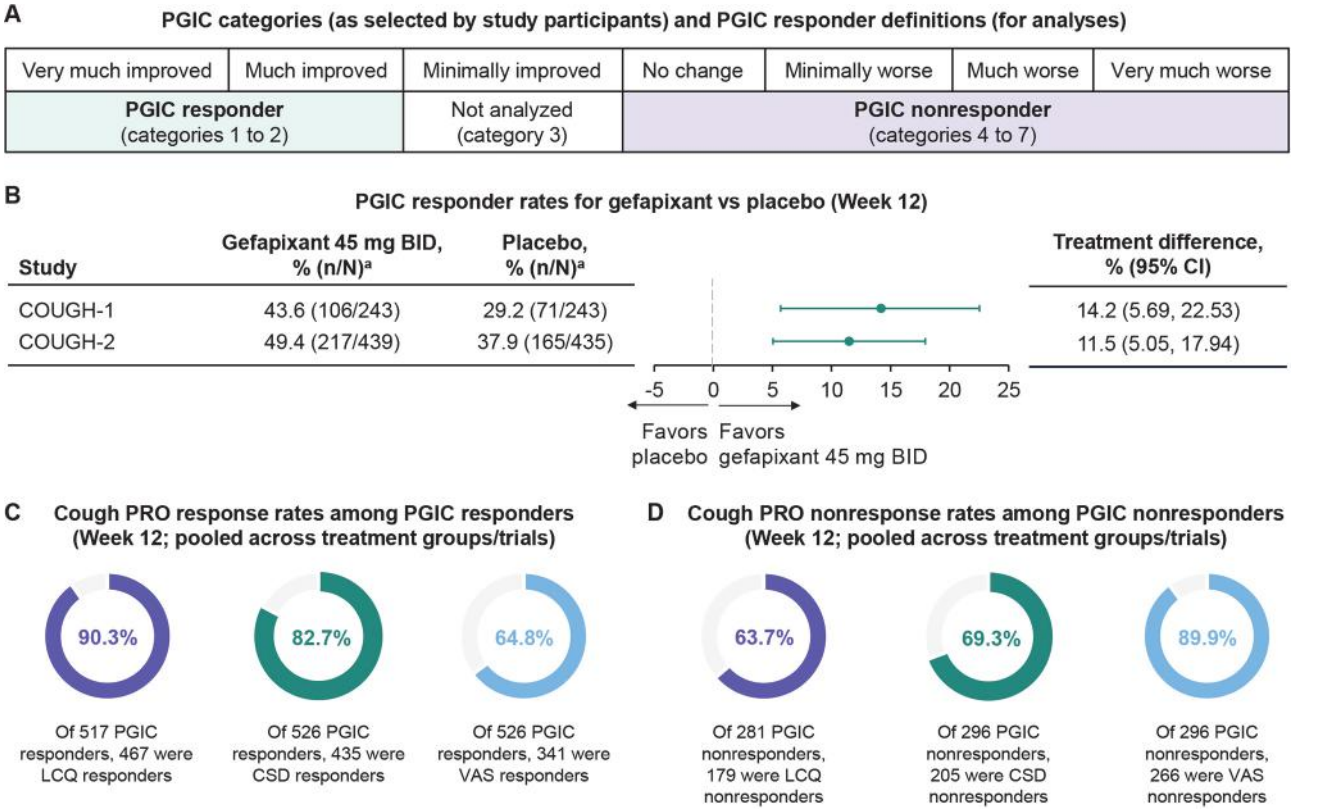
10.1136/thorax-2025-BTSabstracts.298

**Introduction and Objectives** Cough patient-reported outcomes (PROs) used in gefapixant clinical trials may not be widely understood or used in clinical practice. The Patient Global Impression of Change (PGIC) PRO may be more intuitive and relevant to physicians. Here, we use prespecified PGIC responder analyses and post hoc analyses to assess the relationship between clinically meaningful responses as defined by cough PROs vs PGIC in two phase 3 trials (COUGH-1/-2 [NCT03449134, NCT03449147]).

**Methods** COUGH-1/-2 methods have previously been reported (McGarvey et al. *Lancet*. 2022;399:909–923). In prespecified analyses, PGIC responder rates were compared between gefapixant 45 mg twice daily and placebo. Post hoc analyses assessed the relationship between PGIC responders (ie, *very much improved* or *much improved*) and cough PRO responders pooled across both treatment groups and both trials.

**Results** PGIC responder rates in COUGH-1/-2 were higher for gefapixant vs placebo; the 95% CIs for treatment differences excluded 0. Across treatment groups/trials, there was strong agreement between PGIC and cough PRO responses (figure 1).

**Conclusions** In COUGH-1/-2, there were more PGIC responders in the gefapixant vs placebo group. The relationship between responses on the PGIC and cough PROs suggests cough PRO responses reported in COUGH-1/-2 are similar to the observed results of the PGIC, an endpoint that may be more easily interpreted in clinical practice.




**Abstract P117 Figure 1** **(A)** PGIC categories and responder definitions. **(B)** Proportions of PGIC responders (gefapixant 45 mg BID vs placebo) and treatment difference. **(C)** Agreement between PGIC and cough PROs for responders. **(D)** Agreement between PGIC and cough PROs for nonresponders. LCQ response was defined as ≥1.3-point increase. CSD response was defined as ≥1.3-point decrease. VAS response was defined as ≥30-mm decrease. Nonresponse to LCQ, CSD, and VAS was defined as not meeting responder criteria. $_a$n=PGIC responder, and N=number of participants with data after nonresponder imputation at the time point. BID, twice daily; CSD, Cough Severity Diary; LCQ, Leicester Cough Questionnaire; PGIC, Patient Global Impression of Change; PRO, patient-reported outcome; VAS, visual analog scale

# Respiratory realities: the patient perspective

## P118 UNDERSTANDING THE EXPERIENCE AND UNMET NEEDS OF PATIENTS LIVING WITH SEVERE ASTHMA ON BIOLOGIC THERAPY; INCLUDING VIEWS ON CLINICAL REMISSION

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10.1136/thorax-2025-BTSabstracts.299

**Introduction and Objectives** Severe asthma (SA) affects a minority of asthma patients but has a disproportionately high impact on quality of life. Despite advances in biologic therapies, patients still face significant daily burdens. Little is known about how patients perceive their care, including their understanding of the concept of clinical remission (CR). This study explored the lived experience of people with SA receiving biologic therapy and identified perceived gaps in care.

**Methods** On behalf of GSK, Ipsos conducted a 10-minute online survey among 100 UK adults with SA. Participants were recruited via Asthma+Lung UK and online panels. This analysis focuses on the 75 patients currently receiving biologics (67% [n=50] female, mean age 49 years, regionally diverse). The survey explored symptom burden, care experiences, treatment goals, and familiarity with the concept of CR.

**Results** Breathlessness (62%; n=44) and low energy (54%; n=38) were reported as the most burdensome symptoms. Over half of patients indicated SA negatively impacted key areas of life (figure 1). One in four expressed ongoing worry about their condition, and one in five reported struggling to cope. Experiences of care varied, with challenges noted in accessing and managing treatment. Over half (61%; n=46) were unfamiliar with the term CR, though 55% (n=41) considered it achievable and 95–97% (n=71–73) recognised the importance of treatment goals that define it. A majority (77%; n=58) felt it was very important that healthcare professionals discuss treatment goals as part of shared decision-making. Respiratory consultants/specialists (64%; n=48), specialist nurses (51%; n=38) and GPs (45%; n=34) were the most valued sources of support. However, some patients reported dissatisfaction with diagnostic delays (24%; n=18), referral speed (19%; n=14), and information provision (19%; n=14).

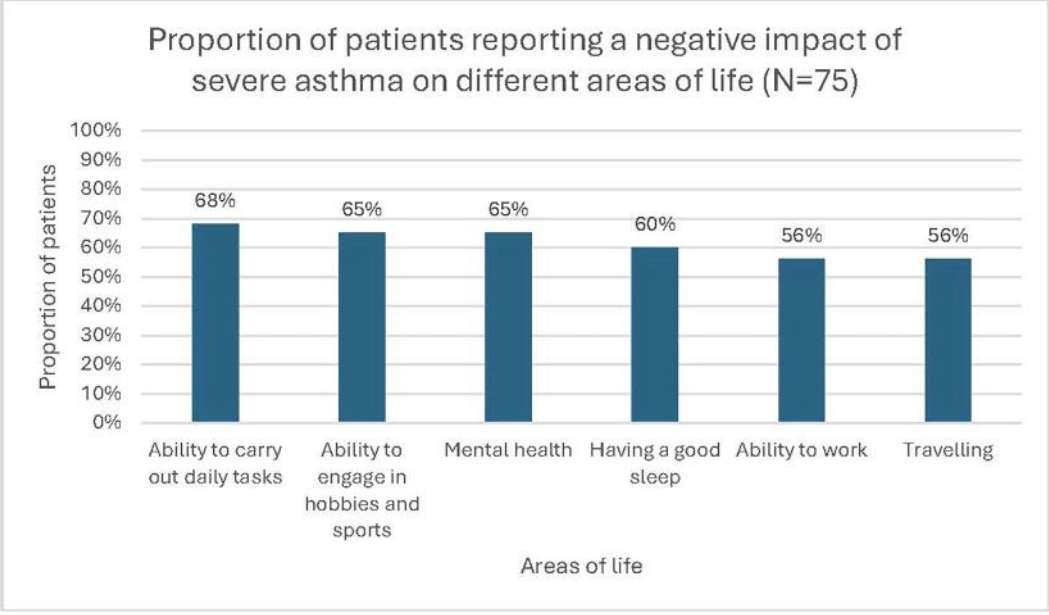
**Conclusions** SA continues to place a significant emotional burden and practical limitation on patients' lives. Individual markers of CR are seen as attainable goals, yet awareness of the concept itself is limited. Patients value shared decision-making and support from specialists, but gaps in care persist. These findings highlight the need for more consistent, patient-centred care prioritising mental health, timely access to services, and meaningful patient engagement through improved communication, education, and shared decision-making.

## P119 IMPLEMENTING INHALER RECYCLING SCHEMES – EXPERIENCE WITHIN A LARGE UK NHS TRUST

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10.1136/thorax-2025-BTSabstracts.300

**Introduction** Inhaler devices contribute to 3% of the overall NHS greenhouse gas emissions, with the majority of emissions coming from propellant gases in pressurised metered dose inhalers (pMDI). Many inhalers are disposed of when they have doses remaining,[1] resulting in medicines wastage, healthcare costs and adverse environmental impacts.




**Abstract P118 Figure 1** Proportion of patients reporting a negative impact of severe asthma on different areas of life

There are no national inhaler recycling schemes available, and the cost of recycling has been prohibitive in many areas of the UK. We report the outcome of a hospital-based inhaler recycling scheme.

**Methods** Data are available from April 2024 to June 2025. We partnered with Grundon Waste Management Ltd to recycle pMDI inhalers, whilst DPI are incinerated locally with energy capture. Inhalers were collected from wards and respiratory outpatient clinics at the Leeds Teaching Hospitals NHS Trust. The number of doses remaining in each device was used to calculate carbon emission savings. For inhalers without a dose counter, the number of doses remaining was assumed to be the same as collected inhalers with a dose counter in the same drug class,or estimated from previous studies.[1]

**Results** A total of 2,907 inhalers were collected, of which 1,934 (66.5%) were pMDIs. The most common class of inhaler device collected were SABA (1,127), ICS/LABA (474), and ICS/LABA/LAMA inhalers (445). The calculated carbon footprint of recycled inhalers was 13.2 tonnes CO2e.

We identified a significant wastage of drug; inhalers with a dose counter had an average of 65% of their labelled number of doses remaining, and 46% of these had been in use for no more than three days. The overall wastage costs were estimated at £27,072.

**Conclusions** Hospital-based inhaler recycling has a significant impact on reducing carbon emissions. Significant wastage was identified, likely due to treatment optimisation during hospital admissions. We aim to ensure the long-term viability of our scheme by working with other local hospitals to share recycling costs, and from savings achieved by reducing waste.

**Acknowledgements** This project was supported by a project grant from Chiesi Limited.

## REFERENCE

1. Murphy AC, *et al. BMJ Open Respir Res*. 2024;**11**:e002579.

## P120 HOW CAN WE EMBED SUSTAINABILITY IN THE INHALER PRESCRIPTION-USE-DISPOSAL PATHWAY?

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10.1136/thorax-2025-BTSabstracts.301

**Background** The UK has taken steps to promote sustainability in respiratory medicine, with policies focusing on reducing the environmental impact of inhalers by increasing low-carbon inhaler prescription and supporting patients on low-carbon inhalers vis-à-vis the NHS net zero plan, BTS/SIGN/NICE guidance and the UK Government's Environment Act 2021. Pharmaceutical companies and others have also led pilot inhaler recycling schemes, and in 2023, an inhaler waste recycling company, Grundon, was launched. However, evidence of improvements remains patchy and organised approaches to bring the literature together into actionable insights have been lacking. This mixed methods study builds on our previous systematic review (n=63 included studies, PROSPERO CRD42024541927) to bring together survey and qualitative data and generate actionable insights using the people, product and process framework.

**Methods** Two online surveys for patients and healthcare professionals (HCPs) were disseminated via social media, respiratory HCP associations, Asthma+Lung UK and other charities in respiratory health. Interviews with stakeholders along the inhaler prescription-use-disposal pathway were conducted and analysed using the Framework method. The study took place between August 2024 and January 2025, with ethical approval obtained from the University of Birmingham.

**Results** 141 people completed the surveys, and 27 interviews were conducted. Amongst HCPs completing the survey, 86% were aware of appropriate inhaler disposal and recycling, but only 12.5% had a formal inhaler disposal or recycling program in place. Limitations identified included policies, resources and infrastructure, with primary care participants being more likely than others to identify inadequate infrastructure as a barrier (63 vs 38%, $p<0.02$). 54% of patients identified their inhalers by colour, while 43% supported the removal of colours from inhalers to reduce environmental impact. Qualitative insights included uncertainty over responsibility for inhaler returns collection between GPs and community pharmacies, lack of incentives to return inhalers and need for patient education on inhaler adherence and use techniques.

**Conclusion** Actionable insights include routine patient education on sustainable inhaler use, low-carbon prescribing as defaults, and standardised inhaler disposal guidance, infrastructure and pathways as priority projects to improve sustainability in respiratory medicine.

## P121 USER DATA FROM A VALIDATED ONLINE INHALER TECHNIQUE COMPETENCY TEST: A NOVEL APPROACH TO STANDARDISED ASSESSMENT

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10.1136/thorax-2025-BTSabstracts.302

**Background** Correct inhaler technique is essential for effective management of airways diseases. Errors in technique remain common amongst patients and healthcare professionals (HCP). UK Inhaler Group (UKIG) has developed an inhaler technique framework and standards document to support competency training of HCPs.[1] We developed and validated an online Inhaler Technique Competency Test to offer a standardised and objective method of assessing whether HCP can identify correct/incorrect inhaler technique. The test features device-specific video scenarios aligned with UKIG standards. We aimed to assess the feasibility of this online assessment as an ongoing training and assessment tool.

**Methods** We engaged local HCPs to complete the assessment. The test required users to view ten short videos of people using inhalers and identify the embedded errors within. Video device scenarios included pressurised metered-dose inhalers (pMDIs), four different dry powder inhalers (DPIs), and soft mist inhalers (SMIs). Data was analysed over a 6-month pilot, including completion rates, user demographics and scoring.

**Results** Forty-three users completed the assessment with 88% passing (score $\geq$69%) on first attempt. Most users worked in hospital settings (65%), followed by community care (23%), general practice (7%), and community pharmacy (2%). Roles included pharmacists (30%), doctors (21%), allied health professionals (21%), nurses (16%), and others (12%). Highest

**Abstract P121 Table 1** Assessing errors made by inhaler device

| | No of videos | Total errors | Average no of errors per video | Most common assessing error made |
|---|---|---|---|---|
| SMI | 2 | 178 | **89** | Inspiratory manoeuvre for the device |
| MDI | 2 | 104 | **52** | Correct posture/ head position and mouth seal |
| DPI Spiromax® | 1 | 43 | 43 | Inhaler device and dose preparation assessing error was highest in Breezhaler® device; assessing the correct breath hold after inhalation being one of the next highest error made. |
| DPI Breezhaler® | 3 | 139 | 46 | |
| DPI Ellipta® | 1 | 7 | 7 | |
| DPI Turbohaler® | 1 | 52 | 52 | |
| DPI total | 6 | 241 | **40** | |

number of errors was found in assessing SMIs, followed by MDIs and then DPIs. Users made half the errors in assessing DPIs compared to SMIs. Users made the most errors in assessing device and dose preparation step, person's posture, head tilt, mouth seal and the correct inspiratory manoeuvre for the device. Notably, only 26% had attended training in the past year but thirty (70%) of users support patients with inhaler education at least monthly with a third doing it daily. See table 1 for errors made per device assessment.

**Conclusions** The e-inhaler technique competency assessment provides a scalable, standardised, and validated tool for assessing inhaler technique competency online. This tool has potential utility in education, clinical practice, and quality assurance, and may support improved outcomes through better targeted technique training.

## REFERENCE

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## P122 THE RELATIONSHIP BETWEEN BREATHLESSNESS AND LONELINESS IN INDIVIDUALS WITH CHRONIC RESPIRATORY DISEASE

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10.1136/thorax-2025-BTSabstracts.303

**Background** Breathlessness is one of the most common and disabling symptoms of chronic respiratory diseases such as COPD, asthma, and interstitial lung disease. It impacts physical activity, mental health and quality of life. Loneliness is also related to poor health outcomes, including increased healthcare use, morbidity and mortality. Qualitative research suggests breathlessness causes loneliness through reducing the quantity and quality of social activities, however quantitative research is lacking. We hypothesised that breathlessness would be associated with loneliness in people with chronic respiratory diseases.

**Methods** Using data from the Asthma + Lung UK 'Life with Lung Condition' survey 2025 we analysed the relationship between breathlessness (modified Medical Research Council (mMRC) dyspnea scale) as a continuous variable and a binary variable ($\geq$1 indicating breathlessness), with loneliness (3-item UCLA Loneliness Scale, continuous with 3 not lonely to 9 very lonely, and dichotomised with lonely $\geq$6). Associations were examined using linear and logistic regression models, adjusted for age, sex, ethnicity, income, and comorbidities.

**Results** A total of 9,350 participants (aged $\geq$18, with chronic respiratory disease) were included in the descriptive analysis (mean age: 65 years; 28.4% male; 91.6% White) and 6,034 in regression analyses. Higher levels of breathlessness were associated with loneliness: (coef: 0.439, 95% CI= 0.39 to 0.48, $p$ <0.001). In subgroup analyses, relationships were materially consistent in analyses of single conditions including COPD, asthma, bronchiectasis and ILD (coef: 0.334, 95% CI= 0.26 to 0.39, 0.447, 95% CI= 0.39 to 0.50, 0.358, 95% CI= 0.25 to 0.46, 0. 388 95% CI= 0.23 to 0.54, $p$ <0.001) These associations remained significant even after adjusting for potential demographic and clinical confounders.

**Abstract P122 Table 1** Regression analyses of the association of breathlessness and loneliness

| | Loneliness | | Loneliness | |
|---|---|---|---|---|
| | Coef (95% CI) | P | Odds Ratio (95% CI) | P |
| Model 1 without adjusting for covariates | 0.541 (0.505 to 0.577) | <0.001 | 2.49 (2.21 to 2.81) | <0.001 |
| Model 2 (adjusted for demographic covariates) | 0.479 (0.437 to 0.521) | <0.001 | 2.15 (1.84 to 2.50) | <0.001 |
| Model 3 (adjusted for demographic and clinical covariates) | 0.0439 (0.398 to 0.481) | <0.001 | 2.02 (1.73 to 2.35) | <0.001 |

**Abbreviations:** Coef: regression coefficient; 95% CI: 95% confidence interval.
Age, sex, ethnicity, and income were included as demographic covariates, having more than three comorbidities was included as a clinical covariate

**Conclusion** In adults with chronic respiratory disease, increased breathlessness was associated with higher levels of loneliness, suggesting that breathlessness may negatively impact aspects of social wellbeing such as loneliness. These findings highlight the need for holistic management and policy.

## P123 DOES ETHNICITY INFLUENCE THE INVESTIGATIVE PATHWAY AFTER PRESENTATION WITH BREATHLESSNESS? – AN EPIDEMIOLOGICAL STUDY USING CPRD RECORDS

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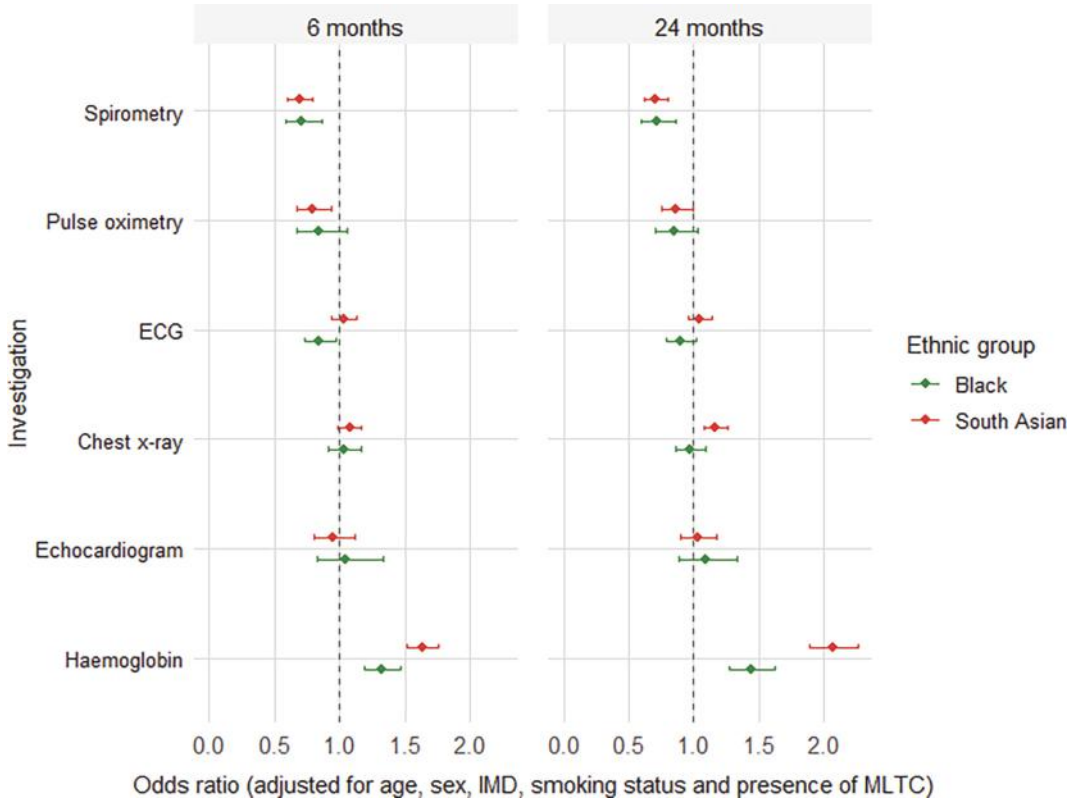
10.1136/thorax-2025-BTSabstracts.304

**Rationale** We previously found that patients of South-Asian and Black ethnicities were less likely to receive an explanatory diagnosis after presentation with breathlessness. As part of further exploration of the underlying reasons for these findings, we investigated the influence of ethnicity on recorded investigations after the point of first presentation with breathlessness

**Methods** Using the Clinical Practice Research Datalink (CPRD) GOLD database, linked with Hospital Episode Statistics, we identified adults with a first-recorded presentation (index date) with breathlessness between 2007 and 2017. The proportions of patients with a recorded code for possible investigations recommended by NHS England after presentation with breathlessness, within 6 months and 24 months of the index date, were identified. The six most frequently recorded investigations across the whole cohort were identified, and logistic regression was used to estimate adjusted odds ratios for receiving each of these across different ethnic groups, during the above timeframes. All models were adjusted for age, sex, index of multiple deprivation (IMD) quintile and presence of underlying multiple long-term conditions (MLTC) (defined as 2 or more long-term conditions).

**Results** We included 88,857 patients, of whom 3,336 were of South-Asian ethnicity and 1,506 of Black ethnicity. Patients with ethnicity coding of Mixed/Other/Unknown (n=12,512) were excluded as we were unable to ascertain their ethnic group with available data. Blood haemoglobin (Hb)(47%), chest x-ray (26%), electrocardiogram (ECG) (20%), spirometry (11%), pulse oximetry (7%), and echocardiogram (7%) were the most frequently recorded investigations within both 6 months, and 24 months of the index date. Compared to patients of White ethnicity, patients of South-Asian and Black ethnicities were significantly more likely to be investigated for Hb. However, both groups were significantly less likely to undergo spirometry or pulse oximetry, compared to patients of White ethnicity (figure 1).

**Conclusion** We observed significant differences in likelihood of some investigations across different ethnic groups, notably a significantly increased likelihood of Hb in patients of South-




**Abstract P123 Figure 1**

Asian and Black ethnicities but a significantly lower likelihood of spirometry and pulse oximetry. Whether these findings represent an inequity along the investigative pathway after presentation with breathlessness, or differences in clinical presentation, warrants further exploration.

## P124 INFLUENCE OF DEPRIVATION ON COPD PATIENTS WITH RESPIRATORY ACIDAEMIA REQUIRING NON-INVASIVE VENTILATION

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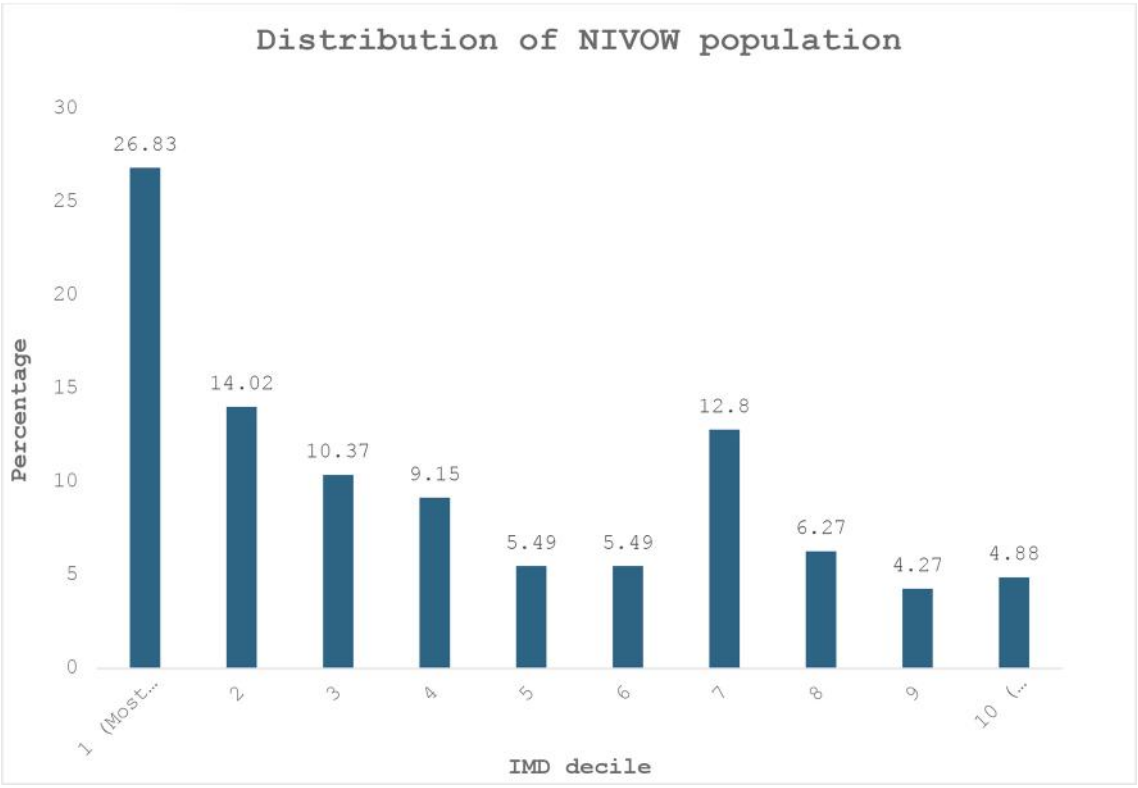
10.1136/thorax-2025-BTSabstracts.305

**Introduction and Objectives** Deprivation is strongly linked to COPD, exacerbations (ECOPD), hospitalisation and poor outcome. The non-invasive ventilation (NIV) outcomes score directed weaning trial (NIVOW: ISRCTN64639614) is a 7-centre RCT comparing two weaning regimes. We explored the impact of deprivation within the study population.

**Methods** Deprivation was assessed using the Index of Multiple Deprivation (IMD) and distribution by decile captured from the most (IMD 1) to least (IMD 10) deprived. Univariate and independent associates were identified, and potential influence of deprivation on length of stay during the index admission (LoS), 30-day readmissions, secondary care review for COPD, and in-hospital and 90-day mortality explored.

**Results** 84/164 (51.2%) participants were in decile 1–3, with 5.5 fold more participants in IMD 1 compared to IMD 10. Deprivation was associated with worse breathlessness and performance status eMRCD (**ρ** =-0.59, p=0.01), but better pre-NIV pH (**ρ**= -8.9, p = 0.04) and pre-NIV PaCO2 (**ρ** =0.19, **ρ**= 0.05), with a trend for more frequent ECOPD within the previous 12 months (**ρ**= -0.14, p = 0.06). Substance use was uncommon, but more prevalent among the most deprived: 7/11 (63.6%) in the lowest 3 deciles. There was no significant difference in outcomes by deprivation decile.

**Conclusion** Deprivation was strongly associated with ECOPD admissions requiring NIV across 7 UK centres. Higher deprivation was associated with worse breathlessness and performance status (eMRCD), and shows a trend toward increased exacerbation frequency. There was no association with FEV1% predicted, BMI, smoking or burden of comorbidities. More deprived participants had less severe respiratory acidaemia on admission which may reflect a less severe insult on a background of poorer performance status. Deprivation had no significant association with outcomes. Respiratory admissions in winter 2022/23 were 2.2 fold higher in IMD 1 (most deprived decile) compared to IMD 10.[1] In stark contrast there was a 5.5 fold difference in our population. There is an urgent need to address health inequalities in this large and disadvantaged population.




**Abstract P124 Figure 1**

## REFERENCE

1. Breathing issues are the leading cause of all emergency admissions | Asthma + Lung UK

## P125 DEDICATED RESPIRATORY SERVICE FOR ROUGH SLEEPERS

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10.1136/thorax-2025-BTSabstracts.306

**Introduction and objectives** Rough-sleepers **(RS)** are group of homeless people who sleep on the streets. Barriers to health care include drug and alcohol misuse, social isolation and mental health issues. There has been a 20% increase in the numbers of RS in England since 2023 and numbers are still growing.[1] The average age of death for a RS in the UK today is 45. One major cause of premature death is respiratory issues including pneumonia, TB and COPD.

To address these health inequalities and mitigate the impact of respiratory disease, the RS Virtual ward **(RSVW)** was set up in August 2024 to deliver care closer to the homeless, in a collaboration between Nottingham University Hospitals, Nottinghamshire Healthcare NHS Foundation Trust and Framework Housing Association, to improve access to both respiratory diagnostic and treatment services.

**Methods** We analysed RS hospital data pre and post RSVW across Emergency Department (ED), Inpatient (IP) and Intensive care (ITU) cohorts to evaluate the clinic's impact in reducing hospital admissions. Pre and post RSVW attendance were studied, comparing average days in ED, IP, and ITU across 8.5–10.1 years pre-RSVW versus post-RSVW data annualised to 1 year.

**Results** See table 1

The mean annualized number of days in hospital, either in ED, as an in-patient or on ITU was 0.92, 2.56 and 1.28 prior to introduction of the RSVW and 0.72, 1.55 and 0.88, post service introduction, a reduction of 22%, 40% and 32% respectively.

**Abstract P125 Table 1**

| | | |
|---|---|---|
| ***Total RSVW Patients*** | *74 (All cohorts combined)* | |
| ***DNA Rate*** | *22 (30%)* | |
| ***Age Range (Mean Age)*** | *30 – 62 years (46.3 yrs)* | |
| ***Gender*** | *Male: 50 (67.6%)* | *Female: 24 (32.4%)* |
| ***Average duration of pre-RSVW hospital data*** | *8 years* | |

**Conclusion** Rough sleepers utilize significant healthcare services. Respiratory problems are common, but also potentially preventable and treatable.[2] The implementation of a RSVW service significantly reduced hospital utilization (ranging from 21.7% to 39.5%) and hospital re-attendance (ranging from 75 to 92%) suggesting that an optimised service in the community can provide better healthcare access and outcomes to the underprivileged population, in turn saving substantial funds for the National Health Service. These outcomes support broader adoption of RSVWs.

## REFERENCES


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2. Homelessness and respiratory disease. *Lancet Respir Med*. 2013;**1**(3):e17. doi:10.1016/S2213–2600(13)70267–4


## P126 UNDERSTANDING THE ACCEPTABILITY AND PROCESS MECHANISMS OF A PATHWAY NAVIGATION INTERVENTION TO ENGAGE NON-RESPONDERS IN LUNG CANCER SCREENING


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10.1136/thorax-2025-BTSabstracts.307

**Background** Lung cancer has the highest mortality of all cancers in the UK. Targeted low-dose CT screening of asymptomatic high-risk individuals reduces mortality through early detection. However, the impact of national lung cancer screening programmes is limited by inequitable participation, particularly among those experiencing socioeconomic deprivation and with long-term tobacco dependence. Pathway navigation (PN) is an individualised approach, which employs trained navigators to guide individuals experiencing inequalities to overcome barriers to accessing healthcare, including screening. A randomised controlled trial assessed the effect of a telephone-based PN intervention on uptake of lung cancer screening by previous non-responders as part of the Yorkshire Lung Screening Trial. A mixed-methods process evaluation was conducted in parallel to understand the acceptability of the PN intervention, how it might work, barriers preventing participants from attending screening and the strategies adopted by pathway navigators.

**Methods** For the qualitative component, in-depth, remote, semi-structured interviews were conducted with participants purposively sampled from the PN trial intervention arm. Data collection was guided by the COM-B framework and analysed thematically using the framework approach.

**Results** Two groups were recruited based on lung cancer screening history, never responders (N=25) and previous disengagers (N=10). The PN intervention was viewed as highly acceptable across both groups. The personalised, nonjudgemental and actively empathetic approach of the PN team was described as essential in the decision to attend screening, working sometimes to facilitate arranging attendance for those already able and motivated to be screened and other times to navigate practical and emotional difficulties. Travel challenges, fear of lung cancer diagnosis and poor health were the primary barriers reported by participants. Age-related health decline, awareness of smoking as a risk factor for cancer, the perceived benefits of early detection, being reminded/prompted about screening and the offer of travel support motivated and enabled attendance.

**Conclusion** Non-responders found telephone-based PN support to be an acceptable and supportive approach, which acted to prompt, enable or motivate their attendance through bespoke practical and emotional support. Adopting pathway navigation for non-responders within the national lung screening programme may be a potentially powerful tool to help reduce the inequality gap and maximise effectiveness.

## P127 HEALTH INEQUITIES IN PREVENTIVE SCREENING: A QUALITATIVE EXPLORATION OF TLHC NON-ENGAGEMENT IN BAME COMMUNITIES


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10.1136/thorax-2025-BTSabstracts.308

**Background** The Targeted Lung Health Check (TLHC) screening programme aims to detect lung cancer in its early stages, particularly in high-risk populations, using low-dose CT screening. However, uptake amongst Black, Asian, and Minority Ethnic (BAME) communities remains disproportionately low in comparison to their White counterparts. This study explores the reasons behind non-participation in the TLHC screening programme among individuals from BAME backgrounds.

**Methods** A qualitative study was conducted in major English city using semi-structured telephone interviews with individuals from BAME communities who were eligible for TLHC screening but did not attend. Participants were identified using the TLHC/local GP practices' databases. 300+ non-responders were contacted, and 12 interviews were conducted. Thematic analysis was used to identify key barriers and perceptions influencing participation in the programme.

**Results** Several interrelated themes were identified. First, lack of awareness and understanding of the TLHC programme was widespread, where many participants were unfamiliar with its purpose or the fact that they were eligible/had received an invitation. Second, mistrust in healthcare systems – often rooted from previous negative experiences- undermined confidence in screening/using NHS services. Third, cultural and linguistic barriers which had meant limited access to information, with some participants expressing that materials were not culturally appropriate or available in their native languages. Fourth, fear and stigma around cancer, particularly lung cancer, which had led to avoidance and denial. Finally, practical constraints, such as work commitments, transport issues and caregiving responsibilities which had meant they were unable to attend the screening/clinics.

**Conclusion** Non-attendance at TLHC amongst BAME groups is influenced by a complex range of informational, cultural, structural, and emotional factors. Tailored interventions, such as culturally sensitive outreach, multilingual communication, and community-based education, would be beneficial to improve awareness, trust and access in these groups. Addressing these barriers could enhance engagement and reduce health disparities in lung cancer outcomes.

## P128 PATIENT- AND PHYSICIAN-REPORTED SYMPTOM BURDEN AMONG PATIENTS WITH BRONCHIECTASIS IN EUROPE

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10.1136/thorax-2025-BTSabstracts.309

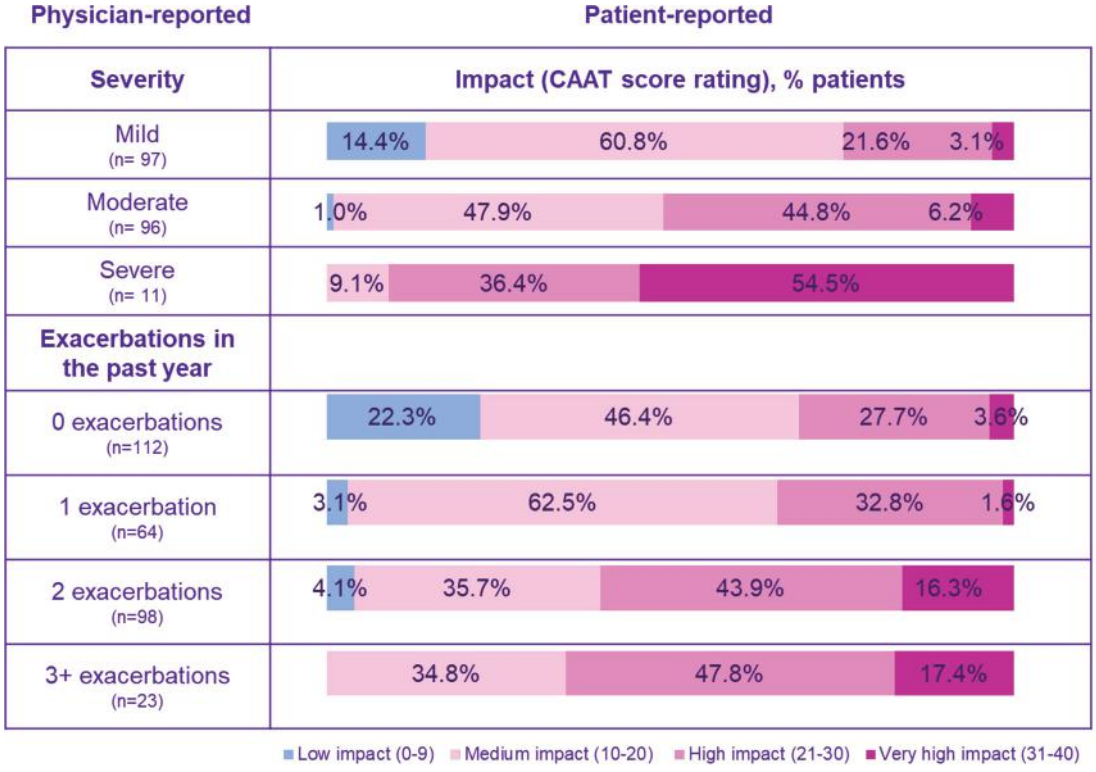
**Background** Alleviating symptoms is a treatment goal for bronchiectasis; however, few studies have explored symptom burden from patient and physician perspectives. This cross-sectional study describes the burden of bronchiectasis symptoms from the perspectives of both.

**Methods** Data were from the Adelphi Bronchiectasis Disease Specific Programme, a cross-sectional survey of patients and their physicians conducted from September 2024-March 2025 in France, Germany, Italy, Spain, and the UK. Surveyed patients aged ≥18 years had a physician-confirmed diagnosis of bronchiectasis (without cystic fibrosis). Surveyed primary care physicians (PCPs) or pulmonologists were responsible for ≥5 patients/month with bronchiectasis. Physicians completed patient record forms and the same patients independently filled out self-completion forms including Chronic Airways Assessment Tests (CAAT). Responses on current symptoms and impact were analysed and stratified by physician-reported exacerbation frequency (0, 1, 2, or 3+ in the past year).

**Results** In total, 59 pulmonologists and 58 PCPs completed 560 patient record forms, and 232 patients filled out self-completion forms.

Physicians reported most patients had symptoms, regardless of exacerbation frequency (89.3% of patients with 0 exacerbations up to 100.0% with 3+ exacerbations). The most impactful physician-reported symptoms (by pulmonologists and PCPs) across most exacerbation groupings were excessive sputum production, dyspnoea, and productive cough. Purulent sputum was in the top five symptoms reported by pulmonologists (26.0% of patients without exacerbations and 46.1% with 1 exacerbation), but not PCPs. Both PCPs and pulmonologists reported increased symptom burden with increased exacerbation frequency.

Among patients, 87.8% reported medium-to-very high impact of symptoms based on CAAT scores; the highest




Overall CAAT scores (0-40) were generated from eight domains (cough, phlegm/mucus, chest tightness, breathlessness on activity, activity limitation, confidence leaving house, impact on sleep, impact on energy). For each specific domain, scores range from 0-5, with 5 being highest impact. A higher overall score indicates a higher impact on the patient's life: low impact (0-9), medium impact (10-20), high impact (21-30), very high impact (31-40). CAAT scores are only reported for those who provided a score across each domain.

Exacerbations were defined in the survey as "events characterised by a worsening in the patient's symptoms for more than 48 hours. If the same pathogen has caused more than one exacerbation event, please mark this as a different exacerbation event". However, there could be variations in the definition of exacerbation used by physicians.

**Abstract P128 Figure 1** Patient-reported impact of bronchiectasis symptoms by Chronic Airways Assessment Test (CAAT) score by physician-reported symptom severity and physician-reported exacerbation frequency

impact symptoms were cough, mucus and breathlessness. Despite being rated as mild by physicians, 85.6% of patients reported that symptoms had a medium-to-very high impact (figure 1). 31.3% of patients with 0 physician-reported exacerbations, 34.4% with 1 exacerbation, and >60% with 2 or 3+ exacerbations reported high-to-very high impact symptoms (figure 1).

**Conclusions** Patients with bronchiectasis experienced symptoms even if they had no physician-reported exacerbations, and symptom burden increased as exacerbation frequency increased. Most patients reported medium-to-very high impact symptoms even where physicians rated their disease severity overall as mild, highlighting differences in perspectives of symptom burden.

## P129 IKEN-ILD: INVOLVEMENT, KNOWLEDGE, ENGAGEMENT IN INTERSTITIAL LUNG DISEASE (ILD) – A NOVEL PATIENT PASSPORT

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10.1136/thorax-2025-BTSabstracts.310

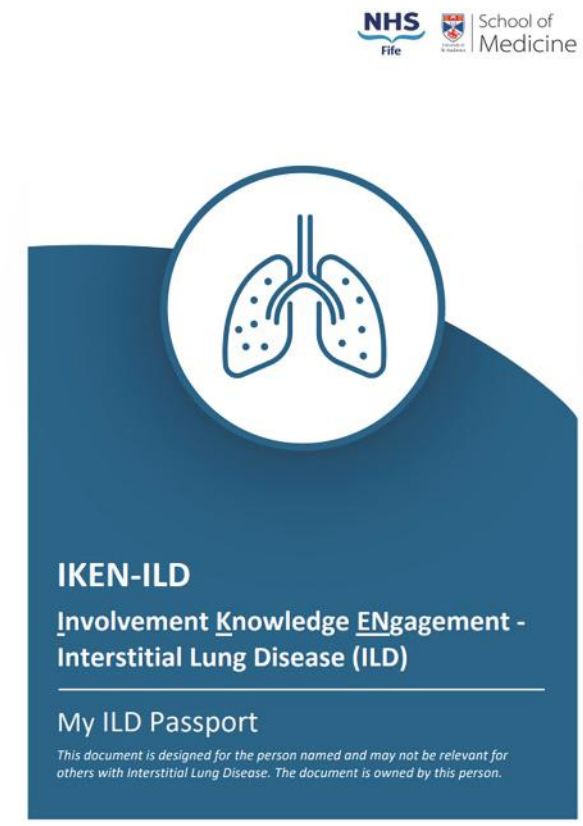
**Background** The cause of a patient's interstitial lung disease (ILD) may be challenging for clinicians to identify and diagnose and for patients to understand. ILD care necessarily demands multi-professional involvement of doctors, specialist nurses, physiotherapists, palliative care specialists, psychologists, pharmacists and dieticians. The complexity of diagnosis, treatment decisions and the breadth of clinical interactions means patients often have significant and rapidly changing information needs. Local patient advocacy groups raise this as a critical unmet need. They have requested clearer care plans, improved communication between professionals, and a recurring question of 'how do I manage my pulmonary fibrosis today?'.

**Aims** To co-design a patient passport to improve communication and care for patients with ILD. Our conceptual framework includes patient involvement, better understanding of diagnosis and management, and shared more visible communications with professionals.

**Methods** Co-design evolved over several meetings with clinicians and patient advocacy groups. An initial draft document underwent several iterations in response to wider patient feedback. The emerging passport was intended to be a live document, held and maintained by the patient. A questionnaire was simultaneously created, both online and in paper format, composed of 10 key co-developed questions to evaluate the value of the patient-passport.

**Results** The development of a new patient-passport, '**IKEN-ILD: I**nvolvement-**K**nowledge-**EN**gagement in **ILD**', including sections on the working-diagnosis, investigations, treatments, symptom management and anticipatory care planning. Section headings use simple, non-technical language capturing the patient's perspective (eg. 'My treatments', 'My wishes', 'My future'). The pilot passport and questionnaire are now being rolled out to our local ILD community. Responses will shape any future iterations.

**Discussion** The 'IKEN-ILD My ILD Passport' produced through co-design aims to meet the needs of the local ILD community in direct response to their call for improved care. Formal evaluation of key features around value, purpose and accessibility are being progressed and will inform the next passport iteration. Discussions to build a smartphone app version have been initiated. The goal remains to provide a live value-based tool for supporting the patient in understanding their diagnosis and care.




**Abstract P129 Figure 1**

## P130 IMPROVING RESPIRATORY OUTCOMES IN PEOPLE WITH LEARNING DISABILITIES: EVALUATION OF THE 'KEEPING MY CHEST HEALTHY' PATHWAY

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10.1136/thorax-2025-BTSabstracts.311

**Introduction** People with a learning disability experience disproportionately high rates of respiratory illness, premature death, more frequent and longer hospital admissions. The Keeping My Chest Healthy (KMCH) pathway was developed to address these inequalities by providing proactive, personalised respiratory risk management.

It incorporates the identification and management of modifiable risks using a structured scoring tool, which was refined following the British Thoracic Society Clinical Statement (2023). The pathway also includes accessible care plans and co-produced multilingual resources.

**Objective** To evaluate the impact of the KMCH pathway on emergency and unplanned respiratory-related healthcare usage among people with a learning disability.

**Method** A retrospective service evaluation was conducted comparing respiratory-related healthcare usage in the 12 months

**Abstract P130 Table 1**

| Outcome | Pre-KMCH | Post-KMCH | % Reduction |
|---|---|---|---|
| Days in hospital | 641 | 50 | 92.2% |
| Days in HDU/ICU | 40 | 11 | 72.5% |
| Hospital admissions | 29 | 7 | 75.9% |
| A&E attendances | 42 | 25 | 40.5% |
| Paramedic call-outs | 54 | 29 | 46.3% |
| GP consultations | 131 | 77 | 41.2% |
| Out-of-hours consultations | 20 | 12 | 40.0% |
| Antibiotic courses (chest infections) | 94 | 56 | 40.4% |

before and after pathway completion. Of 122 individuals who completed KMCH between 2022 and 2024, 46 met inclusion criteria (≥12-month follow-up). The cohort included individuals with degenerative or progressive conditions and those receiving palliative care but excluded individuals documented as being at end of life. Data were extracted from clinical records.

**Results** All outcome measures showed ≥40% reductions. Hospital bed days reduced by 92% (641 to 50), ICU/HDU days by 73% (40 to 11), and hospital admissions by 76% (29 to 7). A&E attendances, GP consultations, and antibiotic use each reduced by over 40%. None of the respiratory-related admissions were attributable to COVID-19.

Estimated cost reductions, based on NHS tariffs for primary admission reasons, included £2,614 in primary care, £123,104 in non-elective hospital stays, and £13,701 in emergency care during the 12 months following KMCH completion.

**Conclusions** KMCH was associated with marked reductions in unplanned respiratory-related healthcare use among people with a learning disability, suggesting potential for wider adoption.

As a retrospective evaluation without a control group, findings are descriptive and may have been influenced by factors such as seasonal variation, vaccination status, or wider respiratory illness patterns during the COVID-19 pandemic. Nonetheless, these findings support KMCH as a promising approach to reducing health inequalities and improving respiratory outcomes in this high-risk population. Further research, including prospective or controlled studies, is recommended to establish effectiveness, long- term cost benefits, and scalability.

## P131 SEVERE ASTHMA PATIENT PERCEPTIONS AND PREFERENCES ON THE FREQUENCY OF BIOLOGIC ADMINISTRATION; INCLUDING OPINIONS ON UPCOMING 6 MONTHLY BIOLOGIC

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**Introduction** Shared decision-making and patient preferences are often overlooked in severe asthma biologic selection, with very little published literature to help guide clinicians on patient preferences. This study aims to lend a voice to patients' views on biologic treatment frequency, especially as we proceed into a new era of an upcoming biannually administered asthma biologic.

**Methods** Quantitative data was collected via a questionnaire from a consecutive sample of adult severe asthmatics who were either about to commence their first biologic or already receiving biologic treatment between January and March 2025.

**Results** A total of 180 patient responses were collected and categorized into biologic-naïve (n = 95) and biologic-experienced (n = 85) groups. The demographic characteristics were consistent with those typical of a severe asthma population (table 1). Regarding treatment frequency preferences, 35% of biologic-naïve patients favoured monthly injections, whilst 39% had no preference, and 10% preferred a twice-yearly biologic regimen. Among biologic-experienced patients, a similar distribution was observed: 33% preferred monthly treatment, 28% had no preference, and 12% favoured a 6-monthly option. With regard to transitioning to a 6-monthly biologic, 48.5% of biologic-experienced patients expressed interest, 27.5% were not interested, and 24% had no preference. Biologic-naïve patients showed comparable responses, with 44% expressing an interest, 33% stating no interest, and 23% reporting no preference.

Across the entire sample, 41% of patients expressed concern about treatment response with a 6-monthly dosing schedule, compared to 28% for monthly biologic therapy. Concerns related to side effects, cost, and appointment accessibility were more frequently associated with monthly treatment.

**Conclusion** This study provides a previously unknown insight into severe asthma patient views on their preferred frequency of biologic administration and seems to suggets an appetite for engagement with an upcoming 6-monthly biologic Depemokimab.

**Abstract P131 Table 1**

| Demographics | | | | | |
|---|---|---|---|---|---|
| **Biologic treatment naïve (n=95)** | | | **Biologic treatment experienced (n=85)** | | |
| **Age (years) *** | 55 (± 14.9) | | **Age (years)*** | 53 (±15.2) | |
| **Female** | 61 (64%) | | **Female** | 53 (62%) | |
| **Asthma onset (n=87) **** | Early | 46 (53%) | **Asthma onset (n=83)**** | Early | 49 (59%) |
| | Adult | 22 (25%) | | Adult | 20 (24%) |
| | Late | 19 (22%) | | Late | 14 (17%) |
| **Smokers** | 4 (4.2%) | | **Smokers** | 1 (1.2%) | |
| **Employment status** | Full time employment | 33 (35%) | **Employment status** | Full time employment | 27 (32%) |
| | Part time employment | 11 (11%) | | Part time employment | 13 (15%) |
| | Retired | 32 (34%) | | Retired | 23 (27%) |
| | Student | 4 (4%) | | Student | 3 (4%) |
| | Other | 15 (16%) | | Other | 19 (22%) |
| **Biologic due to be commenced (n=92)**** | Benralizumab | 21 (23%) | **Current Biologic Treatment (n=84)**** | Benralizumab | 11 (13%) |
| | Dupilumab | 0 (0%) | | Dupilumab | 17 (20%) |
| | Mepolizumab | 17 (18%) | | Mepolizumab | 8 (9.5%) |
| | Omalizumab | 7 (8%) | | Omalizumab | 4 (5%) |
| | Reslizumab | 3 (3%) | | Reslizumab | 8 (9.5%) |
| | Tezepelumab | 44 (48%) | | Tezepelumab | 36 (43%) |
| **Results** | | | | | |
| **Concerns with taking monthly biologic treatment** | Long terms side effects | 22 (23%) | **Concerns with taking monthly biologic treatment** | Long terms side effects | 18 (21%) |
| | Side effects | 20 (21%) | | Side effects | 24 (28%) |
| | Treatment response | 22 (23%) | | Treatment response | 28 (33%) |
| | Cost to attend hospital | 4 (4%) | | Cost to attend hospital | 8 (9%) |
| | Availability to attend hospital | 6 (6%) | | Availability to attend hospital | 12 (14%) |
| | Other | 17 (18%) | | Other | 8 (9%) |
| **Concerns with taking 6 monthly asthma biologic treatment** | Long term side effect | 16 (17%) | **Concerns with taking 6 monthly asthma biologic treatment** | Long term side effect | 20 (24%) |
| | Side effects | 12 (13%) | | Side effects | 16 (19%) |
| | Treatment response | 43 (45%) | | Treatment response | 30 (35%) |
| | Cost to attend hospital | 2 (2%) | | Cost to attend hospital | 4 (5%) |
| | Availability to attend hospital | 5 (5%) | | Availability to attend hospital | 6 (7%) |
| | Other | 17 (18%) | | Other | 14 (16%) |
| **Asthma biologic administration preference** | Every 6 months | 10 (10%) | **Asthma biologic administration preference** | Every 6 months | 10 (12%) |
| | Every 2 months | 8 (8%) | | Every 2 months | 14 (16%) |
| | Once a month | 33 (35%) | | Once a month | 28 (33%) |
| | Once every 2 weeks | 7 (7%) | | Once every 2 weeks | 8 (9%) |
| | No preference | 37 (39%) | | No preference | 24 (28%) |
| **Interest in switching to 6 monthly biologic** | Extremely not interested | 6 (6.3%) | **Interest in switching to 6 monthly biologic (n=84)**** | Extremely not interested | 10 (12%) |
| | Not interested | 25 (26.3%) | | Not interested | 13 (15.5%) |
| | No preference | 22 (23%) | | No preference | 20 (24%) |
| | Interested | 25 (26.3%) | | Interested | 27 (32%) |
| | Very Interested | 17 (18%) | | Very Interested | 14 (16.5%) |

Data presented as no – (%) unless otherwise stated.

*Data presented as mean (±SD)

** Reduced sample sizes (n) indicate missing data due to non-response

## P132 ANTIFIBROTIC PRESCRIBING IN A TIER 2 CENTRE: THE ILD INTEGRATED CLINICAL CARE PATHWAY IN PRACTICE

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**Introduction** Nintedanib and pirfenidone reduce the rate of progression in fibrotic lung diseases. Until 2024 these medications could only be prescribed by tertiary specialist centres in England. We have piloted a tier 2 prescribing service in secondary care, in accordance with the objectives of the One Voice ILD integrated care pathway for optimum ILD care.[1] We report the outcomes from the first 6 months of antifibrotic prescribing in our trust.

**Methods** A retrospective review of patients discussed in the regional ILD MDT and given approval to start anti-fibrotic medications between December 2024 and June 2025. A patient satisfaction survey was sent to the first 53 surviving patients.

**Results** 62 patients were approved to start treatment. 48/62 patients were male, mean age 73 years (range 45–87). 55/60 started Nintedanib, 5/60 Pirfenidone. 1 patient declined treatment, 1 has delayed treatment start.

All had a consultation with an ILD specialist nurse and/or pharmacist. All patients had monthly blood tests as scheduled and received a follow up phone call within the first month.

At 3 months, 24/30 continued antifibrotic medication, 1 patient had died of progressive IPF, 4 had stopped due to side effects, 1 had treatment paused. Dose reduction was required in 17/60 patients. In comparison, of 39 patients from our trust who started antifibrotics in 2024, 30 remained on treatment at 3 months, 10 had dose reduction.

57/62 (92%) were approached about the BTS ILD registry, 26/62 have consented to participate thus far. 2 are actively considering clinical trials.

Patient satisfaction was high (survey response rate 24/53). All reported they knew who to contact with problems and found it 'quite easy' or 'very easy' to obtain advice on side effects. 21/24 patients preferred having all their care in-trust. 3/24 had no preference.

**Conclusion** Antifibrotics can be prescribed safely and effectively in Tier 2 centres. This approach provides care closer to home, improves continuity of care and monitoring with excellent patient satisfaction. Resources are needed to ensure access to other aspects of care, including research and registries.

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# Paediatric infection, inflammation and physiology

## P133 IMPACT OF ELEXACAFTOR/TEZACAFTOR/IVACAFTOR ON CLINICAL OUTCOMES AND QUALITY OF LIFE IN CHILDREN WITH CYSTIC FIBROSIS AGED 6–11 YEARS IN THE REAL-WORLD SETTING

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10.1136/thorax-2025-BTSabstracts.314

**Introduction** The TRAJECTORY Study, an ongoing real-world observational study, previously showed that elexacaftor/tezacaftor/ivacaftor (ELX/TEZ/IVA) improves clinical outcomes and

**Abstract P133 Table 1** Summary of Mean CFQ-R Domains at Baseline and Change from Baseline

| CFQ-R Domain[1] | Parents/Caregivers[2] of Children Aged 6-11 Years with at least 12 months post-ELX/TEZ/IVA initiation | | | | Patients Aged 6-11 Years[2] with at least 12 months post-ELX/TEZ/IVA initiation | | | |
|---|---|---|---|---|---|---|---|---|
| | n | Baseline[3] Mean (SD) | n | Average absolute change from baseline to 0-12 months[4] Mean (SD) | n | Baseline[3] Mean (SD) | n | Average absolute change from baseline to 0-12 months[4] Mean (SD) |
| Respiratory Symptoms | 107 | 76.8 (18.6) | 94 | +11.7 (20.6) | 107 | 77.0 (17.9) | 95 | +11.9 (16.4) |
| Body Image | 108 | 74.7 (30.2) | 95 | +7.3 (22.3) | 107 | 83.8 (22.0) | 94 | +3.2 (17.9) |
| Digestive Symptoms | 107 | 73.3 (20.3) | 94 | +10.0 (18.9) | 107 | 73.8 (27.9) | 95 | +8.8 (27.9) |
| Eating Problems | 108 | 72.2 (29.1) | 95 | +9.4 (25.6) | 108 | 79.2 (23.7) | 95 | +6.3 (20.7) |
| Emotional Functioning | 108 | 79.4 (15.9) | 95 | +4.1 (13.3) | 108 | 74.1 (13.8) | 95 | +3.7 (12.5) |
| Health Perceptions | 108 | 72.7 (21.4) | 94 | +7.8 (20.4) | | | | |
| Physical Functioning | 106 | 82.7 (8.4) | 93 | +8.8 (16.9) | 108 | 81.9 (18.9) | 95 | +7.1 (16.0) |
| Social Functioning | 108 | 74.0 (18.6) | 95 | +5.3 (16.6) | 89 | 68.2 (18.0) | 77 | +2.9 (16.0) |
| Treatment Burden | 108 | 54.4 (25.3) | 95 | +9.4 (21.0) | 106 | 65.6 (25.0) | 92 | +7.2 (23.8) |
| Vitality | 106 | 69.7 (14.7) | 93 | +3.9 (13.4) | | | | |
| Weight | 106 | 63.8 (37.1) | 93 | +14.3 (36.0) | | | | |

CFQ-R: Cystic Fibrosis Questionnaire – Revised; ELX/TEZ/IVA: elexacaftor/tezacaftor/ivacaftor; SD, standard deviation. [1]Please note that this is a medical chart abstraction study where the CFQ-R is collected as part of standard of care. Not all sites collected the CFQ-R as part of the standard of care. The counts vary by domain. CFQ-R domain scores range from 0-100, with higher scores indicating better health-related quality of life. [2]Parents/caregivers completed the proxy version of the CFQ-R for Parents/Caregivers of Children Ages 6-11 in which they answered for their children, while children were given the interviewer-administered CFQ-R for Children Ages 6-11 [3]Start of ELX/TEZ/IVA therapy is defined as the study baseline. [4]Average absolute change through the post-baseline is calculated as the average of all measurements during the post-baseline period minus baseline. Measurements within four weeks following ELX/TEZ/IVA initiation were excluded.

quality of life (QoL) in people with CF aged ≥12 years. We examine the effect of ELX/TEZ/IVA on health outcomes among children aged 6–11 years after 12 months of treatment.

**Methods** Medical record data was collected from 42 sites (United Kingdom, Canada, Germany, Italy, Portugal, Spain) for 12 months prior to (baseline) and up to 5 years after ELX/TEZ/IVA initiation (follow-up). Outcomes included body mass index (BMI) BMI z-score, percent predicted forced expiratory volume in one second ($ppFEV_1$), annualized event rates of pulmonary exacerbations (PEx) and hospitalizations, and QoL as measured by the Cystic Fibrosis Questionnaire-Revised (CFQ-R; Children Ages 6–11 [Interviewer-administered], Parents/Caregivers [proxy for children ages 6–11]). Descriptive analyses were stratified by prior CF transmembrane conductance regulator modulator (CFTRm) use and baseline $ppFEV_1$.

**Results** Among the 163 children aged 6–11 years who initiated ELX/TEZ/IVA between August 2019–June 2023 and had 12 months of follow-up, 49.7% (81/163) were female, 49.1% (80/163) were homozygous for *F508del-CFTR*, and 23.3% (38/163) had prior CFTRm use. At ELX/TEZ/IVA initiation, the mean (SD) age, BMI z-score, and $ppFEV_1$ were 8.2 (1.6) years, -0.25 (0.85), and 95.2 (14.9). Most had $ppFEV_1$ >90 (62.0%, 101/163) and $ppFEV_1$ ≥70 to ≤90 (31.9%, 52/163). At 12 months, ELZ/TEZ/IVA treatment led to mean (SD) increases in BMI z-score (+0.14 [0.45]) and $ppFEV_1$ (+8.7 percentage points [10.8]), and decreased annualized event rates of PEx (1.1 to 0.6) and hospitalizations (0.6 to 0.2).

All CFQ-R domains showed improvement through 12 months regardless of prior CFTR modulator use. Domains with the most improvement observed in mean (SD) change for parents/caregivers were Weight (14.3 [36.0]), Respiratory Symptoms (11.7 [20.6]), Digestive Symptoms (10.0 [8.9]), Eating Problems (9.4 [25.6]), and Treatment Burden (9.4 [21.0]) (table 1); consistent improvements were reported for children ages 6–11 in all domains.

**Conclusions** ELX/TEZ/IVA treatment showed improvement in function and nutritional status, and respiratory and non-respiratory CFQ-R domains for children aged 6–11 years after 12 months of treatment.

## P134 INTRODUCTION OF LUNG CLEARANCE INDEX (LCI2.5) MEASURED BY NITROGEN MULTIPLE BREATH WASHOUT (MBW) INTO CLINICAL PRACTICE

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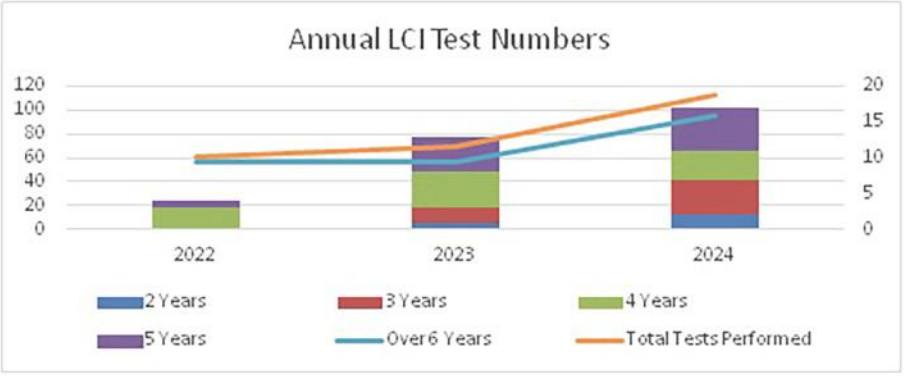
**Introduction** $LCI_{2.5}$ is used as an outcome measure in research trials for Cystic Fibrosis (CF) therapies, although use in routine clinical practice is limited. Spirometry is the most common lung function test is not always reliable in children under six years. Spirometry is thought to only detect moderate to severe lung disease, and therefore improvements in treatments for CF have normalised spirometry. LCI detects CF airways disease earlier than spirometry, allowing the opportunity for earlier intervention.

**Method** In Edinburgh, the paediatric clinical MBW service started in 2022, and there has been a yearly increase in the number of tests performed. Initially, patients aged 6–16 were seen. In 2023 after further training this reduced to aged 3 and by the end of 2023 to aged 2. This has early respiratory diagnostics in a patient group where spirometry is not possible. Annual test service data are shown in figure 1.

**Results** MBW in the group aged 2–5 years with CF has allowed the clinical team to measure the response to Elexacaftor/Tezacaftor/Ivacaftor (ETI) therapy using pre and post LCI in a patient group who were previously modulator naive. Baseline success rates in 2-year-olds seen (3) are 100% by using distraction techniques who had a mean baseline $LCI_{2.5}$ of 6.89 (6.25–7.91) only one of these has current LCI data which shows a fall in LCI post ETI.

In a group of 5 3-year-olds seen in 2024 pre ETI therapy $LCI_{2.5}$ mean 7.39 (6.79–8.85) and post mean 6.55 (5.94–8.29) showing a fall.

In the 6 years plus group annual and ad-hoc MBW is performed alongside spirometry. A group of 6–11-year-olds showed a baseline mean of 7.01 with a trend towards improvement in mean LCI post ETI therapy of 6.31.




**Abstract P134 Figure 1** Annual test service data

In both groups trend data is used to find out if there is an improvement or decline in LCI.

**Conclusion** The introduction of the LCI service clinically has allowed earlier access to diagnostic testing, treatment monitoring, treatment intervention and reduction in treatment burden, which previously would not have been possible.

## P135 PAEDIATRIC BRONCHIECTASIS – THE UNMET NEED FOR PSYCHOLOGICAL SUPPORT IN A TERTIARY CENTRE

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**Background** Bronchiectasis is characterised by a chronic wet cough, recurrent chest infections and abnormal bronchial dilatation. Previous work has shown that children with bronchiectasis have significantly worse self-reported and parent-perceived health-related quality of life, including symptoms of anxiety and depression. Childhood chronic illness affects the psychological well-being of primary caregivers.[1] The standard of care for patients with cystic fibrosis is to have access to a designated psychologist. The same standard is not afforded to patients with other long-term respiratory conditions.

**Method** We screened paediatric bronchiectasis patients using the Strengths & Difficulties Questionnaire (SDQ) during clinic visits. Socioeconomic status, hospital admissions, and antibiotic use were also recorded.

**Results** 17 patients completed the SDQ, mean age 11.29 years (SD 3.87), with 4 female (24%) and 13 male (76%). Most of the patients were in the most deprived deciles (IMD 1–4, 71%). 4 had admissions for IV antibiotics in 2024 and 14/17 had at least one course of oral antibiotics (mean 2.7 courses, min 0, max 6).

Most participants scored within the average range on the SDQ (70%). A proportion had borderline (12%) or above average (18%) scores, indicating possible emotional or behavioural difficulties. Five children scored on the impact scale (1 borderline; 4 above average), suggesting these issues were affecting their daily lives. Among the three children who scored high on both the SDQ and impact scales, none had required hospitalisation, but two had received multiple courses of antibiotics.

**Conclusions** The SDQ is a screening tool which identified a possible unmet psychological need in 30% of our patient population, the same proportion that has been found in other national screening studies.[2] This highlights the need for improved psychological support for children with bronchiectasis and their families.

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## P136 CLINICAL CHARACTERISTICS OF A COMBINED ADULT AND PAEDIATRIC COHORT OF PATIENTS WITH PRIMARY CILIARY DYSKINESIA

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**Introduction** Primary ciliary dyskinesia (PCD) is an underdiagnosed orphan lung disease. Late diagnosis results in poor respiratory outcomes. Understanding the local population is essential to guide diagnosis and treatment. We describe clinical characteristics of an adult and paediatric population with PCD.

**Methods** Retrospective observational study, clinical and radiological data, pulmonary function and results of other diagnostic testing were collected from patient electronic records.

**Results** In total 37 patients were identified [17 paediatric:20 adult]. Age range 4 to 37 years; 19/37 (51%) patients were female. Mean age at diagnosis was 10 in the adult cohort and 3.7 years in the paediatric patients. 43% had situs inversus and 46% had hearing loss. 73% had cilial analysis performed. Of those tested 74% Class 1 defect; 22% had a Class 2 defect. Genetic testing was performed [in all but one]; 65% having a pathogenic variant identified. In adults, median FEV1% in patients with mucus plugging on CT was 63% (IQR 36% to 69%) versus 69% (IQR 65.5% to 105%) in those with no mucus plugging ($p=0.045$). Median FEV1% in adults with inflammatory nodules was 63% (IQR 39% to 69%)] versus 85% (IQR 66% to 106%) in those without ($p=0.011$). Positive microbiology and lower lung function was also more likely in adults with CT changes compared to adults with no mucus plugging or inflammatory nodules. Median FEV1% in adults with Pseudomonas aeruginosa 42% (IQR 26% to 52%) and children with Pseudomonas aeruginosa 79% (IQR 66% to 93%) was significantly lower than patients culturing other organisms, FEV1% was 67% (IQR 50% to 70%, $p=0.034$) in adults and 89% (IQR 77% to 97%, $p=0.012$) in children.

**Conclusion** The implementation of an early diagnostic pathway in paediatrics has resulted in patients being identified around 6 years earlier compared to a historical adult cohort, highlighting the need for investment in a robust diagnostic process. Lung function was low from a young age and lower in those with active infection and inflammatory changes on CT. These findings suggest that age of diagnosis is improving and that a delay in diagnosis and treatment results in significant lung function decline and respiratory morbidity.

## P137 EXPLORING PARENTAL EXPERIENCES AND COPING WITH HAVING CHILDREN WITH PRIMARY CILIARY DYSKINESIA: A QUALITATIVE SECONDARY DATA ANALYSIS

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10.1136/thorax-2025-BTSabstracts.318

**Background** Primary Ciliary Dyskinesia (PCD) is a chronic condition that causes reduced mucociliary clearance and mainly affects the respiratory system. PCD currently has no cure and is managed by daily chest physiotherapy and frequent hospital appointments with varying specialties. Paediatric patients are often fully supported by a parent or caregiver which can have significant impacts on parents' lives primarily in the psychosocial and physical wellbeing domains.
**Aims** To explore parents' experiences of having children with PCD focusing on coping mechanisms, parental adaptation and barriers to coping
**Methods** I performed secondary data analysis on anonymised transcripts (N=26) of interviews exploring parental quality of life of having children with PCD and coded that data using NVivo. I followed this with inductive reflexive thematic analysis to analyse the impact the diagnosis had on parents' lives and the function of the different coping mechanisms that were utilised.
**Results** Three key themes were identified: parents' adapting narratives, facilitators of coping and barriers to coping. All parents recognised that upon experiencing uncertainty, the formation of a routine was essential to coping and accepting the condition as a part of life. Majority of parents acknowledged the idea of teamwork being a fundamental factor in sharing the burden associated with caring for their child and creating a reliable support network. Parents also responded to their child's diagnosis with hypervigilance consisting of meticulous monitoring and striving for mastery of condition.
**Conclusion** Reducing perceived treatment burden and enabling self-efficacy were factors associated with improved parental adaptation and had facilitated coping. Collaborative approaches to managing care by relying on teamwork with partners and healthcare professionals were shown to reduce anxiety and was a sustainable coping strategy. Parents acknowledging their stressors and managing their anxiety could help improve affected parental mental health since this was identified as a key barrier to coping.

## P138 OUTCOMES OF CHILDREN UNDERGOING CLINICALLY INDICATED BRONCHOSCOPY FOR SEVERE RECURRENT RESPIRATORY SYMPTOMS

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10.1136/thorax-2025-BTSabstracts.319

**Introduction** Severe childhood wheeze, cough and lower respiratory tract infections (LRTI) are often managed empirically with inhaled corticosteroids (ICS) and/or antibiotics based on symptoms alone. We investigated the following outcomes associated with targeted treatment determined by bronchoalveolar lavage (BAL) results: episode frequency, prescribed treatment, diagnosis and long-term outcomes in children undergoing clinically indicated bronchoscopy.
**Methods** Retrospective case-note review of 116 children aged 1–17 years who underwent elective, clinically indicated bronchoscopy between 2016–2024 for severe wheeze/asthma, recurrent cough and/or LRTI.
**Results** 70/116 (60.3%) children were preschool age (<6 years). 74/116 (63.8%) children had BAL-positive bacterial cultures; 52/70 (74.3%) were preschool and 22/46 (47.8%) were school-age. BAL bacterial pathogens differed significantly between preschool and school-age: *Moraxella catarrhalis* (33.3%), *Haemophilus influenzae* (29.3%) and *Streptococcus pneumoniae* (17.3%) were most common in preschool, while *Staphylococcus aureus* (36%) was most common in school-age children. All BAL bacterial positive children received a minimum of 2 weeks targeted antibiotics post-bronchoscopy. Rhinovirus, enterovirus and adenovirus were the most detected viruses across all ages.

12-month follow-up data were obtained from 70/116 (60.3%) children. Median annual episodes reduced from 6 pre-bronchoscopy, to 2 in 75% of preschool children, and from 6.5 to 3.5 in 25% of school-age children. 17/49 (34.7%) preschool children had symptom improvement/resolution ($P<0.0001$) in the 12 months after bronchoscopy; 12/49 (24.5%) had new diagnoses of aspiration syndromes. In all children 12 months post-bronchoscopy, prophylactic antibiotic use increased from 18/70 (25.7%) to 35/70 (50%) ($P<0.01$); aspiration as an indication increased from 0% to 8/35 (22.9%) ($P<0.05$).

110 children were followed up to final/most recent visit (median 33 months post-bronchoscopy). LRTI diagnoses decreased from 72/110 (65.5%) pre-bronchoscopy to 22/110 (20%) by the final visit ($P<0.0001$). Asthma/wheeze diagnoses increased from 4/110 (3.6%) to 16/110 (14.5%) at the last visit ($P<0.0001$). 14 patients had aspiration diagnoses by the final visit compared to none pre-bronchoscopy ($P<0.0001$). 44/97 (45.4%) children were discharged after median follow-up of 17.5 months.
**Conclusion** Bronchoscopy with BAL informs targeted treatment in children with complex respiratory symptoms and LRTI diagnoses. Nearly half of patients achieved symptom resolution and were discharged from tertiary care at approximately three years after the procedure.

## P139 DETERMINANTS OF TIDAL VOLUME IN NEPALI INFANTS LESS THAN 6 WEEKS OF AGE: A CROSS-SECTIONAL STUDY

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10.1136/thorax-2025-BTSabstracts.320

Neonates and infants are more susceptible to lung diseases influenced by age. Spirometry measures lung volume changes to diagnose and monitor respiratory conditions. Children experience significant lung development. Reference values for tidal volume, especially in neonates, are limited in countries like Nepal due to a shortage of trained personnel and the scarcity of advanced spirometers. This study examines tidal breathing pulmonary function in neonates under six weeks and its determinants.

A cross-sectional study was conducted at a tertiary hospital in southwestern Nepal from January 1 to December 31, 2023. Various factors related to babies and mothers in post-natal wards and NICU were documented. Infant pulmonary function testing, specifically tidal volume measurement, was performed on infants less than six weeks old.

A total of 866 babies were included; 55.3% (n=479) were male. Most were term (84.9%), with 15.1% preterm (n=131). The mean birth weight was 2773 ± 302 g.

The mean tidal volume (VT) in this study was 19.25 ml, and the volume per kilogram (VT/kg) was 7.08 ml/kg. No significant differences in VT ml (p=0.34) or VT ml/kg (p=0.89) were observed by sex or delivery mode. Infants weighing 2500 grams or more had significantly higher tidal volumes (p=0.02) and volumes per unit weight (p=0.045) than those under 2500 grams. No maternal factors—such as sleep duration, folic acid intake, smoking, diet, primiparity, hypertension, religion, or oligohydramnios—affected VT ml or VT ml/kg. However, significant differences were seen in VT ml and VT ml/kg among mothers with adequate iron and calcium intake during pregnancy.

This study aims to provide baseline data on tidal volumes for Nepalese infants, which will inform further studies. Birth weight and gestational age were significant neonatal factors influencing tidal volume. Iron and calcium intake by mothers during pregnancy had a significant influence on the development of higher tidal volumes in babies.

## P140 HOME SPIROMETRY TO OPTIMISE RESPIRATORY SECONDARY CARE NEW PATIENT APPOINTMENTS

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10.1136/thorax-2025-BTSabstracts.321

Home spirometry (HS) can provide longitudinal evidence of variable airflow obstruction, reversibility and obstruction during symptomatic episodes to support respiratory diagnosis and treatment optimisation. The utility of home spirometry for children aged 5–18 y has not been widely reported. One study has suggested asthma overdiagnosis and underdiagnosis is potentially as high as 15% and 40% respectively in primary care (PC).[1]

**Methods** The aim of this study was to determine if the waiting time between referral and first appointment could be

**Abstract P140 Table 1** Demographics and results

| | Referred | Onboarded |
|---|---|---|
| n | 25 | 22 |
| male/female | 64% / 36% | 68% / 32% |
| Age (mean (SD), range) | 9.5y (2.5y), 5.5y - 14.1y | 9.5y (2.6y), 5.5y - 14.1y |
| Index of multiple deprivation (median (range)) | 8 (2-10) | 8 (4-10) |
| Weeks from referral to consultant appointment (mean (SD), range) | | 12 (2), 8-15 |
| Assessment length (mean, SD) | | 9 (2), 6-13 |
| N spirometry tests (mean (SD), range) | | 26 (11), 3-41 |
| FeNO at clinic, <35ppb/ >35ppb | | 57% / 43% |
| % with obstructive spirometry (home) | | 17, 77% |
| % with obstructive spirometry (one clinic test) | | 12, 62% |

utilised to provide enhanced clinical information (more than a single spirometry and FeNO value) to facilitate respiratory diagnosis and treatment. Children and young people (CYP) aged 5–16 y referred from PC to a respiratory specialist consultant were offered a home spirometry assessment during the 8–12 week waiting period between referral and clinic appointment. Spirometry was coached by videocall with a paediatric respiratory physiologist.

**Results** Demographics are shown in table 1. 22 of 25 referred children were onboarded and recorded on average 26 spirometry tests each (target 4–5 tests weekly). HS supported a diagnosis of asthma in 76% of children. 77% of children had evidence of obstruction on HS despite a normal clinic FeNO in 57% (<35ppb). In 5 there was obstruction when symptomatic but normal clinic spirometry, 3 of which also had a normal clinic FeNO result.

3/22 children had normal spirometry and were discharged (not asthma). 9/22 children were confirmed as asthmatic with evidence of variable airflow obstruction and/or a current steroid inhaler, enabling treatment optimisation. The secondary care team reported that fewer attempts were required to record spirometry and FeNO in the clinic than those not referred for HS, further improving the efficiency of secondary care appointments.

**Conclusion** Home spirometry can be a beneficial way to collect information relevant to diagnosis and treatment in CYP, reducing misdiagnosis and improving the speed and accuracy in confirming or disproving asthma and streamlining secondary care clinical assessment.

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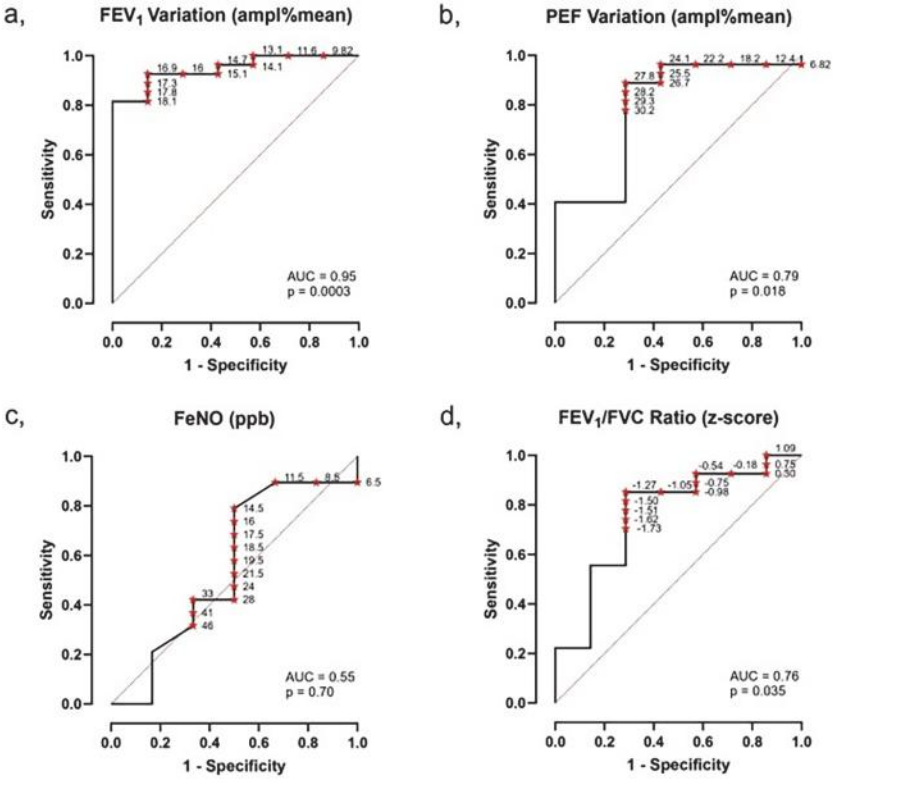
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## P141 THE UTILITY OF HOME SPIROMETRY MONITORING IN CHILDREN WITH RESPIRATORY SYMPTOMS

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**Introduction and Objectives** Misdiagnosis of paediatric asthma is a well-recognised problem. Current NICE guidelines recommend single occasion tests which may not represent a child's full clinical picture. Monitoring lung function variation over time provides clinicians with additional information. This can be done using home spirometry, but little is known about its role as a diagnostic tool.




**Abstract P141 Figure 1** Receiver operating characteristic (ROC) curves for a, FEV1 variation (ampl%mean) b, PEF variation (ampl%mean) c, FeNO and d, FEV1/FVC ratio (z-score). P-value and area under the curve (AUC) for each test is written in the bottom left of the graphs. Numbers in the graph represent the associated cut-off values for the different parameters with the same units as listed in the graph titles. The red dotted line represents an AUC of 0.5 and is the diagnostic accuracy equivalent to that of an algorithm based on chance

We hypothesised that lung function variation has greater diagnostic accuracy than single occasion tests. This study aimed to assess the sensitivity and specificity of lung function variability over a 4-week monitoring period compared to single occasion tests.

**Methods** In this retrospective cohort study, children aged 5–16, referred to hospital asthma clinics for diagnostic confirmation or management optimisation, carried out supervised home spirometry for a 4-week period using a NuvoAir spirometer. Forced expiratory volume in 1 second/forced vital capacity ($FEV_1$/FVC) ratio, $FEV_1$ and peak expiratory flow (PEF) were measured 4 or 5 times weekly and when symptomatic. FEV1 and FVC were graded using the American Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines. Baseline FeNO was assessed at the first clinic. A paediatric respiratory clinician used all available data to determine diagnosis. The diagnostic accuracy of baseline $FEV_1$/FVC z-score and fraction of exhaled nitric oxide (FeNO) and variation of $FEV_1$ and PEF were calculated using receiver operating characteristic (ROC) curves.

**Results** 43 children were recruited with 34 (mean age 9.97 years, 20 male) producing sufficient home spirometry for inclusion in the analysis. The 'confirmed asthma' group (27) had lower baseline lung function values but only home spirometry results were significantly lower than the 'no asthma' group. $FEV_1$ variation had the highest area under the curve (AUC) of 0.96 (p=0.003) and would have correctly diagnosed 19/25 children with asthma. FeNO had the lowest (0.55, p=0.7) and would have only correctly diagnosed 8/25 children with asthma. Sensitivity and specificity based on ERS/ATS cut-offs for $FEV_1$ variation were 1.00 and 0.14 compared to 0.42 and 0.67 for baseline FeNO.

**Conclusion** Home spirometry measurements have greater utility for diagnosing asthma in children than single occasion testing. Further research is needed to assess the utility and cost-effectiveness of home spirometry.

## P142 HOSPITAL ATTENDANCE FOR RECURRENT PRESCHOOL WHEEZE IS NOT DETERMINED BY DISEASE SEVERITY

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**Introduction** Preschool wheeze is a common cause of emergency department (ED) attendance consuming substantial healthcare resources. Unfortunately, there is little evidence that self-management strategies are effective in preventing ED visits (BTS/SIGN Guidelines 2019).

**Method** We collected and analysed demographic and treatment data for children aged 1–5 years attending our ED with acute wheeze between December 2022 - November 2023 and all reattendances for the year following their first attendance. Data included an index of multiple deprivation based on post code (IMD score) and the treatment given for all attendances.

**Results** 664 children (64% male) attended over the one year period. 226 of these children (33%) reattended with wheeze at least once within the subsequent year accounting for 472 additional ED attendances [twice: 120 children (18%), x3: 50 (7%), x4: 18(3%), x5: 18(3%), x6: 10(1%), >6: 10(1%)]. Children who were reattenders were decreasingly likely to be treated with repeated doses of inhaled salbutamol (burst treatment either by nebuliser or metered dose inhaler) or oral steroids when compared with children who did not reattend (see table 1).

IMD score was not associated with frequency of readmission. Similarly, IMD score was not predictive of disease severity as indicated by treatment need or the need for hospital admission after ED attendance for more than 4 hours [Score 1–3 (most deprived) 57% admitted, score 4–6 - 63%, score 7–10 - 54%].

**Discussion** Pre-school children presenting with acute wheeze in our locality have a 1 in 3 chance of reattending with the same diagnostic code within the next 12 months. Our data highlights how reattendances might not be driven by disease severity. Supportive care measures that address parental anxiety about the severity of symptoms might help families to safely manage recurrent wheezy episodes without the need for hospital attendance.

## P143 CHILDHOOD ASTHMA AND AIR QUALITY CONVERSATIONS: COMMUNITY PHARMACY INTERVENTION CONVERSATIONS

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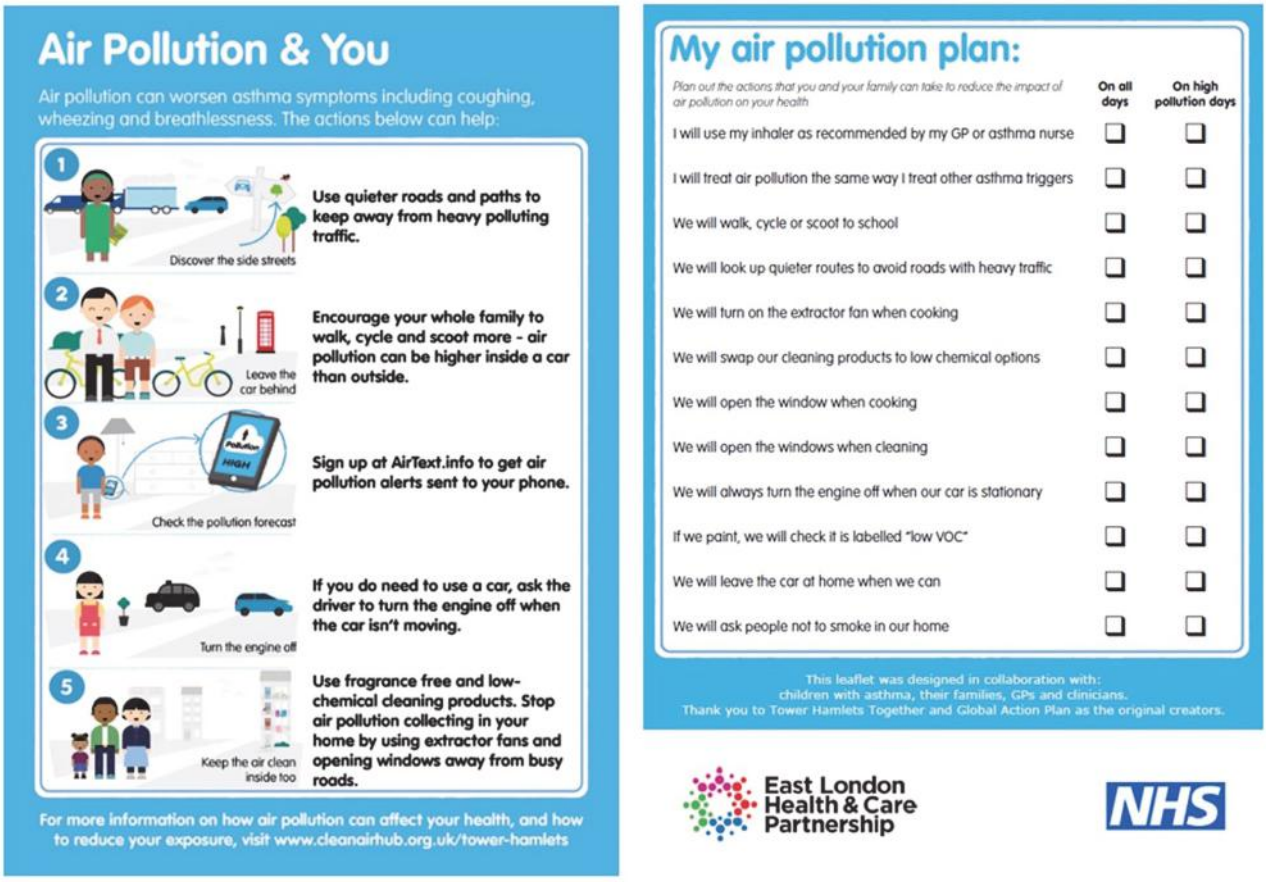
10.1136/thorax-2025-BTSabstracts.324

**Introduction** Air pollution harms health, especially for children with asthma. In the UK, it contributes to 30,000 deaths annually and costs the economy £27 billion annually. Newham is in the top 10 places in the UK for air pollution, and the worst in North East London (NEL) with over 10 child deaths relating to asthma in NEL since 2017, and the highest proportion of high-risk asthma patients in NEL.

This project aimed to evaluate the impact of a pharmacist-led consultations with patients and their representatives in Newham.

**Abstract P142 Table 1**

| No of admissions over 12 months | 1 | 2 | 3 | 4 | 5 | 6 | >6 |
|---|---|---|---|---|---|---|---|
| Received Burst Treatment | 277/438 (63%) | 95/240 (40%) | 28/150 (32%) | 18/72 (25%) | 16/90 (18%) | 9/60 (15%) | 10/86 (12%) |
| Received Oral Steroids | 154/438 (35%) | 76/240 (32%) | 41/150 (27%) | 17/72 (24%) | 12/90 (13%) | 10/60 (17%) | 10/86 (12%) |




**Abstract P143 Figure 1** 'Air Pollution and You' leaflet

**Methods** Community pharmacists completed opportunistic, or GP-referred conversations with high-risk children under 18 prescribed an inhaler (based on inhaler and medication use/prescriptions). Conversations included inhaler education including use and their carbon footprint alongside pollution exposure. Resources used included the 'Air Pollution and You' leaflet and DEFRA's air aware tool.

A retrospective cross-sectional study assessed implementation at participating pharmacies. Quantitative data was recorded in the pharmacy collection tool PharmOutcomes from November 2023 to May 2025; with patient representative feedback via Microsoft Forms survey collected January-May 2025.

**Results** From 602 interventions recorded on PharmOutcomes, 39% of these were referrals from primary care. From 602, 41% had been prescribed >three SABA inhalers in the past year, with 35% being under 8-years-old. In all consultations inhalation technique was checked, with corrections identified in 84% of conversations.

From 75 survey responses 67% found both the discussion about the leaflet plus having inhaler technique checked useful. From the leaflet, using quieter roads was agreed by all, 81% encouraged less use of the car, 73% agreed to turn the car engine off when not moving and change cleaning habits. Notably, 93% agreed the actions improved asthma symptoms.

**Conclusions** Community pharmacist-led consultations support patients understanding of asthma and its management including the impact of air pollution. Patient representatives and patients are keen to make simple changes if they are educated to do so, to improve control, symptoms and outcomes. Further interventions by community pharmacists would be beneficial to support patients and the wider healthcare system.

## P144 WHAT INFORMATION SHOULD A PAEDIATRIC ASTHMA DISCHARGE LETTER CONTAIN – A DELPHI STUDY

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10.1136/thorax-2025-BTSabstracts.325

**Background** Despite established standards for ward and ED discharge letters, the quality of paediatric asthma discharge letters remains variable, and information can lack salient details, including management and follow-up. There is a need for a consensus on the information and structure required for discharge letters to ensure safe and effective handover of care to the primary care clinician, child's school or parent/carer.

**Aim** To establish agreed core elements for standardised paediatric asthma discharge letter template.

**Method** The National Respiratory Audit Programme (NRAP) Children and Young People Asthma Team used the Delphi method, comprising of multiple survey rounds, to secure a consensus from subject experts. We recruited clinicians from all healthcare settings involved in paediatric asthma care directly and via national groups: British Paediatric Respiratory Society, British Thoracic Society, Association of Respiratory Nurses, Primary Care Respiratory Society, School and Public

Health Nurses Association, National Paediatric Respiratory and Allergy Nurses Group, and teams participating in the National Respiratory Audit Program audit. For Patients and Public Involvement and Engagement, we consulted the RCPCH.

**Initial Results** 55 participants, including clinicians (general practitioners, general and respiratory paediatric consultants, nurses, pharmacists), and parents/carers were asked to identify the top 3 discharge elements out of a list of 25, which were established during initial open consultation with stakeholders. Each choice was then graded by the level of importance on a scale of 1–5. 71% of participants identified red flags (e.g. previous hospitalisation, high salbutamol use), as the most important element, followed by treatment plan (31%), record of previous exacerbations/hospitalisations (31%), inhaler technique check and provision of asthma action plan (27%), hospital treatment (27%), clear diagnosis (22%), and instructions for GP (24%). The remaining elements were scored as of lower relevance, with less than 20% of participants voting for them.

**Conclusions** This initial DELPHI round to develop best practice standards for CYP with Asthma identified patient red flags as the most important core element of the discharge letter; further rounds are required to reach consensus on the other elements, which will inform the outline of the discharge letter template.

## P145 IMPACT OF BREATHING PATTERN INTERVENTIONS ON BREATHING PATTERN DISORDERS IN TEENAGERS WITH POST-COVID SYNDROME

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10.1136/thorax-2025-BTSabstracts.326

**Introduction** Post-COVID syndrome (PCS) is defined as the persistence of physical symptoms 3-months after an initial COVID-19 infection that cannot be explained by alternative causes. Nearly half of young people with PCS experience breathlessness,[1] which may be linked to breathing pattern disorders (BrPDs) caused by autonomic nervous system disturbances.[2]

**Aims** To determine incidence of BrPD diagnosis at rest and during exercise and explore the impact of breathing retraining on BrPDs in teenagers with PCS.

**Methods** Prospective analysis as part of a broader study in PCS (IRAS: 315063). Participants underwent cardiopulmonary exercise testing (CPET) with perceived breathlessness scores (BORG), spirometry, gas transfer and breathing pattern assessment by a physiotherapist at baseline (T1) and invited for breathing pattern retraining intervention. Assessments were

**Abstract P145 Table 1** Participant demographics and assessment data

| | Total n = 32 | Group 1: ≤2 physiotherapy appointments n = 12 | Group 2: 3≤ physiotherapy appointments n = 18 |
|---|---|---|---|
| **Demographics** | | | |
| Sex (male) | 11 (34.4%) | 3 (25%) | 8 (44.4%) |
| | **Mean (SD)** | **Mean (SD)** | **Mean (SD)** |
| Age | 15.31 (1.73) | 14.85 (1.82) | 15.61 (1.5) |
| BMI | 21.16 (4.17) | 22.04 (4.61) | 20.53 (3.83) |
| FEV1 (% predicted) | 94.20 (11.99) | 92.39 (10.41) | 90.66 (14.63) |
| FVC | 91.39 (20.29) | 96.04 (12.24) | 87.80 (24.59) |
| Gas transfer (TLCO SB % predicted) | 94.54 (13.17) | 96.59 (15.75) | 92.01 (12.39) |
| **Physiotherapy assessment** | | | |
| T1 Diagnosis of DB by physiotherapist | 27 (84.4%) | 11 (91.6%) | 16 (88.9%) |
| | **Mean (SD)** | **Mean (SD)** | **Mean (SD)** |
| T1 Nijmegen score | 33.13 (9.78) | 34.17 (10.77) | 32.44 (9.33) |
| T2 Nijmegen score | 25.60 (8.25) | 26.58 (9.86) | 24.94 (7.22) |
| **CPET data** | | | |
| T1 diagnosis of DB at rest | 29 (90.6%) | 12 (100%) | 17 (94.4%) |
| T1 diagnosis of DB during exercise | 24 (75%) | 9 (75%) | 15 (83.3%) |
| | **Mean (SD)** | **Mean (SD)** | **Mean (SD)** |
| T1 breath frequency at rest | 19.31 (4.71) | 18.5 (5.75) | 19.78 (4.22) |
| T2 breath frequency at rest | 17.9 (4.72) | 18.5 (5.65) | 17.5 (4.12) |
| T1 resting BORG | 1.1 (1.07) | 1.25 (1.11) | 1.15 (1.07) |
| T2 resting BORG | 1.11 (1.06) | 1.60 (1.34) | 0.89 (0.82) |
| T1 CPET duration | 9.32 (2.51) | 9.33 (2.90) | 9.28 (2.37) |
| T2 CPET duration | 9.8 (2.62) | 9.33 (2.64) | 10.11 (2.63) |
| Improvements seen during CPET on breathing pattern from T1 to T2 | 21 (65.6%) | 10 (83.3%) | 11 (61.1%) |

repeated after six-months (T2). To assess the impact of number of physiotherapy appointments, participants were classified in two groups: ≤2 or ≥3 appointments. BrPDs on CPET were diagnosed if there was evidence of hyperventilation (increased breathing frequency or tidal volumes), stunted increase in tidal volume or erratic breathing pattern in response to exercise. Improvement in any of these BrPD parameters was assessed at T2. Parametric and non-parametric statistical analysis were undertaken as appropriate.

**Results** 32 teenagers were recruited, 21 (66%) female, mean age 15.3 (SD 1.73) with mean BMI 21.2 (SD 4.2). All had normal spirometry and gas transfer. Assessment at rest confirmed BrPD in 27 (84%) participants via physiotherapy assessment and 29 (91%) via CPET at T1. BrPD was agreed in 26 (82%) participants via both assessments. Only one participant completed 6 physiotherapy appointments, the median 3 (range 0–6) appointments.

Nijmegen scores ($p<0.001$) and improvement in CPET parameters ($p<0.05$). Reduced resting breath frequency, pre-test BORG scores and increased CPET duration were observed in all subjects but did not reach significance in those who had more physiotherapy appointments (table 1).

**Conclusions** BrPDs are a predominant cause of breathlessness in teenagers with PCS. Further studies are required to determine whether the improvement seen after 6 months is due to BrPD intervention or effect of time.

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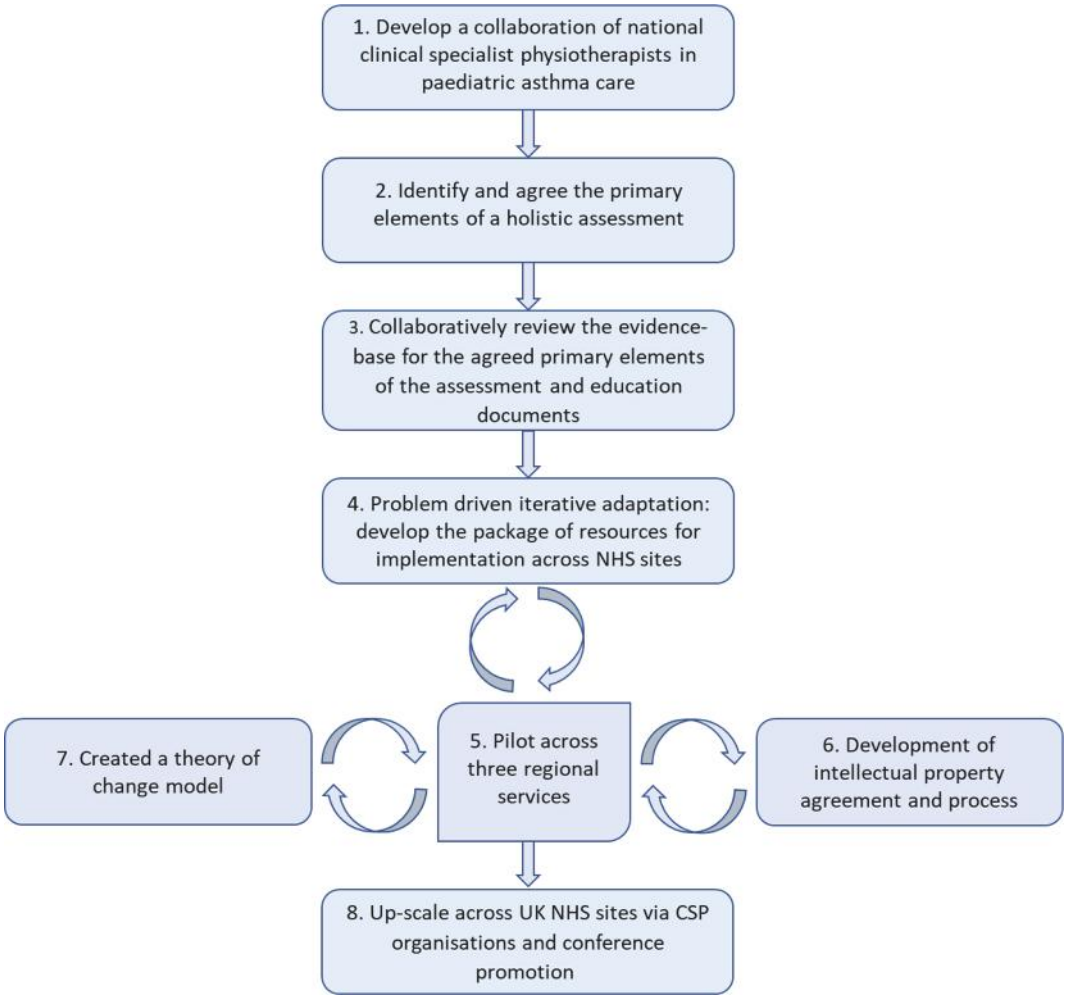
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## P146 DEVELOPMENT AND IMPLEMENTATION OF A PHYSIOTHERAPY STANDARDISED ASSESSMENT PACKAGE FOR CHILDREN WITH ASTHMA OR DYSFUNCTIONAL BREATHING ACROSS THE NHS

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10.1136/thorax-2025-BTSabstracts.327

**Introduction** Asthma diagnosis and management are evolving with the introduction of the BTS/NICE guidelines (2024) and the Asthma Capability Framework. There are inconsistencies across NHS paediatric physiotherapists in assessment and treatment of asthma and the nuanced analysis of respiratory symptoms.[1] This creates disparity in service provision at the detriment of patient outcome. To improve care for children and young people (CYP) across the NHS, there is a pressing need for national data collection and the development of consistent, evidence-based physiotherapy services.




**Abstract P146 Figure 1** Project flow diagram

The aim of this project was to design a standardised assessment and supporting educational package for physiotherapists working in paediatric asthma and dysfunctional breathing (DB) services.

**Methods** Figure 1. depicts the development and dissemination process.

**Results** Kids' Cornerstone Collaborative (KCC), a group of national physiotherapy specialists in paediatric asthma care, was created with the aim of developing staff, promoting consistent care across the NHS, and initiating broader collaborations and comprehensive big data collection.

An evidence-based physiotherapy assessment pack was developed which includes:

- Implementation playbook to support sites to embed the assessment in their clinical services
- Paediatric asthma and DB interactive assessment proforma in word, PDF, and Microsoft Forms versions
- Paediatric asthma and DB assessment supplementary education document
- Competencies document to guide the development of physiotherapists - NHS band 5 to 8

Intellectual property agreements were created across inventor NHS sites to enable free access and use of the resources created. An implementation pilot was conducted across three regional sites, with problem-based iterative adaptations throughout, supporting the refinement of Theory of Change (TOC) model. Disseminated nationally via clinical interest groups and national conferences.

52 secondary and tertiary care NHS Trusts spanning all UK regions requested access to this resource package; 25 completed a user agreement and have received the assessment package.

**Conclusion** A standardised physiotherapy assessment package for CYP with asthma and/or DB across the NHS was successfully developed and disseminated. Future work will review implementation of this package, biannual content review, and researching the impact of the package on staff competencies and outcomes for CYP.

## REFERENCE

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## P147 A NOVEL APPROACH TO THE USE OF AN ADJUSTABLE ONE-WAY VALVE IN PAEDIATRIC TRACHEOSTOMY PATIENTS WITH COMPLEX AIRWAYS: A CASE SERIES

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**Introduction and objectives** There are many benefits to one-way valve use including restoring physiological PEEP, improving postural tone, vocalisation and re-sensitisation of the upper airway. Our organisation adopts a multidisciplinary pathway to establish appropriateness for one-way valve assessment. Our clinical experience shows that a small

**Abstract P147 Table 1** Demographic and Transtracheal Pressure measurements: TRACOE® Phon Assist I one-way valve vs closed bias Valve

| Case Identifier | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| **Age at valve tolerance** | 2years 6months | 11 months | 4 years | 5 years | 4 years | 2 years | 8 years | 18 weeks CGA |
| **Weight at assessment** | 12.5kg | 11.6kg | 14.8kg | 16.2kg | 21.4kg | 10.7kg | 26.8kg | 3795g |
| **Gestation at birth (weeks)** | $29^{+2}$ | $39^{+2}$ | $24^{+4}$ | $36^{+4}$ | 24+4 | $26^{+2}$ | $24^{+3}$ | $26^{+2}$ |
| **Sex** | M | M | M | M | M | F | F | F |
| **Ethnicity** | White British | White British | White British | White British | Black African | White British | Mixed | White British |
| **Tracheostomy details** | 3.5 paed uncuffed | 4.0 neo uncuffed | 4.0 paed uncuffed | 4.0 paed uncuffed | 4.5 Paed uncuffed | 4.0 neo uncuffed | 4.5 paed uncuffed | 3.0 neo uncuffed |
| **Airway pathology** | Subglottic stenosis, abnormal vocal cord movement | Vocal cords fixed paramedian position | Bulbar palsy | Subglottic stenosis – multiple dilatations | Subglottic stenosis, tracheomalacia with anterior collapse | Subglottic stenosis | Subglottic stenosis | Cervical Teratoma Tracheomalacia |
| **Initial MLB findings** | Flickering vocal cords, laryngospasm causing false cords | Abnormal vocal cord tone - twitching | Normal | Subglottic stenosis 25-40% | Subglottic narrowing Grade 1, Mild bronchomalacia | Suproglottic cord oedma Subglottic stenosis 93-71% | Grade 3 subglottic stenosis | Grade 4 view of larynx due to mass (excised prior to one way valve trial) |
| **Tracheostomy details** | 3.5 paed uncuffed | 4.0 neo uncuffed | 4.0 paed uncuffed | 4.0 paed uncuffed | 4.5 paed uncuffed | 4.0 neo uncuffed | 4.5 paed uncuffed | 3.0 neo uncuffed |
| **TTP ($cmH_2O$) TRACOE® Phon Assist I one-way valve** | <5 | <5 | 5-10 | <10 | 0-5 | 4-5 | 0-5 | 0-3 |
| **Valve closure (full or partial or open)** | Full | Full | Full | Full | Full | Full | Full | Full |
| **TTP ($cmH_2O$) closed bias valve** | 10-20 | 15-20 | >20 | 20 | 20 | >10 | 15-20 | 10-20 |

proportion of our caseload do not tolerate closed bias valves. We present 8 paediatric cases and evaluate the tolerance and patient experience in terms of vocalisation/communication, tracheobronchial secretion management and feeding/swallowing when wearing the Atos Medical TRACOE® Phon Assist I one-way valve.

**Methods** Data was collected for 8 paediatric patients, demographics are detailed in table 1. Additional data was collated from the clinical assessment including Transtracheal pressure (TTP)manometry. TTP was compared between the two valve types. Likert rating scales and a semi structured parent/carer interview followed by thematic analysis.

**Results** Of the 8 sets of data for children using the TRACOE® Phon Assist I one-way valve, one was excluded from full analysis as they were transferred to a different tertiary hospital. Our data showed where a closed bias-valve resulted in high TTP and non-tolerance, use of the TRACOE® Phon Assist I one-way valve resulted was well tolerated (table 1).

All parent/carer responses reported a positive change in communication/vocalisation. The top ranking theme (86%) was that the vocalisation was louder with the valve on. Six (86%) responses reported a positive change in tracheobronchial secretion management. No (0%) responses reported a change in oral feeding. The top ranking theme stated by 6 (86%) was that the child was tube fed.

**Conclusions** The findings of this study show that for paediatric patients who do not tolerate closed bias one-way valve, that the TRACOE® Phon Assist I one-way valve provides a safe and well tolerated alternative. The infinitely adjustable side openings allow adjustment of breathing resistance which facilitates airflow through the upper airway and out the side openings aiding tolerance. Our small but complex population of paediatric tracheostomy patients who do not tolerate closed bias one-way valves, can now to gain the benefits associated with use of one-way valves by using the TRACOE® Phon Assist I one-way valve.

## Risk predictors and diagnostics in COPD

### P148 AI-DRIVEN IDENTIFICATION OF HIGH-RISK COPD PATIENTS FOR BIOLOGIC THERAPY: PATHWAY DEVELOPMENT OPPORTUNITIES

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10.1136/thorax-2025-BTSabstracts.329

**Background** Biologic therapies targeting eosinophilic inflammation hold promise for COPD management. Realising their benefits will require effective patient identification and pathway development to improve access. AI-based risk prediction models offer a novel approach to stratify patients and optimise treatment delivery.

**Methods** Using de-identified routine clinical data from Glasgow SafeHaven, we established a cohort of c38,000 patients with a coded COPD diagnosis. AI-based models were applied to the 2021 dataset, to identify 3,639 patients at highest risk of hospital admission (6 months) or mortality (12 months). Among these, 382 patients had an eosinophil count >300 cells/μL in the prior 12 months despite triple inhaler therapy, suggesting eligibility for biologic treatment.

**Results** The high-risk group's adverse deprivation demographics mirrored COPD burden in the wider population. Most biologic-eligible high-risk patients were aged >60 years and resided >5 km from central hospital sites where biologic therapy is typically initiated. However, a high proportion live <5 km from community vaccination hubs, presenting an opportunity to adapt treatment initiation locations. Based on RCT data, a projected reduction of 520 hospital admissions/year could be achieved in our organisation if biologic therapy could be provided to this highest risk cohort.

**Conclusion** AI-driven risk prediction enables targeted identification of COPD patients who may benefit from biologic therapy. Model derived insights can support pathway reconfiguration to improve access and equality, particularly via decentralised treatment initiation, facilitating timely intervention and better outcomes.

### P149 THE LANDSCAPE OF MARKETED AND PIPELINE DIAGNOSTIC TESTS FOR CHRONIC LUNG CONDITIONS

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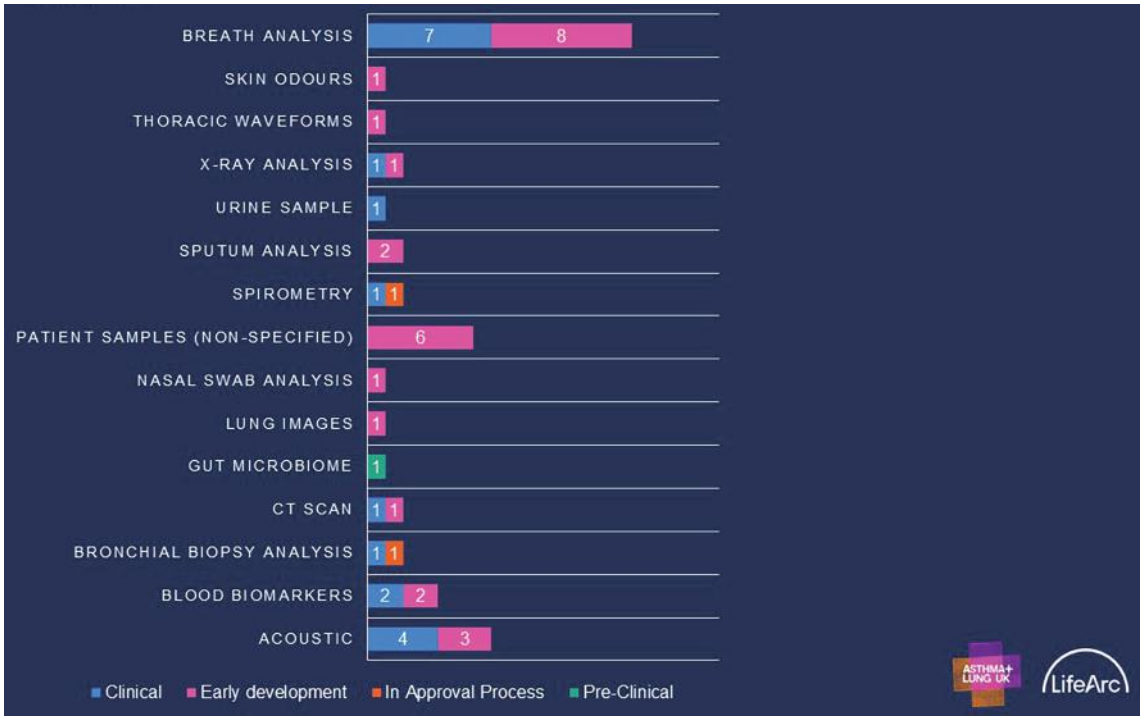
One in five people in the UK will develop a lung condition in their lifetime, often facing years before diagnosis. In 2023, LifeArc and Asthma + Lung UK held a workshop to identify essential diagnostic tests needed to transform respiratory care. A key priority is developing tools for timely and accurate diagnosis in primary care or expedited triage to secondary care—a 'Simple Lung Test.'

Landscaping was conducted to develop a patient-focused Target Product Profile (TPP) for a Simple Lung Test. The aim was to base the TPP on the current diagnostic landscape, unmet clinical needs, and innovation opportunities in respiratory diagnostics. By mapping existing technologies, this work seeks to guide innovation, avoid duplication, and highlight promising innovations for diagnosis and patient triage.

For the four priority chronic respiratory diseases (asthma, chronic obstructive pulmonary disease [COPD], bronchiectasis [BE], and interstitial lung disease [ILD]), 138 approved tests were identified, about half being lung function tests, primarily spirometers. Also approved are several breath analysis tests, such as FeNO and capnography tests, alongside in-vitro tests for measuring patient biomarkers, and lung imaging software.

48 tests specifically aiming to provide diagnosis are in development, covering a broad range of modalities, and mostly for providing diagnosis of COPD or asthma. The pipeline has moved away from traditional lung function tests, focusing instead on novel biomarkers, reflecting the limitations of lung function tests.

Key areas of innovation include novel breath analysis tests and acoustic measurement tests, both showing active pipelines in clinical and preclinical stages. Artificial intelligence is a recurring theme for enhancing data analysis and interpretation for novel acoustic tests, blood biomarker tests, and breathomic approaches.




**Abstract P149 Figure 1** The pipeline of diagnostic tests for specified lung conditions currently in development

These promising technologies offer putative advantages over current diagnostic tests, such as effort independence, rapid results, and improved accessibility, aligning with clinical priorities for novel tests.

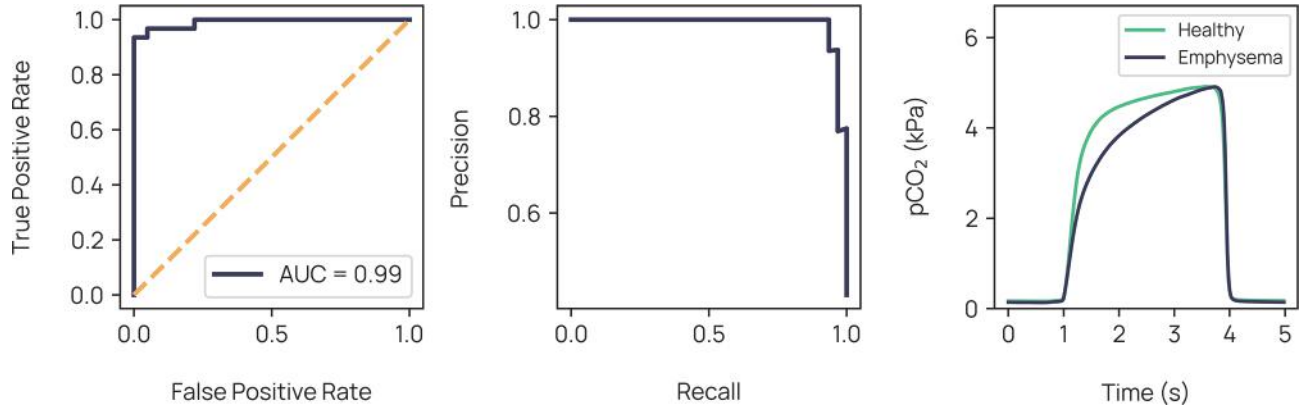
## P150 DIAGNOSTIC CLASSIFICATION OF THE EMPHYSEMA SUBTYPE OF COPD USING N-TIDAL® CAPNOGRAPHY

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10.1136/thorax-2025-BTSabstracts.331

**Introduction and objectives** Chronic obstructive pulmonary disease (COPD) consists of two main phenotypes: emphysema-predominant and airway-predominant COPD. The reference standard for diagnosing emphysema is the high-resolution computed tomography (HRCT) scan. However, its widespread use is challenged by cost, diagnostic availability, and exposure to radiation. The development of easy-to-use and non-invasive diagnostic methods is therefore necessary to detect emphysema in patients with COPD. A proposed method is capnography, using the TidalSense N-Tidal® handheld capnometer, which has already been proven to be an accurate test for distinguishing the severity of COPD through quantification of changes in physiologic dead space and alveolar compliance.

The objective of this research was to accurately differentiate emphysema-predominant COPD from healthy controls by applying interpretable machine learning (ML) techniques to a




**Abstract P150 Figure 1** Receiver operating characteristic (ROC, left figure) and precision-recall (PR, middle figure) curves for the logistic regression model. The right figure shows the normalised average waveforms (averaged across the cohort) for emphysema-predominant COPD patients and healthy controls

75-second tidal breathing $CO_2$ capnogram captured using the N-Tidal® capnometer.
**Methods** 31 patients with emphysema-predominant COPD (collected on the Exploring Novel Biomarkers for Emphysema Detection (ENBED) study, NCT05825261) and 41 healthy controls (HC) were recruited. HRCT was performed in all patients to confirm emphysema, with% emphysema ranging from 3% to 45%. Low complexity discriminative machine learning models were trained on 93 features extracted from the breath waveform. Leave-One-Out Cross-Validation (LOOCV) was used to evaluate model performance.
**Results** Logistic regression classification yielded the following metrics: AUROC 0.991, sensitivity 0.936, specificity 0.976, positive predictive value (PPV) 0.967 and negative predictive value (NPV) 0.952. The primary waveform features driving classification were derived from the alpha angle region, which represents the transition of alveolar gas to larger airways. This is consistent with hypothesis based on the pathophysiologic changes that are known to occur in emphysema. Features from this region also correlated with FEV1/FVC (highest $r$ = 0.61), supporting their hypothesised role as markers of obstruction.
**Conclusions** This initial dataset has shown that the N-Tidal® device can be used alongside interpretable ML algorithms to accurately distinguish emphysema-predominant COPD patients from healthy subjects.

## P151 HOME HIGH-FLOW THERAPY: INSIGHTS FROM REMOTE-MONITORING AND LINKED ROUTINE CLINICAL DATA

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10.1136/thorax-2025-BTSabstracts.332

**Background** We initiate home high-flow therapy (HFT) on case-case basis for patients with COPD who have severe dyspnoea, recurrent exacerbations or emerging hypercapnic respiratory failure (not requiring or not tolerating home NIV) despite optimised management. Remote monitoring including patient-reported outcomes (PROs), clinical events, asynchronous messaging and device usage has been incorporated using our COPD digital service and connected LumisHFT devices (Airview platform, ResMed) to support therapy provision and clinician workflow.
**Methods** A retrospective electronic health record (EHR) and remote-monitoring data review was conducted for 58 COPD patients who commenced home HFT between 2018–2024, with data censored February 2025.
**Results** Annual respiratory-related hospital admissions decreased by one per patient, with a reduction of 14 occupied bed days per patient per year in the subgroup of patients who used home HFT. Median time to readmission or death was 21 weeks in non-users and 52 weeks in HFT users. Exploration of remote-monitoring insights at a per patient level noted improvement in PROs (CAT score, EQ5D5L) and reduction in antibiotic and prednisolone courses following HFT initiation. Patients typically used HFT more frequently during the early adoption phase, and reduced their usage as their condition improves.
**Conclusion** Reduction in healthcare resource utilisation in this observational cohort adds to the evidence-base for home HFT as a valuable intervention for selected patients with COPD. Remote monitoring insights can enrich our understanding of patient and service benefits, and support adoption of this respiratory support modality.

## P152 ADMISSION BLOOD EOSINOPHIL LEVELS IN PATIENTS WITH COPD REQUIRING ACUTE NON-INVASIVE VENTILATION (NIV)

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10.1136/thorax-2025-BTSabstracts.333

**Background** Blood eosinophil counts are increasingly used to guide corticosteroid therapy in stable COPD, but their role in acute exacerbations requiring non-invasive ventilation (NIV) remains unclear. Identifying biomarkers predictive of NIV need could inform earlier interventions and improve outcomes.
**Aim** To assess whether admission blood eosinophil levels differ between patients with acute COPD exacerbations who require NIV and those who do not.
**Methods** A retrospective case-control audit was conducted at a single UK centre in 2024–2025. Thirty-three patients were included: 18 required NIV (NIV group), and 15 did not (control group). Groups were matched for age and smoking status. Admission blood eosinophil counts (within 6 hours) were compared. Additional data included systemic steroid use and prior admissions.
**Results** All patients were on optimised inhaler regimens, with similar smoking status, comorbidities, and inhaler use across groups. Systemic corticosteroid use, common in both groups before or during admission, likely affected eosinophil counts, potentially masking baseline inflammation. Although the control group had a slightly higher average number of hospital admissions in the past year, the clinical relevance is unclear.

**Abstract P152 Table 1**

| Parameter | NIV (n=18) | Control (n=15) |
|---|---|---|
| Mean eosinophil count ($\times 10^9$/L) | 0.144 | 0.153 |
| Median eosinophil count ($\times 10^9$/L) | 0.10 | 0.20 |
| Eosinophils <0.1 $\times 10^9$/L (%) | 39 | 33b |
| Eosinophils ≥0.3 $\times 10^9$/L (%) | 22 | 27 |
| Systemic steroid use (%) | 83 | 87 |
| Mean admissions in past year | 2.2 | 2.7 |

**Conclusions** Lower eosinophil levels were more frequent among patients requiring NIV, suggesting a possible association with a neutrophilic, steroid-insensitive phenotype. While not statistically significant, admission eosinophil count may serve as a rapid, accessible tool to aid early risk stratification. Larger prospective studies are needed to validate its predictive utility.

## P153 ASSOCIATES OF, AND TIME TO RECOVERY FROM, EOSINOPENIA IN SEVERE EXACERBATION OF COPD (A-TREC)

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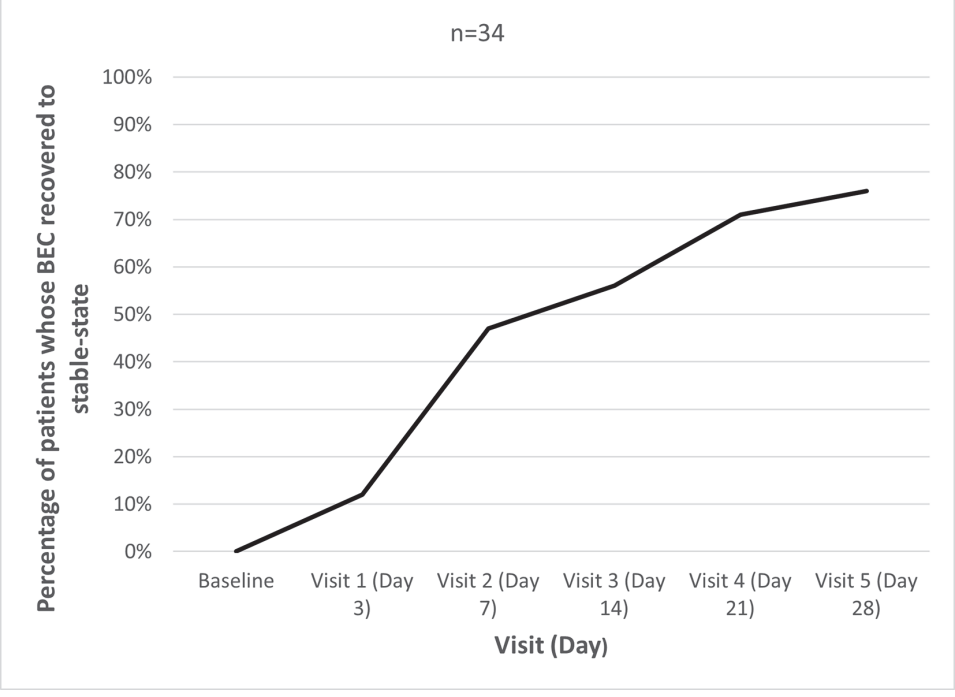
10.1136/thorax-2025-BTSabstracts.334

**Introduction** Blood eosinophil count (BEC) guides selection for inhaled corticosteroid (ICS) and emerging biologics in COPD. Neither GOLD nor NICE specify measuring BEC during clinical stability. 50% of patients admitted with severe exacerbation of COPD have transient eosinopenia (BEC <50 cells/uL).[1]

**Aims** This preliminary analysis from the A-TREC study (clinicaltrials.gov NCT06188065) examines time to recovery in BEC in the subgroup with admission eosinopenia and an uneventful recovery; this will not alter target recruitment. We aim to recruit 200 patients to identify 69 with admission eosinopenia and an uneventful recovery.

**Methods** Patients with a severe exacerbation of COPD and admission eosinopenia were assessed with measurement of BEC on days 3, 7, 14, 21, 28 and 42 following admission. Patients with an eventful recovery (further systemic corticosteroids or emergency hospital admission during follow up) were excluded. Stable-state was defined as day 42 BEC.

**Results** 102/113 patients have completed follow up; 34 had admission eosinopenia with an uneventful recovery. Mean (SD) age 72 (9) years, FEV1 39% (22%) predicted, median (IQR) eMRCD score 4 (4–5). 59% were female, 44% current smokers and 5.9% on LTOT. 26/34 (76%) recovered to stable-state BEC, and 32/34 (94%) crossed the 100 cell/uL threshold by day 28. 10/34 patients (29%) crossed the 300 cell/uL by day 28, with 1 further patient crossing by day 42. Only 7/34 (21%) and 3/34 (9%) crossed a new BEC treatment threshold beyond day 14 and 21 respectively. Additional data will be available for presentation at the BTS Winter Meeting.




**Abstract P153 Figure 1**

### REFERENCE

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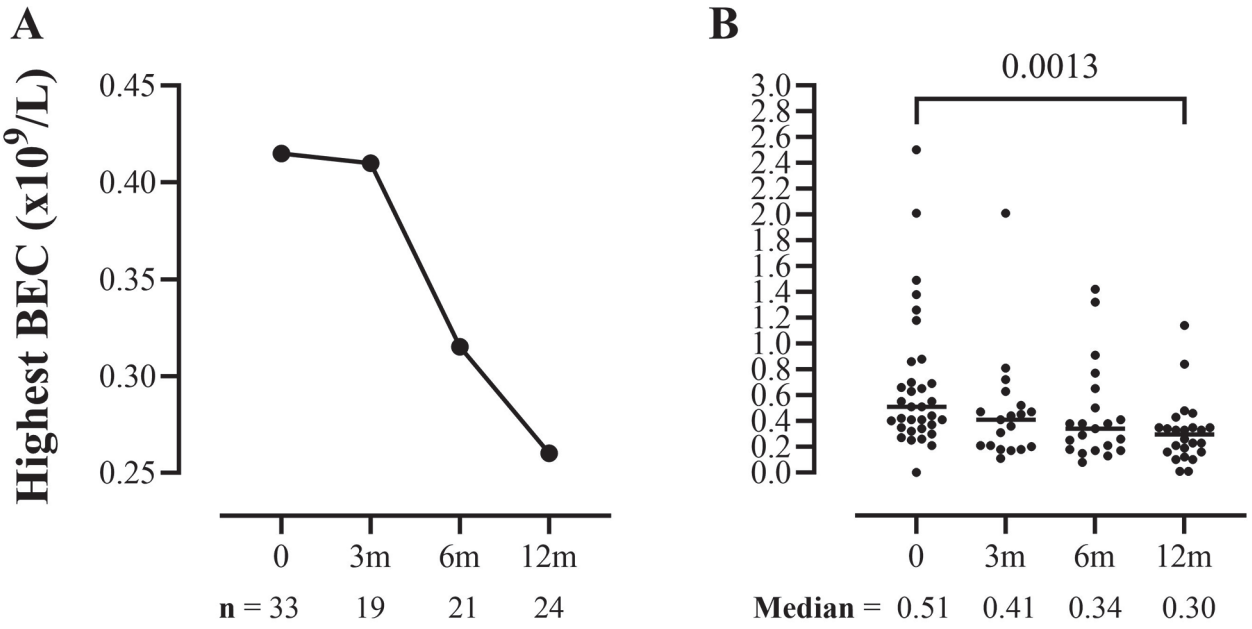
## P154 BLOOD EOSINOPHIL COUNTS AND ESCALATION TO HIGH DOSE ICS, IN COPD PATIENTS FROM A COMMUNITY SERVICE

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10.1136/thorax-2025-BTSabstracts.335

Higher blood eosinophil counts (BECs) in COPD are associated with an increased response to inhaled corticosteroid (ICS). Post-hoc analyses of randomised clinical trials of ICS-containing therapy have also shown that BEC reduction with ICS treatment occurs in some patients and is a biomarker of a positive treatment responses. In COPD patients with ongoing exacerbations plus high BEC while taking ICS, we investigated BEC change and clinical outcomes following an increase in ICS dose.

BEC and exacerbation rates, were retrospectively analysed before and after an increase to high dose ICS, from patients under the care of a COPD community service (CCS), Manchester University NHS Foundation Trust. BEC were collected when patients reported a worsening of symptoms to the CCS. BEC are reported as median ($BEC^{median}$) or the highest ($BEC^{highest}$) of any counts for each time-period; pre-ICS




*n = 33 COPD patients, 249 blood eosinophil counts. ICS dose increased after T0, subsequent follow ups every 3 months thereafter.*

**Abstract P154 Figure 1** Blood eosinophil counts and escalation to high dose ICS, in COPD patients from a community service

change (12-month period), and post-ICS change at 0–3 months (3m), 3–6 months (6m) and 6–12 months (12m). Results are reported as mean (SD) or median [range].

COPD patients (n=33) were 68.4 (9.7) years, 60.6% female with a CAT score of 23.3 (9.0), 30.3% were current smokers with a pack year history of 43.3 (20.2) and had an annual exacerbation rate of 3.2 (3.0). 57.6% of patients had a concomitant asthma diagnosis.

BEC was collected on ≥1 occasion during follow-up for n=33 (249 individual counts); a median of 5.0 [3.0–25.0] counts were reported per patient. A reduction in BEC compared to baseline was observed at 12 months follow up for $BEC^{highest}$(0.51 vs 0.30 x$10^9$/L, respectively, p<0.01, figure 1) and $BEC^{median}$(0.40 vs 0.28 x$10^9$/L, respectively, p=0.04). A numerical decrease in exacerbation rate was observed, however this was not statistically significant (3.0 vs 2.2, respectively, p=0.14).

We provide evidence of a decrease in BEC following increase in ICS dose. Exacerbation rates were numerically (not statistically significantly) lower; these data support a larger study to further investigate higher ICS use in this COPD subgroup.

## P155 DETERMINANTS OF DYNAMIC TROPONIN RISE WITHIN 30 DAYS OF MODERATE COPD EXACERBATION

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10.1136/thorax-2025-BTSabstracts.336

**Introduction** Patients with COPD have an increased risk of major adverse cardiovascular events (MACE) within 30 days of exacerbation. Our study aim was to investigate determinants of troponin rise amongst moderate exacerbations of COPD.

**Methods** Patients with COPD were prospectively recruited from 14 GP practices as part of the STARR2 study.[1] Blood sampling was obtained during steady state, at exacerbation (Day 0) and 14 and 30 days later. Troponin levels were measured using a commercial ELISA (R&D systems, LLD 125pg/mL). Peak dynamic troponin rise was calculated for each participant and defined as the maximum troponin rise between stable state and Day 30 post exacerbation. Significant troponin rise was defined as ≥20 pg/ml ('Troponin High' group).

Univariate analysis between 'Troponin High' and 'Troponin Low' groups included patient demographics, spirometry, MRC score, COPD therapies, cardiac risk factors and inflammatory blood biomarkers. Continuous variables were compared using Mann-Whitney *U* test and categorical variables using Fisher's exact test.

Significant variables on univariant analysis (p ≤ 0.10), were incorporated into a multivariate analysis.

**Results** 65 patients were included in the study, 66% were male, median age was 72 (IQR 66.5–76.5) years, 13 patients (20%) had a history of IHD.

21 patients (32.3%) had a significant troponin rise within 30 days of exacerbation. In the multivariate model, significant determinants of troponin rise were raised BMI (p=0.032); higher FEV1% (p=0.015) and higher stable state troponin (p=0.0013). The overall 'goodness of fit' $R^2$ for the multivariate model was 0.32 and the analysis of variance 0.042.

**Conclusion** Our study suggests that raised BMI, higher FEV1% and higher stable state troponin can help identify patients at risk of troponin rise during moderate COPD exacerbation. Further work is needed to understand potential mechanisms through which obesity may contribute to cardiac

injury at exacerbation. Further studies are required to determine whether higher activity levels amongst patients with less severe COPD leads to increased physiological cardiac stress during moderate exacerbation and explains the increased risk of troponin rise in this patient group.

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## P156 PREVALENCE OF CARDIOPULMONARY EVENTS AND RISK FACTORS BEFORE COPD DIAGNOSIS: ABACOS RISKS STUDY

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10.1136/thorax-2025-BTSabstracts.337

Following COPD diagnosis, individuals are at increased risk of cardiopulmonary events. Little is known about the occurrence of cardiopulmonary events and risk factors before COPD diagnosis.

Electronic health records of incident COPD cohort were identified (identification period 2018–2019) from CPRD Aurum linked with HES from England. Core cardiopulmonary events and broad cardiopulmonary risk factors were pre-specified and quantified for cases of incident COPD prior to index date (first observed COPD diagnosis) and among incident COPD with ≥3 years of pre-index data. Age, sex and ethnicity matched individuals without COPD served as controls.

Of 77,461 incident COPD cases (median age 68 years, 52% male, 93% White), 76.2% had at least 1 core cardiopulmonary event at any time before COPD diagnosis. Compared to controls, patients with COPD had 3.8 (95% CI: 3.7–3.9) times greater odds of having an antecedent core cardiopulmonary event. Of cases with ≥3 years of pre-index data, 56.6% had a recorded core cardiopulmonary event within the 3 years leading to COPD diagnosis with 4.9 (95% CI: 4.8–5.0) times greater odds.

Cardiopulmonary events and risk factors are highly prevalent before COPD diagnosis, with over half the individuals having a core cardiopulmonary event in the 3 years before COPD diagnosis. Early recognition and management of cardiopulmonary events could improve early detection of and outcomes in COPD.

## P157 EFFECT OF BENRALIZUMAB ON AIRWAY FUNCTION MEASURED BY IMPULSE OSCILLOMETRY DURING EOSINOPHILIC EXACERBATIONS: RESULTS FROM THE ABRA STUDY

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10.1136/thorax-2025-BTSabstracts.338

**Rationale** Eosinophilic exacerbations in asthma and COPD are characterized by small airway dysfunction (SAD) and heterogeneous recovery trajectories. Impulse oscillometry (IOS), a non-effort-dependent technique, enables detailed assessment of airway resistance and reactance, providing insights into lung mechanics during resolution.

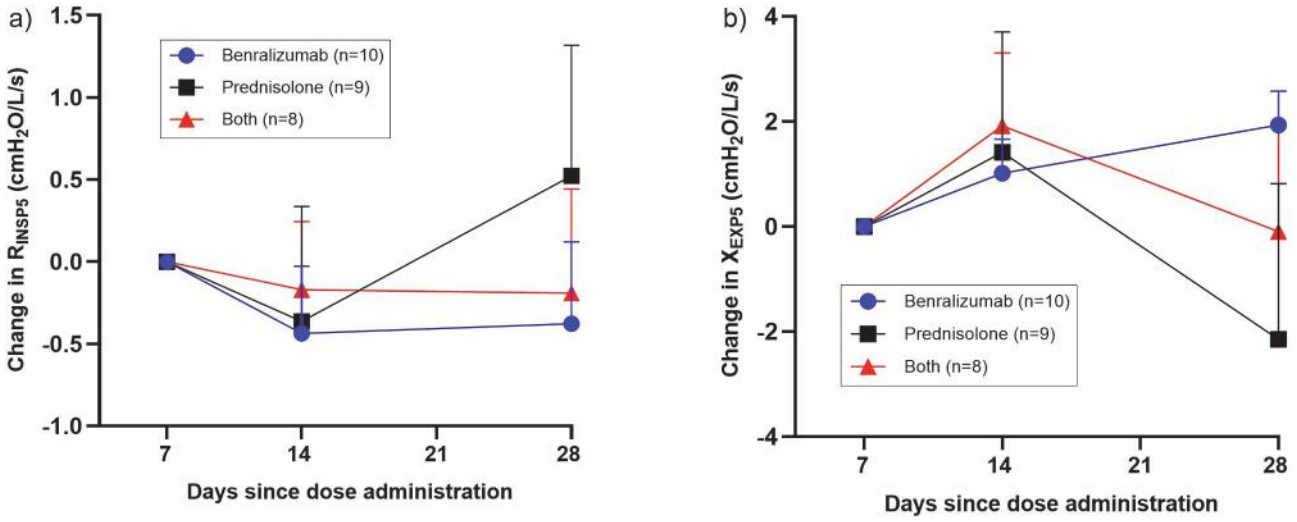
**Methods** This analysis compared IOS measurements between early and late recovery visits in 27 patients enrolled in the Acute Exacerbations Treated with Benralizumab (ABRA) trial. Eligible patients were those experiencing acute eosinophilic exacerbations, defined by a blood eosinophil count >300 cells/μL, and were randomized to receive one of three treatment strategies: benralizumab, prednisolone, or a combination of both. IOS parameters, including inspiratory, expiratory, and total resistance ($R_{INSP5}$, $R_{EXP5}$, $R_{TOT5}$), and reactance metrics ($X_{INSP5}$, $X_{EXP5}$, $X_{TOT5}$) were collected.

**Results** Patients treated with benralizumab showed significant differences when compared to the prednisolone group in $X_{EXP5}$ (-1.902 vs -9.669 cmH2O/L/s, p=0.044), and $X_{TOT5}$ (-1.625 vs -7.367 cmH2O/L/s, p=0.046), suggesting a decline in SAD recovery. In addition, benralizumab and combination

**Abstract P156 Table 1**

| | Incident COPD cases (N=77,461) | | Matched Controls without COPD (N=154,856) | | Incident COPD with ≥3 years data prior to diagnosis (N=70,062) | |
|---|---|---|---|---|---|---|
| **≥1 Core Cardiopulmonary Event*** | 59,039 | 76.2% | 70,615 | 45.6% | 39,633 | 56.6% |
| ≥1 Core Cardiovascular Event | 23,840 | 30.8% | 26,156 | 16.9% | 16,691 | 23.8% |
| **≥1 Broad Cardiopulmonary Risk Factors**** | 75,285 | 97.2% | 132, 429 | 85.5% | 66,343 | 94.7% |
| ≥1 Broad Cardiovascular Risk Factors | 53,627 | 69.2% | 88,698 | 57.3% | 43,305 | 61.8% |

*Core cardiopulmonary events included cardiovascular diagnoses (heart failure, myocardial infarction, cardiac arrest, emergency revascularization, tachyarrhythmia, atrial fibrillation, ischaemic stroke) and/or COPD exacerbations/lower respiratory tract infections; **Broad cardiopulmonary risk factors included core events and following cardiovascular diagnoses (transient ischemic attack, chronic ischemic heart disease, angina pectoris, hypertension, cardiomyopathy, diabetes mellitus, hyperlipidaemia, pulmonary hypertension), respiratory diagnoses (pneumonia, acute bronchitis, hypoxemia, pulmonary hypertension, dyspnoea, chronic cough, sputum and wheeze) and/or lifestyle factors (smoking, family history of cardiovascular disease).




**Abstract P157 Figure 1**

therapy exhibited downward trends in $R_{INSP5}$, $R_{EXP5}$ and $R_{TOT5}$, indicating improvements in peripheral resistance.

**Conclusions** IOS revealed distinct patterns of airway recovery following eosinophilic exacerbations. Benralizumab alone or in combination was associated with consistent improvements in reactance parameters, reinforcing previously observed findings,[1] while oral prednisolone alone appeared less effective in restoring peripheral airway function. One possible explanation may be that benralizumab improves SAD more than prednisolone due to its faster depletion of airway eosinophils, leading to quicker reduction of inflammation.[2] These results highlight the value of IOS as a sensitive method for identifying the resolution of SAD and for tracking the mechanisms underlying responses to steroid and biologic treatments.

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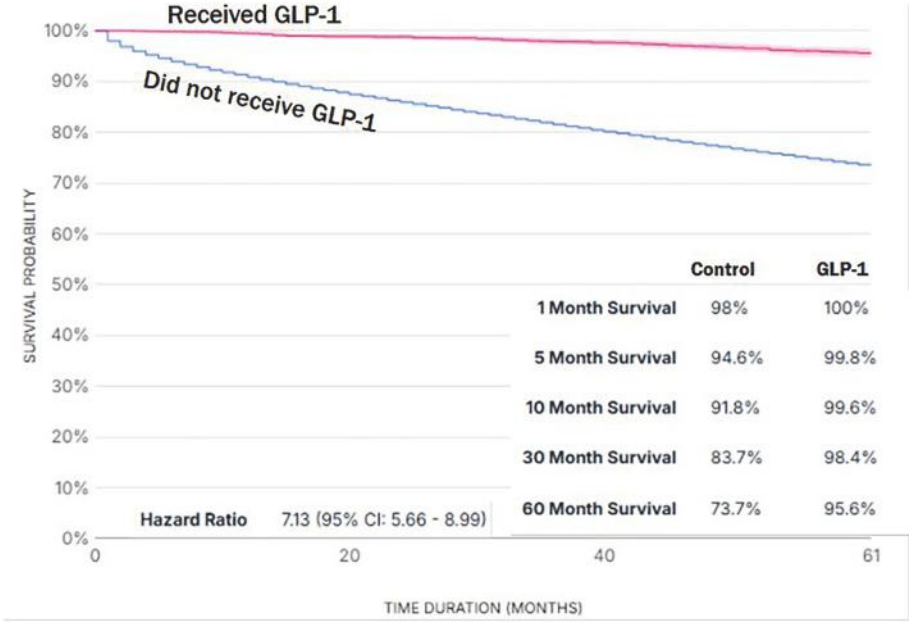
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## P158 THE ASSOCIATION BETWEEN GLP-1 RECEPTOR AGONIST USE AND MORTALITY AMONG COPD PATIENTS

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10.1136/thorax-2025-BTSabstracts.339

**Introduction** Glucagon-like peptide-1 (GLP-1) receptor agonists are novel antihyperglycemic agents that have demonstrated benefits in cardiovascular and renal disease. In murine models of obstructive lung disease, GLP-1 agonists have been shown to reduce airway inflammation and hyperresponsiveness. In humans, some observational studies have shown an association between GLP-1 agonist use and reduced exacerbations of




**Abstract P158 Figure 1**

chronic obstructive pulmonary disease (COPD), as well as a lower risk of non-invasive ventilation, invasive mechanical ventilation, and bacterial pneumonia. We examined the effects of GLP-1 agonists on mortality in a large sample of patients with COPD.

**Methods** In this retrospective case-control study, we identified patients with COPD from a database at our institution. We then identified those who had received a GLP-1 agonist after their COPD diagnosis. Data are presented as median with interquartile range, and a Kaplan-Meier curve was used for mortality analysis.

**Results** We identified 186,985 patients with COPD who had not received a GLP-1 agonist and 1,877 patients who had. The treatment group was followed for a median of 1,247 days (interquartile range: 667 to 2,015), compared to 349 days (174 to 606) in the control group.

The median age in the GLP-1 group was 68.8 years versus 60.3 years in the control group. Females comprised 49% of the GLP-1 group and 48% of the control group. In the GLP-1 group, 80% of patients were obese, with a mean BMI of 32.5, compared to 26.5% in the non-GLP-1 group. Additionally, 89% of patients in the GLP-1 group had type 2 diabetes mellitus, compared to 28% in the control group.

Patients who received a GLP-1 agonist had improved survival at 1, 5, 10, 30, and 60 months compared to those who never received a GLP-1 agonist ($p < 0.001$; hazard ratio: 7.13, 95% CI: 5.66–8.99).

**Discussion** Our study showed that GLP-1 agonists were associated with improved survival in patients with COPD. This may be related to improved cardiovascular mortality in these patients. Further studies are needed to evaluate GLP-1 agonists as potential therapeutic options for patients with COPD and to elucidate the mechanisms underlying this association.

## P159 STABILITY AND RELATIONSHIP BETWEEN DIGITALLY MONITORED INHALED CORTICOSTEROIDS ADHERENCE AND TYPE 2 BIOMARKERS IN COPD

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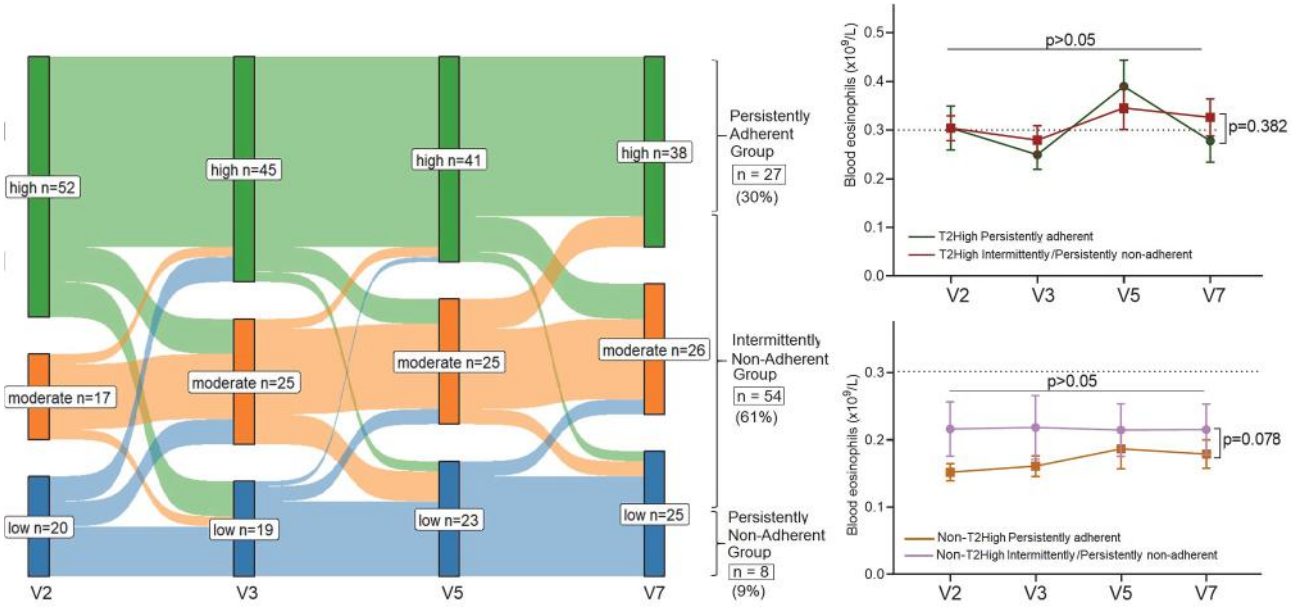
10.1136/thorax-2025-BTSabstracts.340

**Background** Type 2 (T2) inflammatory biomarkers are known to be elevated among patients with eosinophilic COPD receiving inhaled corticosteroids (ICS). However, a significant research gap exists regarding the impact of objective inhaler adherence on these biomarkers.

**Aim** To explore how inhaler adherence stability influences type 2 biomarkers.

**Method** COPD patients on triple inhaled therapy were electronically monitored for inhaler adherence over 6 months using FindAir digital inhalers. Adherence was classified as persistently adherent (adherence rate (AR)≥75% in all visits), intermittently or persistently non-adherent (AR<75%). Blood and sputum eosinophils, immune cell profiles, and fractional exhaled nitric oxide (FeNO) were monitored.

**Result** Of 104 patients (age 67±9 yrs, $FEV_1$=1.1±0.5L), median [IQR] AR per participant was 70% [44 - 86] over 6 months, significantly decreasing over time ($p<0.001$ at month 4 onwards). Prescription refills and self-reports significantly overestimated true adherence ($p<0.001$). Only 30% were persistently adherent to maintenance ICS at all time (figure 1). Participants with eosinophilic COPD tended to be more persistently adherent than their counterparts, 45% vs 25%, $p=0.05$. Persistently adherent patients had higher baseline sputum T2-




**Abstract P159 Figure 1** Alluvial Plot Illustrating Longitudinal Inhaler Adherence. Adherence rate among participants were stratified as (Persistently adherent: adherence rate AR≥75% at all visits, intermittent: AR<75% ≥1 visits, and persistently non-adherent: AR<75% at all visits) alongside transitions in blood eosinophil count over time, at study visits V1, V2, V3, V5, and V7. At baseline, participants were classified as T2High (highest BEC ≥ 0.3 x $10_9$/L in the prior year) or non-T2High. AR at each visit was categorised as low (AR<50%), moderate (AR 50–74%), or high (AR≥75%). Changes in blood eosinophils across visits were analysed using mixed-effect models

polarised T-cells (p=0.03) and lower FeNO levels (13[10–21] vs 17 [12–33] ppb, p=0.05) with greater ICS exposure, (402 [374–445] vs 278 [150–331] mcg BDP equivalence, p<0.001) than the rest. No significant change over time was observed in blood eosinophils, immune cells, FeNO or lung function across adherence groups.

**Conclusion** Findings suggest a disconnect between ICS adherence and T2 inflammatory biomarkers in COPD, a pattern that contrasts with what is typically observed in asthma.

## P160 CAN WE PREDICT LENGTH OF HOSPITAL STAY AND MORTALITY USING NIVO SCORE FOR PATIENTS ADMITTED WITH ACUTE EXACERBATION OF COPD REQUIRING NON-INVASIVE VENTILATION?

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10.1136/thorax-2025-BTSabstracts.341

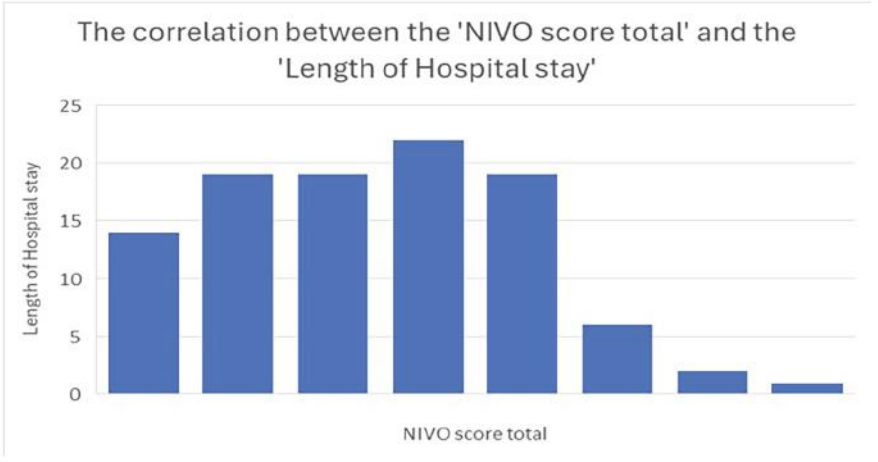
**Background** Non-invasive ventilation outcome (NIVO) score is a tool that demonstrated strong predictive capability for in-hospital mortality with acute exacerbation of chronic obstructive pulmonary disease (COPD). NIVO score includes consolidations on Chest x-ray, Glasgow Coma Scale <14, Atrial Fibrillation, PH <7.25, Time to academia >12 hrs of admission, and Extended MRC dyspnoea scale.

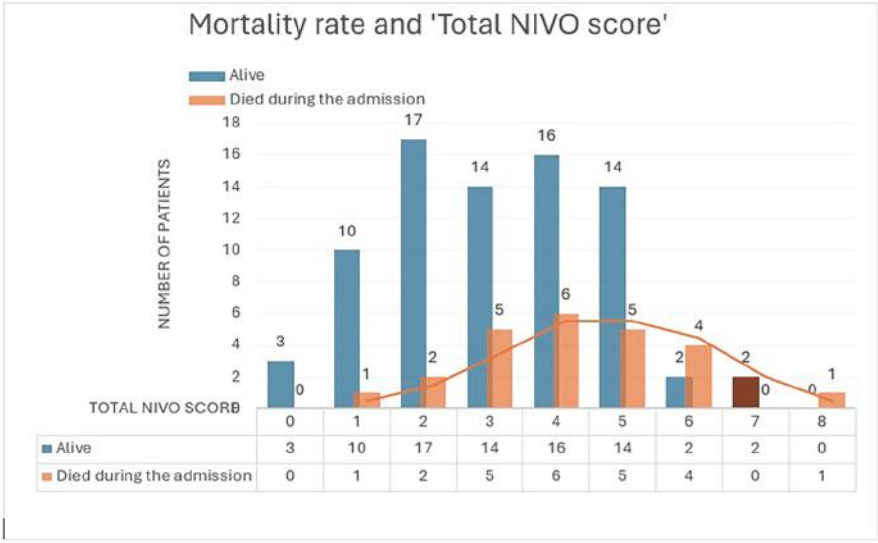
We wanted to study if NIVO score can also be used to predict their length of hospital stay after being admitted with exacerbation of COPD requiring non-invasive ventilation (NIV).

**Hypothesis** With higher the NIVO score, we expect patients to have longer length of hospital stay and higher mortality rate.

**Methods** 102 patients admitted to our hospital with acute exacerbation of COPD treated with non-invasive ventilation (NIV) were studied. NIVO score was calculated retrospectively and correlated with their length of hospital stay and mortality rate for that admission.

**Results** Out of 102 patients, 24 patients died during their hospital admission. Around 87.5% of the patients died scored 3 or more on the 'Total NIVO score'.




**the 2 patients with a total NIVO score of 7: One patient died within 20 days post discharge, the other patient had prolonged hospital admission of 63 days.

**Abstract P160 Figure 1**

Amongst the domains of NIVO criteria, PH <7.25 was analysed as an independent factor related to mortality (70% of the patients died during admission had pH <7.25).

The average NIVO score was 3.4 and the average Length of stay was 15.66 days.

The correlation of length of stay with NIVO score was calculated using Pearson Correlation Coefficient (R-value of 0.0624 and P-value of 0.533252, no significant correlation).

**Conclusion** This study demonstrated an increased length of hospital stay and higher mortality in patients with NIVO score of 3 or more. We recommend larger sample size to study the association between these variables.

## P161 PREDICTORS OF IN-FLIGHT DESATURATION AND NEED FOR SUPPLEMENTARY OXYGEN THERAPY DURING AIR TRAVEL. HYPOXIC CHALLENGE TEST (HCT) IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) [PRELIMINARY RESULTS]

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10.1136/thorax-2025-BTSabstracts.342

**Introduction and Objectives** Patients with COPD planning air travel need careful evaluation by a lung specialist. According to current practice, baseline oxygen saturation and dyspnea are the initial screening tools, followed by a 6-min walking test to direct the need for an HCT. However, an HCT is time-consuming, not widely available in daily clinical practice and an indication for a HCT should be carefully sought. We hypothesized that pulmonary function tests (spirometry, body plethysmography, single-breath gas transfer), including oscillometry, a test that requires minimal patient co-operation, can reliably predict a significant desaturation during air travel, and therefore direct the need for an HCT.

**Methods** Patients with stable COPD were recruited. Dyspnea was assessed by the mMRC scale, baseline SpO2 was measured, followed by a full pulmonary function testing and a 6MWT. The following functional parameters were evaluated: spirometry (FEV1, FVC, FEV1/FVC ratio), static volumes (TLC, FRC), diffusion capacity (DLCO), oscillometry [R5, R20, R5-R20, X5, Ax, Resonant frequency (Fres)]. All patients underwent HCT using a 40% Venturi mask with nitrogen as the driving gas (FiO2:15%) for 20 minutes or until SpO2<85%. A drop of SpO2<85% was considered as positive HCT.

**Results** 15 patients were analyzed (67.9±6.5 years old). 4 (~26%) exhibited a significant desaturation in HCT. Based on ROC curve analysis, $FEV_1$ showed the highest discriminatory ability for a positive HCT test (AUC=0.943, p<0.001), followed closely by Fres (AUC=0.925, p<0.001) and DLCO (AUC=0.926, p<0.001). Baseline $SpO_2$ and $SpO_2$ after 6-MWT showed excellent accuracy (both AUC=0.909, p<0.001) and the mMRC score had a good predictive value (AUC=0.864, p<0.001). Adding oscillometry parameters (Fres) to conventional lung function testing ($FEV_1$, DLCO, mMRC and baseline $SpO_2$) improved the correlation with SpO2 during the HCT test, as assessed by multiple regression analysis (adj $R^2$=0.77, p=0.022, including oscillometry vs adj $R^2$=0.62, p=0.027 without oscillometry). No significant effects were observed for lung volume measurements.

**Conclusions** $FEV_1$ exhibited the highest predictive value for a significant desaturation during an HCT and therefore, a low $FEV_1$ should be actively sought during a pre-flight assessment in patients with COPD. Oscillometry improves the performance of conventional function testing in a preflight respiratory screening.

## P162 CHANGES IN BLOOD CYSTATIN C DURING COPD EXACERBATIONS

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**Introduction** Exacerbations of COPD are associated with cardiovascular events. Biomarkers are needed to identify those at risk. Cystatin-C (Cys-C) is a cysteine protease inhibitor that predicts future cardiovascular risk and is a sensitive marker of renal function. Though Cys-C is known to be elevated in COPD[1] its pattern at exacerbation is uncertain. This study aimed to determine how Cys-C levels change over the course of an exacerbation, and the relationship of this to important biomarkers of cardiorenal disease.

**Methods** Patients from the London COPD exacerbation cohort (EXCEL) were reviewed at stability, exacerbation-onset, 2-weeks and 6-weeks post-exacerbation. At each visit, serum Cys-C was measured, together with inflammatory (CRP, fibrinogen); renal (urea, creatinine, eGFR) and cardiovascular (troponin, BNP) biomarkers. An in-house enzyme-linked immunosorbent assay was developed to determine Cys-C levels, whereas other markers were processed by the local clinical laboratory. The absolute and percentage change in Cys-C across exacerbation time points was measured. We used Spearman's rank to test the correlation between Cys-C and other biomarkers.

**Results** The study included 22 patients with COPD who had a mean age of 74.05 (± 9.31) years and 8 (36.36%) were female. The median (interquartile range) of Cys-C values at stability, exacerbation, 2-weeks and 6-weeks post-exacerbation were 9.32 (6.38–15.50), 11.76 (6.30–18.13), 8.02 (5.75–13.39) and 7.35 (4.96–9.58) ng/ml respectively. There was a non-significant increase from baseline to exacerbation. Cys-C was statistically increased at exacerbation compared to 6-weeks post-exacerbation (p=0.021). There was only one significant correlation between Cys-C and the other biomarkers at any timepoint or interval: with CRP at baseline (p=0.47, p=0.0376).

**Conclusion** This study demonstrated a non-significant increase in Cys-C from baseline to exacerbation, in line with existing literature, whilst also demonstrating a significant decrease at 6-weeks post-exacerbation to below baseline; a unique profile which correlates with no other biomarker tested. Further work is needed to validate this finding and to determine the relationship of Cys-C to systemic complications of exacerbation.

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# Cancer: from screening to therapy

## P163 AN UPDATE REGARDING THE MESO-ORIGINS STUDY: PART OF THE PREDICT-MESO INTERNATIONAL ACCELERATOR NETWORK

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10.1136/thorax-2025-BTSabstracts.344

**Objectives** The PREDICT-Meso International Accelerator Network (>150 investigators, 90 institutions, 16 countries) aims to define the biology driving Pleural Mesothelioma (PM). This requires assembly of a suitably characterised tissue resource spanning asbestos-associated pleural inflammation (AAPI)-to-PM evolution. The Meso-ORIGINS study is delivering this component, incorporating learning from a prior multi-centre feasibility trial where the PM evolution rate was 14% (95%CI 10.5–19.2). The primary objective is to collate longitudinal tissue pairs from patients with AAPI who evolve to PM. Secondary objectives are (a) generation of a multiomic risk prediction model for use in AAPI (b) characterisation of intra-patient tumour heterogeneity using multi-region thoracoscopic biopsies.

**Methods** Meso-ORIGINS is a prospective multi-centre observational study, comprising two arms (A, which includes an MRI sub-study, & B). Arm A (target n=300) patients with histologically-confirmed AAPI undergo 6-monthly surveillance for 2 years, with clinical suspicion of PM evolution prompting repeat pleural biopsy and tissue banking. Arm A recruits also have baseline risk profiling via serum proteomics, exhaled breath metabolomics ± perfusion MRI. Arm B (target n=250) patients with suspected PM requiring thoracoscopy have multi-region thoracoscopic biopsies (aim 4–6/case).

**Results** Recruitment opened in July 2022 and will end in August 2026. Across 25 UK sites (see figure 1), 408 patients have been recruited: 197/300 (66%) to Arm and 211/250 (84%) to Arm B. 18 PM evolutions (47% target) have been occurred in Arm A, where the current evolution rate is 11.3%, and the 95%CI overlaps with the prior feasibility study (7.3–17.4). 73/211 (35%) and 92/211 (44%) Arm B recruits have been diagnosed with PM and AAPI, respectively. 48/73 (66%) AAPI cases have subsequently been recruited to Arm A, permitting linkage of multi-region pre-PM biopsies. A mean (SD) of 3.9 (1.8) multi-region biopsies have been collected per Arm B case, generating 677 samples.

**Conclusions** Meso-ORIGINS is generating a unique longitudinal, multi-region tissue resource spanning AAPI-to-PM evolution. This material is being processed in downstream PREDICT-Meso work-packages focused on multiomic target identification (including WES, spatial transcriptomics) and used for generation of new pre-clinical models and drug screening. Risk profiling results will inform eligibility criteria for future intervention trials for pre-PM patients.

## P164 INSIGHTS FROM PUBLIC INTERVIEWS IN THE UNITED KINGDOM ABOUT THE ACCEPTABILITY OF RISK-BASED ELIGIBILITY CRITERIA IN LUNG CANCER SCREENING

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10.1136/thorax-2025-BTSabstracts.345

**Introduction and Objectives** Multivariate risk prediction models are superior to category-based approaches for identifying those most likely to benefit from lung cancer screening. However, little is known about public perception of using these models to restrict screening to those at high risk of lung cancer. We aimed to understand the acceptability of specific risk-based eligibility criteria for lung screening to inform population and individual-level communication strategies.

**Methods** As part of a larger multi-method series of studies, a qualitative study was conducted using remote semi-structured interviews with individuals (n=28) living in the UK aged 45–80 years. Participants were asked open-ended questions about their views towards targeting lung cancer screening by individual risk, the acceptability of each risk-based criterion and their anticipated reaction to disclosure of their eligibility status. Applied thematic analysis was used to interpret themes.

**Results** Acceptability of the risk-based criteria varied according to the specific criteria. Understanding the rationale for the criteria was highlighted as important in influencing acceptability. Most participants found the criteria based on familiar and logical risk factors (age, smoking, family history) to be acceptable. Participant views around ethnicity and educational level as eligibility criteria were more complex due to lack of understanding about the relevance of these factors in influencing lung cancer risk with a minority of participants expressing concern that these criteria may be perceived as unfair and discriminatory. The exclusion of people who have never smoked but felt at risk due to passive smoking was a concern for some participants. Ineligibility evoked feelings of disappointment and worry, and there was misperception that ineligibility may signify good lung health.

**Conclusions** This is the first study to seek the UK public's perspectives on the acceptability of targeting lung cancer screening by individual risk. Individuals were broadly accepting of risk-based eligibility criteria for lung screening where there was evidence linking the criteria to increased risk of lung cancer. Lack of understanding about the risk factors for lung cancer development as well as misperceptions around the diagnostic capability of the risk eligibility criteria highlight the need for widespread public communication before direct invitation to lung screening.

## P165 A SERVICE EVALUATION OF THE TARGETED LUNG HEALTH CHECK (TLHC) SCREENING PROGRAMME IN A MAJOR ENGLISH CITY

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10.1136/thorax-2025-BTSabstracts.346

**Background** Lung cancer is the leading cause of cancer-related deaths in the world. It progresses without symptoms and is typically detected in its advanced stages. Survival rates often depend on staging. Therefore, early detection can lead to more curative surgeries and a better prognosis. Low-dose CT lung cancer screening has been successful, with a proven 20% decrease in mortality.
**Aims** This study aims to evaluate the effectiveness of the targeted lung health check (TLHC) service in a major English city. Data from 2020–2024 were used to assess diagnosis rates, treatment pathways and outcomes of lung cancer patients identified through the screening programme.
**Methods** Patients eligible for the study were identified using the TLHC's database. The timelines of their screening, treatments, surgical interventions, and outcomes were collected using the hospital's CHARTS system. Microsoft Excel was used to analyse the data.
**Results** 240 suspected lung cancer patients (2.94%) were detected from a screened population of 8154 over that period. 195 patients (81.25%) were diagnosed at stage I or II. 109 patients (45.41%) had undergone lung resections, and 73/109 had curative resections as the only required intervention. 99/240 patients (41.25%) had undergone other treatments including neoadjuvant/adjuvant therapy. 54 (22.50%) patients received palliative care.
**Conclusion** 28% of lung cancer diagnoses in the UK were stage I/II in 2019; however, this study has shown an increase in early diagnosis rates with TLHC, as 81.25% of this cohort were diagnosed at stage I/II. For histologically proven cancer, the average surgical resection rate for England is 24.6% (range 14.2%-34.4%). 45.41% of patients who were referred via this TLHC program were able to access lung resections, majority at an earlier stage. In this population, the TLHC programme has proven beneficial in improving the number of asymptomatic patients diagnosed at earlier and more treatable stages.

## P166 LUNG CANCER SCREENING PROGRAMME IN SOCIALLY DEPRIVED EAST LONDON: OUTCOMES AND EARLY CANCER DETECTION

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**Background** The UK's Lung Cancer Screening Programme aims to facilitate earlier lung cancer detection in adults aged 55–74 with a smoking history. Social deprivation is strongly correlated with increased lung cancer incidence. The East London boroughs of Tower Hamlets, Barking and Dagenham, and Newham are among the UK's most deprived areas, whilst Havering is near the national average for deprivation. This study evaluates outcomes of Lung Cancer Screening in these boroughs from 2023 to 2025.
**Methods** We conducted a retrospective analysis of the programme's data from 18th April 2023 to 18th March 2025 across the specified East London boroughs. Scan outcomes were triaged into different pathways based on urgency for clinical action.
**Results** 45,123 patients were invited during the designated time period with an uptake of 58.6% (26,433 patients) completing the health checks. A total of 12,415 low dose CT scans were performed involving 10,251 patients. 2753(22%) required follow-up for lung nodules closer than the routine 24-month interval. Non-urgent referrals to secondary services were made for 716 (5.8%) patients due to other pulmonary or extra-pulmonary findings. Urgent clinical action was required for two patients (large pneumothorax, severely dilated ascending aortic aneurysm). A total of 322 (2.6%) scans had concerning findings necessitating urgent review, including potential lung cancer, severe active Tuberculosis, dilated aorta, unilateral pleural effusion, and other extrapulmonary lesions suspicious for malignancy. 117 patients had histologically confirmed lung malignancy, with 86 (74%) diagnosed at an early stage (Stage I or Stage II). 34% of the patients with confirmed lung cancer were under the age of 65, 59% were male, and only 24% were from an ethnic minority background. The distribution of confirmed lung cancer cases by borough was: Havering 7 (0.7% of patients from borough scanned), Newham 24 (1.0%), Barking & Dagenham 29 (1.1%), and Tower Hamlets 55 (1.4%).
**Conclusion** Lung cancer screening in East London effectively identified a significant number of lung cancer cases, predominantly at early stages, within a socially diverse and often deprived population. Further analysis is warranted to explore how specific social deprivation indices correlate with participation rates, referral pathways, and stage at diagnosis.

## P167 THE RISK OF LUNG CANCER IN THE POPULATION THAT DECLINED ENROLMENT IN TARGETED LUNG HEALTH CHECK PROGRAMME

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The Staffordshire Targeted lung health check (THLC) project commenced in 2019 and joined the national TLHC programme in 2022. During this period the project used the same eligibility criteria as the national programme: age 55–75 years of age; ever smoker and registered with a GP. During the initial phase of the project people were invited to make an appointment for discussion and assessment of lung cancer risk, this changed later to people opting out of appointments, as this improved uptake in the programme. The lung cancers identified through the screening programme were compared to this individuals that did not participate in the programme, by reviewed the local cancer registry for the GP practices that had been involved in the project at that time, and patients who would have been in the eligible population and would have been offered an invitation to participate.

**Abstract P167 Table 1**

| | Screened population | Non-screened population |
|---|---|---|
| Total population | 31355 | 34032 |
| Total lung cancer | 252 | 387 |
| Stage 1 | 151 | 104 |
| Stage 2 | 26 | 36 |
| Stage3 | 46 | 67 |
| Stage 4 | 26 | 156 |
| Unknown stage | 3 | 24 |

Between 2019 and 2024 the project screened 31,355 individuals, with 34,032 not screened. 252 lung cancers were identified through screening, while 387 lung cancers were identified in the non-screened population.

Comparing the stage of cancers between the 2 groups there was statistically significant greater risk of developing lung cancer in the group that were diagnosed through the non-screening route. Reinforcing the need to try to reach this group of people who declined enrolment in the TLHC programme, as this group are at a higher risk of developing lung cancer.

## P168 REASSESSMENT OF 'MODERATE RISK' LUNG CANCER SCREENING PARTICIPANTS IN THE SOMERSET, WILTSHIRE, AVON AND GLOUCESTER (SWAG) CANCER ALLIANCE

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**Introduction** NHS England's Lung Cancer Screening (LCS) programme identifies individuals at high risk of lung cancer

**Abstract P168 Table 1** Comparison of risk score parameters at initial assessment and 24-month reassessment

| | Initial assessment | Reassessment | Interpretation |
|---|---|---|---|
| Age - years, median (IQR) | 65 (60-70) | 68 (62-73) | +3 years |
| Smoking duration - pack years, median (IQR) | 16 (7.5-28.9) | 22 (10-38) | +6 pack years |
| Current smokers, n (%) | 45 (13.4) | 49 (14.5) | 5 smokers quit, 9 ex-smokers relapsed |
| Previous pneumonia, n (%) | 53 (15.7) | 59 (17.5) | 6 new pneumonia cases |
| Previous TB , n(%) | 6 (1.8) | 6 (1.8) | No new TB cases |
| Emphysema / COPD / chronic bronchitis, n (%) | 79 (23.4) | 92 (27.3) | 13 new diagnoses |
| Previous history of cancer, n (%) | 41 (12.2) | 57 (16.9) | 16 new cancers |
| Family history of lung cancer, n (%) | 35 (10.4) | 45 (13.4) | 10 new family lung cancer diagnoses |
| Prior asbestos exposure, n (%) | 63 (18.7) | 67 (19.9) | 4 additional reports of asbestos exposure |
| BMI, kg/m$^2$ (median) | 27.7 (24.6-31.1) | 27.6 (24.8-31.4) | No significant change |
| **Risk model scoring** | | | |
| $PLCO_{2012}$ score, median (IQR) | 0.9 (0.4-1.4) | 1.1 (0.5-1.9) | +0.2 |
| High risk on $PLCO_{2012}$ score, n (%) | 0 | 124 (36.8) | - |
| $LLP_{v2}$ score, median (IQR) | 2.1 (1.8-2.3) | 2.7 (1.7-3.4) | +0.6 |
| High risk on $LLP_{v2}$ score, n (%) | 0 | 189 (56.1) | - |

using the $PLCO_{2012}$ and $LLP_{v2}$ risk models. National guidance advises that participants who do not initially meet the risk threshold should be reassessed at the point they are likely to become high risk. However, as an evolving programme, the optimal timelines for reassessment and the associated clinical implications are not known. We evaluated the rate of conversion to high-risk and subsequent screening outcomes of a cohort of 'moderate-risk' people who were reassessed at 24 months.

**Methods** Individuals with an initial $PLCO_{2012}$ score of 1.3–1.5% or $LLP_{v2}$ score of 2.0–2.49% were reassessed between January and June 2025. We documented cases where reassessment resulted in a high-risk score, i.e. $LLP_{v2} \geq 2.5$ or $PLCO_{2012} \geq 1.51$, and investigated the change in risk variables between the two assessments. For participants who converted to high-risk, we described scan results and clinical outcomes.

**Results** 337 individuals met the criteria for reassessment, of whom 230 (68.2%) converted to high-risk on reassessment: 106 (31.5%) on $LLP_{v2}$ criteria, 41 (12.2%) on $PLCO_{2012}$ criteria and 83 (24.6%) on both. To date, 148/230 (64.3%) have undergone CT scans, with the remainder awaiting scheduling. Most scans (n=127/148; 85.7%) were normal and participants will undergo 24-month follow-up. 16/127 participants (10.8%) entered nodule surveillance pathways, whilst 5/127 (3.4%) had abnormal imaging necessitating Screening Review Meeting discussion and/or referral to secondary care cancer services.

Age, smoking duration, new diagnoses of airways disease, personal history of cancer, and family history of lung cancer were the factors that most often changed between assessments, driving increases in risk scores (table 1).

**Conclusion** A significant proportion of 'moderate-risk' LCS participants converted to high-risk at 24-months reassessment. This resulted in increased CT scanning requirements with associated downstream resource implications. LCS programmes will need to incorporate this in future capacity modelling.

Moving forwards, national guidance is required around reassessment timeframes. Risk modelling to predict when participants might transition to high risk, based on age and pack year calculations, could facilitate targeted reassessment to ensure efficient resource utilisation.

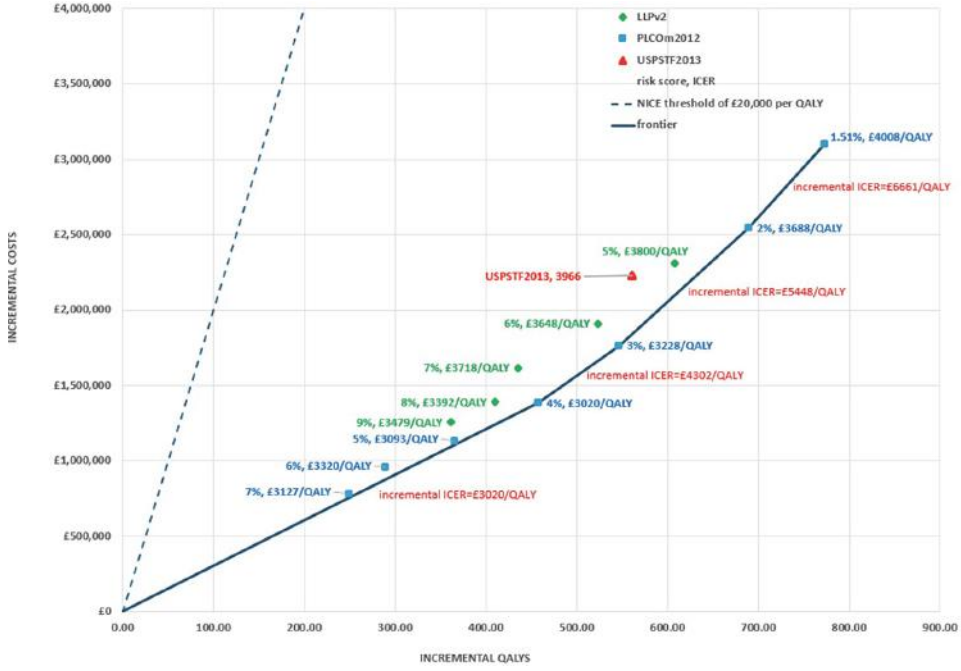
## P169 COMPARATIVE COST-EFFECTIVENESS OF SCREENING FOR LUNG CANCER USING DIFFERENT RISK SCORES AND THRESHOLDS: THE YORKSHIRE LUNG SCREENING TRIAL

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**Introduction and Objectives** Lung cancer (LC) is the most common cause of cancer death in the UK and worldwide, but screening with low-dose computed tomography (LDCT) has been shown to reduce LC deaths. The UK National Screening Committee has recommended nationwide roll-out of LDCT screening by 2029, but the optimal risk thresholds for eligibility remain uncertain. This study interrogates the cost-effectiveness of competing risk scores and thresholds, informed by the Yorkshire Lung Screening Trial (YLST).

**Methods** We conducted a cost-effectiveness analysis using data from the YLST to compare different risk thresholds ($USPSTF_{2013}$, $PLCO_{M2012} \geq 1.51\%$ and $LLP_{v2} \geq 5\%$). A Markov model was used to estimate the long-term implications of different screening approaches, including a no-screening counterfactual. Payouts per detected LC were calculated using LC




**Abstract P169 Figure 1**

mortality and utility estimates from the literature, and cost data from the literature and the YLST. Scenario analyses assessed how increasing thresholds for the $PLCO_{M2012}$ and $LLP_{v2}$ affected LC detection, costs, and quality-adjusted life years (QALYs).

**Results** In the YLST, 6,650 participants underwent LDCT screening after meeting at least one of the three eligibility criteria, and 282 invasive LCs were diagnosed over two screening rounds. The incremental cost-effectiveness ratios (ICER) for the three eligibility criteria used in the YLST (*vs.* no screening) were £3,949 for $USPSTF_{2013}$, £3,797 for $LLP_{v2} \geq 5\%$ and £4,013 for $PLCO_{M2012} \geq 1.51\%$. Of the three criteria, $PLCO_{M2012}$ resulted in the largest numbers of people screened and LCs detected, the most QALYs gained and highest incremental net monetary benefit (iMNB), and was the optimal choice based on cost-effectiveness. The figure 1 below shows that $PLCO_{M2012}$ 4%, 3%, 2%, 1.51% form the efficiency frontier, delivering more QALYs for lower costs compared to $LLP_{v2}$ and $USPSTF_{2013}$ criteria.

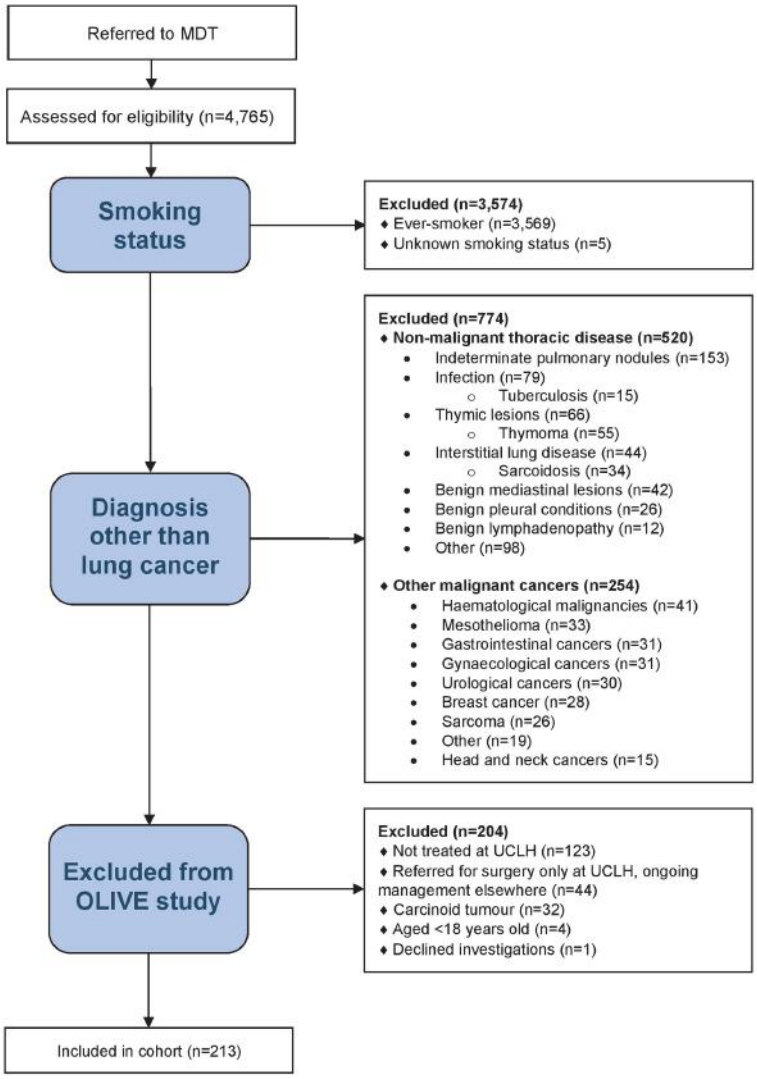
**Conclusion** All three screening eligibility criteria used in the YLST are cost-effective according to UK's willingness to pay threshold of £20,000/QALY, with $PLCO_{M2012} \geq 1.51\%$ being the optimal approach. Although it was not possible to robustly test the impact of lowering risk score thresholds further (i.e. below the thresholds used in the YLST), these and other data suggest that this is likely to detect more cases of LC whilst remaining cost-effective.

## P170 DIAGNOSES OF NEVER-SMOKERS REFERRED TO THE LUNG CANCER MULTI-DISCIPLINARY TEAM: LUNG CANCER AND BEYOND

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**Background** Characteristics of lung cancer in never-smokers in the UK are poorly understood. We examine outcomes of never-smokers referred to lung cancer MDT and report on individuals subsequently diagnosed with lung cancer.




**Abstract P170 Figure 1** Flow diagram of individuals referred to lung cancer multi-disciplinary team (MDT) and reasons for exclusion from OLIVE study

**Methods** We reviewed electronic health records of adults referred to lung cancer MDT at University College London Hospital between 2019 and 2025. Individuals diagnosed with lung cancer were recruited to the OLIVE study funded by NIHR (NIHR303686) and Ruth Strauss Foundation. Demographic and clinical data were compared to National Lung Cancer Audit data.

**Results** Of 4,765 individuals discussed at MDT (figure 1), 75% (n=3,574/4,765) were excluded due to ever- (n=3,569) and unknown (n= 5) smoking status.

35% (n=417/1,191) of never-smokers discussed at MDT were diagnosed with lung cancer. 51% (n=213/417) were recruited to OLIVE. 71% (n=151/213) were coded as never-smokers in electronic health records while 29% (n=62/213) had documentation in clinical notes only. Median age at diagnosis was 67 years (IQR 57–79). 71% (n=152/213) were female.

37% (n=80/213) were diagnosed with early-stage (30%, n=66/213 Stage I; 7%,n=14/213 Stage II), and 62% with late-stage disease (16%,n=34/213 Stage III; 46,n=99/213 Stage IV). One-year survival was 74% (n=158/213).

Comparatively, national data[1] on all adults with lung cancer report median age at diagnosis as 74.2 years (IQR 67–80, p<0.001) and 50% as female (p=0.003). 28% are diagnosed at Stage I, 8% Stage II, 12% Stage III and 43% Stage IV (p=0.925). One year-survival is 46% (p<0.001).

44% (n=520/1,191) of never-smokers had clinically significant non-malignant intrathoracic findings; this included indeterminate pulmonary nodules (n=153), infection (n=79), thymic lesions (n=66) and interstitial lung disease (n=44). 21% (n=254/1,191) were diagnosed with other malignancies including haematological (n=41), mesothelioma (n=33), gastrointestinal (n=31), gynaecological (n=31), urological (n=30) and breast (n=28).

**Discussion** In our cohort, 25% of individuals referred to lung cancer MDT were never-smokers. 35% of these were diagnosed with lung cancer. Never-smokers with lung cancer are likely to be female and younger. In our small cohort, despite having similar stage, survival is significantly better. Further research in this understudied group, including on lung cancer risk prediction, is ongoing.

## REFERENCE

1. National Lung Cancer Audit, *State of the Nation 2025*.

## P171 IS THERE ADDITIONAL DIAGNOSTIC VALUE OF STAGING COMPUTED TOMOGRAPHY (CT) TO POSITRON EMISSION TOMOGRAPHY (PET-CT) IN PATIENTS WITH SUSPECTED EARLY-STAGE LUNG CANCER?

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**Background** Lung cancer NICE (National Institute for Health and Care Excellence) guidelines suggest investigating suspected early-stage lung cancer with a contrast-enhanced staging CT imaging and PET-CT. NICE guidelines suggest contrast CT should include liver, adrenals and lower neck. In practice non-enhanced CT thorax is often followed by PET-CT, while staging CT is often omitted. There is no consensus among clinicians if staging CT adds additional diagnostic value.

**Aims** To evaluate whether staging CT demonstrates clinically relevant new findings in addition to PET-CT.

**Methods** Retrospective review of 280 patients, who underwent PET-CT for suspected primary lung cancer (March 2023 – November 2024).

**Results** The cohort included 136 males (48.6%) and 144 females (51.4%) with a mean age of 71.1 years (range: 40–95). The majority had PET staging of T1a, T1 b and T1c (220/280, 78.6%), followed by T2a and T2b (36/280,12.8%) and T3, T4 (24/280, 8.6%). Nodal staging was predominantly N0 (260/280, 92.9%), whereas 20/280 (7.1%) were Nx.

A single suspicious nodule was found in 216 patients (77.1%), while 47 (16.7%) had two nodules and 17 (6.1%) had three or more. Malignancy was confirmed histologically in 148/280 patients (52.9%) via biopsy and/or post-surgery.

CT was performed in addition to PET-CT in 160/280 patients (57.1%). Only one patient (0.6%) had significant additional findings on CT, revealing liver metastasis. However, the seven-month interval between PET-CT and CT-AP suggests disease progression rather than a missed finding.

**Conclusions** When early-stage lung cancer is suspected on thoracic CT, PET-CT demonstrates reliable diagnostic accuracy, while staging CT scan generally does not provide additional clinically significant information beyond that obtained through PET-CT.

## P172 NOVEL USE OF SOMATOSTATIN RECEPTOR IMAGING WITH 99MTC-EDDA/HYNIC-TOC IN LUNG NEUROENDOCRINE NEOPLASMS

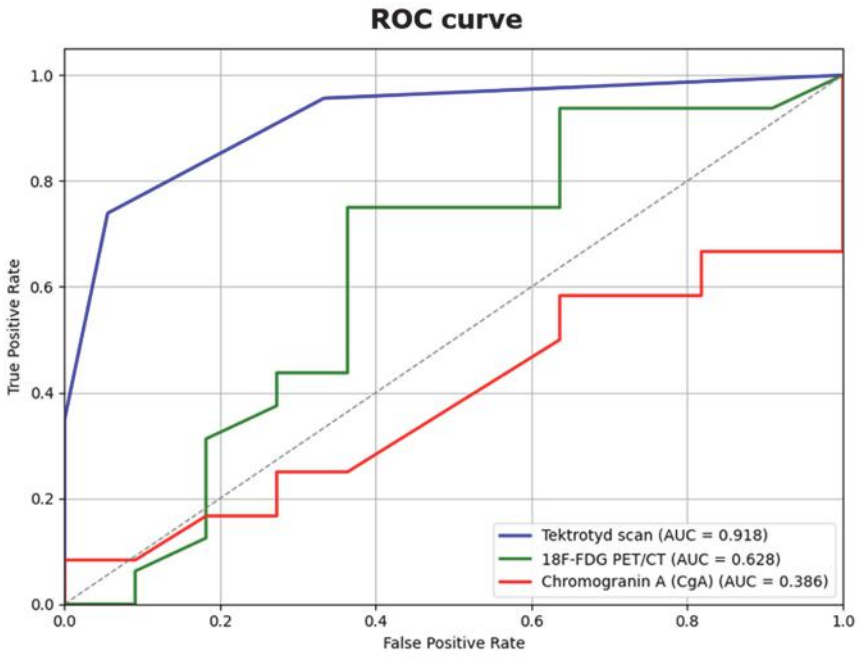
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**Introduction** Lung neuroendocrine neoplasms (NENs) are a rare group of lung cancers that often express somatostatin receptors (SSTR2&5), with potential of diagnostic and therapeutic radiolabelled somatostatin analogues (SSAs). 99mTc-EDDA/HYNIC-TOC (Tektrotyd) is a readily available SPECT-based radiolabelled SSA that offers a cost-effective alternative with improved count rate and image resolution compared to earlier SSTR imaging agents. In lung NENs, we aim to evaluate the novel use of Tektrotyd alongside conventional assessment.

**Methods** Single-centre retrospective analysis was conducted on 38 patients with 41 lung lesions (n=41) who underwent Tektrotyd SPECT between March 2012 and April 2023 for suspected lung NENs. Diagnostic performance metrics (sensitivity, specificity, PPV, NPV) and area under the ROC curve (AUC) were calculated and compared across Tektrotyd SPECT (n=41), 18F-FDG PET/CT (n=27), and serum Chromogranin A (CgA) (n=23). ROC analysis using Krenning scores (KS), SUVmax, and CgA levels as continuous predictors. Final diagnoses and outcomes were reviewed.

**Results** Among 41 diagnoses, 23 were lung NENs, all confirmed with histology. 22 of them were Tektrotyd-positive (KS $\geq$1): typical carcinoid (n=16), atypical carcinoid (n=1), metastatic carcinoid (n=4; 2 typical carcinoid, 1 large cell neuroendocrine carcinoma, 1 pancreatic carcinoid), and DIPNECH (n=1). 1 Tektrotyd-negative (KS=0): typical carcinoid (n=1). In the 18 cases without lung NENs, there were 6 false positive and 12 true negative Tektrotyd SPECT results. Diagnostic




**Diagnostic performance and ROC analysis for Tektrotyd SPECT, 18F-FDG PET/CT, and serum CgA.**

Tektrotyd SPECT: 95.65% sensitivity, 66.67% specificity, 78.57% PPV, 92.31% NPV, AUC 0.918.

18F-FDG PET/CT: 93.75% sensitivity, 9.09% specificity, 60.00% PPV, 50.00% NPV, AUC 0.628.

CgA: 41.67% sensitivity, 45.46% specificity, 45.46% PPV, 41.67% NPV, AUC 0.386.

**Abstract P172 Figure 1** Diagnostic performance and ROC analysis for Tektrotyd SPECT, 18F-FDG PET/CT, and serum CgA

performance metrics and ROC analyses for Tektrotyd SPECT, 18F-FDG PET/CT, and serum CgA are summarised in the figure 1. Most confirmed lung NENs (n=17) underwent surgical resection; others received SSA therapy, Everolimus, surveillance, or supportive chemotherapy.

**Conclusions** Tektrotyd SPECT demonstrated superior diagnostic accuracy for lung NENs compared to 18F-FDG PET/CT and CgA testing, with particularly high sensitivity and AUC. FDG uptake often occurs in the more undifferentiated lung NENs. Hence this supports the utility of Tektrotyd SPECT as a reliable and cost-effective imaging modality for the initial evaluation and staging of suspected lung NENs. If available Ga 69 DOTATE PET/CT would of use in a similar approach. The moderate specificity and PPV suggests that positive Tektrotyd results should be reviewed in the clinical context supported by additional biomarkers and ideally confirmed by histopathology.

## P173 UNDERSTANDING LATE-STAGE AND EMERGENCY LUNG CANCER PRESENTATIONS ACROSS DORSET

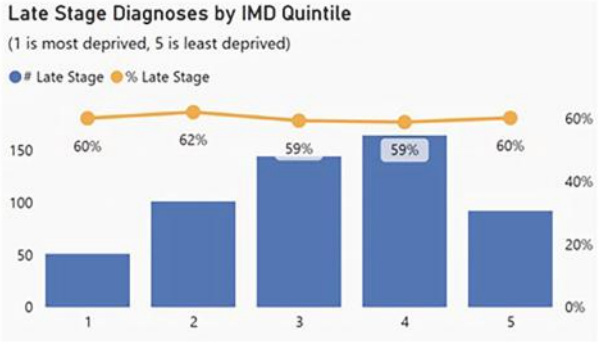
[1]S Wood, [1]E Saliu, [1]G Ruffino, [2]M Khan, [2]R Piper, [2]F Rabbani, [2]K Sann, [2]E Strawbridge, [1]H Davies, [1]J Graves. *[1]Dorset County Hospital, Dorchester, UK; [2]University Hospitals Dorset NHS Foundation Trust, Poole and Bournemouth, UK*

10.1136/thorax-2025-BTSabstracts.354

**Introduction and objectives** The 2023 National lung cancer audit highlighted that 31% of patients were diagnosed during an emergency admission and 43% of all lung cancer diagnoses were of stage IV disease, with associated poorer outcomes. Our aim was to better understand these presentations.

**Methods** We retrospectively reviewed emergency lung cancer presentations between April 2022 and December 2024 across the three acute hospitals in Dorset. We utilised the Dorset Intelligence Insight System to review population demographics within this cohort and for all stage IV lung cancer diagnoses between 2022 and 2024.

**Results** Of 410 patients diagnosed via an emergency presentation, 181/410(44%) were female, the median[IQR] age was 76 [69–83] years and WHO performance status ≤2 in 237/410 (58%). Histological diagnosis was achieved in 223/410(54%), with non-small cell lung cancer (NSCLC) accounting for 172/223(77%). Of those with NSCLC, 147/172(85%) had stage




**Abstract P173 Figure 1**

III-IV disease. 130/187(70%) of those with only a radiological diagnosis had stage III-IV disease. 40/410(10%) were offered treatment with curative intent, 222/410(54%) best supportive care. 320/410(78%) patients had died at the point of data collection.

At presentation, respiratory symptoms were reported by 248/410(60%) and 277/410(68%) had attended the emergency department at least once within 6 months of diagnosis. Only 102/410(25%) were eligible for lung cancer screening.

Only 9% of the Dorset population fall into the 20% most deprived nationally, based on index of multiple deprivation. Of the emergency presentations, all were from a primary care network with ≤2% of patients in the most deprived 20% of the population nationally. Across Dorset, 552 patients presented with stage IV disease between 2022 and 2024. There was no significant difference between percentage of stage IV diagnoses between the most and least deprived quintiles within Dorset at 51/85(60%) and 92/153(60%) respectively (graph one).

**Conclusions** Our data confirms that emergency lung cancer presentations often present with advanced disease and only 10% of patients were offered treatment with curative intent. Lung cancer screening aims to identify early stage, treatable disease, but only 25% of this cohort were eligible. Across Dorset, degree of deprivation did not affect the proportion of patients presenting with late-stage disease.

## P174 UTILISING PULMONARY REHABILITATION SERVICES TO DELIVER RAPID INTENSE PREHABILITATION FOR SELECTED LUNG CANCER PATIENTS ON DIAGNOSTIC TIMED PATHWAYS

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10.1136/thorax-2025-BTSabstracts.355

**Introduction** The benefits of prehab for lung cancer patients are accepted but services vary across England; with financial constraints potentially limiting commissioning of new services. Locally we have a cancer prehab service involving telephone assessment and signposting to local health opportunities/exercise programmes. We also utilise rapid access to pulmonary rehabilitation (PR) for patients on lung cancer pathway with potentially curable disease but clinical assessment of 'borderline fitness' for surgery. PR offers a face/face programme with tailored therapy plan including optimisation of co-existent respiratory disease. Local PR has a 33% 'drop out' rate; capacity we prioritise for referred cancer patients aiming initiation within 7 days.

**Methods** Retrospective study of patients referred for PR on lung cancer pathway June 2023 - Feb 2025. We collected PFTs, staging, performance status (PS) pre/post PR, surgical complications and total length of stay (LOS).

**Results** 31 patients were referred for PR (11 male, 20 female, mean age 70.8, all initially assessed PS1–2), 27/31 confirmed NSCLC (3 benign, 1 radiological), 7 later upstaged to 3B/4 disease. 11/31 declined PR. PR averaged 7 face/face sessions/patient (median 6) with ave 69 days referral to surgery. Within PR group mean FEV1 72%/ TLCO 59% vs no PR 71%/ 56%. 19/20 PR group were confirmed NSCLC- 7/19 improved PS on tertiary assessment,13/19 remained the same. Within the no PR group, 8/11 NSCLC- 2/8 improved PS; 4/8 unchanged; 2/8 declined. 18/20 PR group had active treatment (8 surgery, 5 SACT, 5 SABR/radical radiotherapy) 2 declined. Within no PR group 7 surgery, 1 declined. All surgical procedures were VATS, with average LOS 6.1 days PR group vs 7.3 days no PR.

**Conclusions** Many patients attending PR had improvement in PS (37%) over the diagnostic pathway with no PS decline (25% PS decline in refusal group) and an average lower surgical length of stay. We feel the preserved PFTs but clinical assessment of 'borderline fitness' points to significant deconditioning which may be improved by the intensive nature of PR.

With current financial restraints we have found rapidly available capacity in an established respiratory service that could improve patients' lung cancer treatment and outcomes.

## P175 USING DYNAMIC 19F MRI OF LUNG VENTILATION PROPERTIES TO PRIORITISE FUNCTIONAL LUNG IN RADIOTHERAPY TREATMENT PLANNING FOR NON-SMALL CELL LUNG CANCER

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10.1136/thorax-2025-BTSabstracts.356

**Introduction** Patient harm may be reduced by directing radiation dose away from well-functioning lung tissue. Radiotherapy planning requires a balance between ensuring adequate dose to the tumour whilst minimising dose exposure of susceptible normal tissues. Radiation induced lung injury, a consequence of radiotherapy, occurs in 5–50% of non-small cell lung cancer (NSCLC) patients, causing serious sequelae.[1]

Regional lung function for radiotherapy planning has been explored using CT/PET, 4DCT and 129Xe-MRI.[2]19F MRI of inhaled perfluoropropane (PFP) is an emerging method for quantitative imaging of ventilation that does not use ionising radiation and has a lower barrier to entry than hyperpolarized gas MRI. We report the first assessment of 19F-MRI to inform radiotherapy planning.

**Methods** Multiple breath dynamic 19F-MRI of inhaled 79% PFP/21% oxygen gas mixture was performed in 12 NSCLC patients before radiotherapy. Regional Lung Clearance Index (RLCI; number of breaths required to turnover 97.5% of gas in each image voxel) was calculated across lung volume to give a quantitative regional ventilation map. Lung was stratified into 'well', 'moderately' and 'poorly' ventilated regions.

'Functional avoidance plans' were created, using the stratified RLCI mapped in Raystation to participant radiotherapy planning CT scans. Radiation doses delivered to lung were measured in both standard and functional avoidance plans.

**Results** 19F MRI scans were acquired from 12 NSCLC patients and RLCI maps created. 6 participants have had

standard and avoidance plans created to date, showing a mean change of -14.48% (range -33.1 to +10.1) in volume of well-ventilated lung within the tissue receiving 20 Gy or more radiation.

**Conclusions** Maps of lung ventilation from dynamic 19F MRI can stratify lung tissue according to ventilation and be used in treatment planning of NSCLC patients. This has potential for improved sparing of well-ventilated lung tissue in cancer patients. This tool can advance our understanding of lung ventilation patterns in patients, with the aim of improving clinical outcomes such as incidence, severity, and persistence of radiation induced lung injury.

## REFERENCE

1. Bucknell NW, *et al*. Functional lung imaging in radiation therapy for lung cancer: a systematic review and meta-analysis. *Radiotherapy and Oncology*. 2018(129) 2.

## P176 CENTRAL AIRWAY OBSTRUCTION: PATIENT, CARER AND PUBLIC PERSPECTIVES

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10.1136/thorax-2025-BTSabstracts.357

**Background** Malignant central airways obstruction (CAO) is present in ~10% of patients newly diagnosed with lung cancer and is a medical emergency. Lung cancer patients with CAO have poorer outcomes than those without. Despite this, the optimal management of CAO is poorly defined and research in this patient population is scarce. Therefore we engaged with CAO patients and people being screened for lung cancer to explore their willingness to participate in clinical trials.

**Methods** Telephone interviews were conducted in patients and carers with lived experience of CAO (N=4) and participants being screened for lung cancer (N=5). Themes covered among patient with CAO and their carers were their lived experience of CAO; the impact of disease on carers; willingness to participate in randomised controlled studies where treatment may not be received; and key outcomes that should be studied.

**Results** The main symptom described by CAO patients was breathlessness and associated fear ('suffocating', 'couldn't breath at all', 'never experienced such fear', 'thought was going to die'). Patients thought clinical trials were vital and essential, and were willing to participate, Key trial outcomes identified were improved a cough and breathlessness. Barriers to participation included additional blood tests and the fear of the unknown. Interviewees voiced concerns over being judged for not wanting to partake.

Carers reflected on the overnight transition from 'being a partner to a carer', and the 'helplessness'. A common theme was the importance of communications throughout, and the positive impact this had despite the gravity of the situation.

**Conclusions** We found CAO patients were willing to be involved in study design and participate in clinical trials. Overcoming identified barriers could improve uptake in future trials.

## P177 DOUBLE JEOPARDY: ESCALATING MORTALITY TRENDS AND DISPARITIES IN LUNG CANCER PATIENTS WITH SEPSIS – A RETROSPECTIVE EPIDEMIOLOGICAL STUDY

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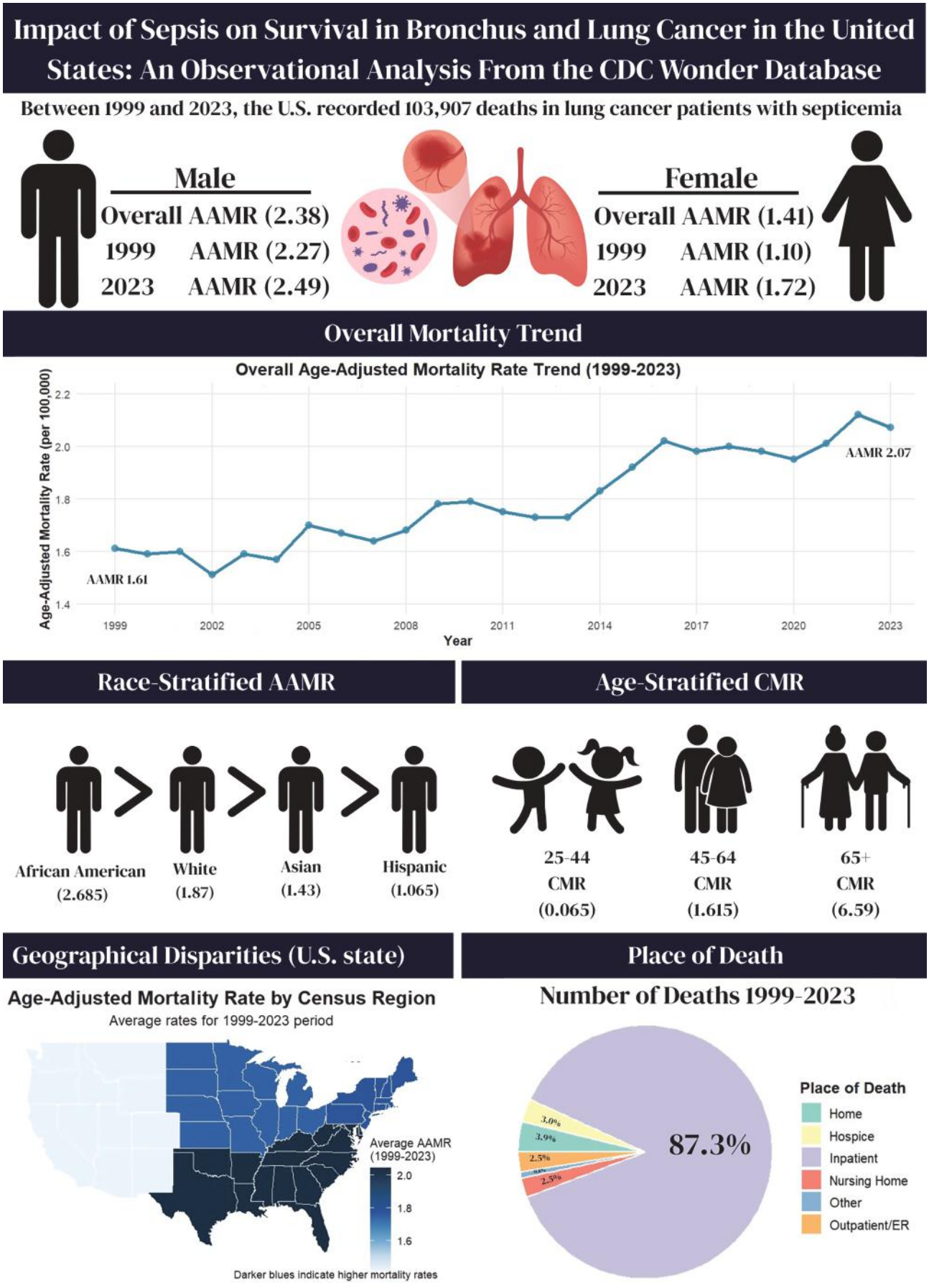
10.1136/thorax-2025-BTSabstracts.358

**Background** Lung cancer (LC) remains the leading cause of cancer-related mortality globally. Studies indicate LC patients with sepsis have significantly lower survival rates, yet the temporal trends and disparities in mortality remain unexplored. We examined mortality trends and disparities of LC complicated by sepsis across different sociodemographic strata.

**Methods** This retrospective study analysed death certificate data from the Centers for Disease Control and Prevention's Wide-Ranging Online Data for Epidemiologic Research database from 1999–2023, for individuals ≥25 years with multiple causes of death listed as lung/bronchus cancer (ICD-10: C34) with sepsis. Age-adjusted mortality rates (AAMRs) per 100,000 population were calculated and stratified by sex, race/ethnicity, and geographic region, with Joinpoint regression analysis to determine the change in mortality trends.

**Results** From 1999–2023, 103,907 deaths were attributed to LC with co-existing sepsis, demonstrating increasing mortality trends throughout the study period. AAMRs rose significantly from 1.61 in 1999 to 2.07 in 2023 (AAPC: 1.32%; $p<0.000001$). Sex-stratified analysis revealed men had consistently higher overall AAMRs (2.32/100,000) with an AAPC of 0.49% (95% CI: -0.12 to 1.12; $p=0.12$) compared to women (1.37/100,000) with an AAPC of 1.94% (95% CI: 1.74 to 2.14; $p<0.000001$). Patients aged ≥65 years exhibited the highest mortality rates (6.59%). Racially, non-Hispanic (NH) Black individuals had the highest overall AAMRs with increasing mortality patterns (AAMRs: 2.57/100,000; AAPC: 0.32%; 95% CI: 0.045 to 0.599; $p=0.02$), while Hispanic populations recorded the lowest AAMR (1.01/100,000; AAPC: 0.61%; 95% CI: 0.156 to 1.068; $p=0.01$). Regionally, the South demonstrated the highest mortality (AAMRs: 2.04), followed by the Northeast (AAMRs: 1.78), Midwest (AAMRs: 1.75), and West (AAMRs: 1.42). Most deaths occurred in inpatient medical facilities (90,425 deaths; 87%).

**Conclusion** Mortality rates increased significantly over the study period with a significant increase during recent years. Notable disparities across sex, race/ethnicity, and geographic regions were observed, with NH Black populations and Southern states showed highest mortality rates. These findings highlight the urgent need for targeted sepsis prevention protocols in LC patients, enhanced surveillance systems for high-risk demographics, and comprehensive region-specific interventions to address underlying healthcare disparities and improve clinical outcomes across all populations.




**Abstract P177 Figure 1**

# Respiratory infections: pathogens to patients

## P178 CLINICAL AND BIOCHEMICAL FEATURES OF ADULTS HOSPITALISED WITH HUMAN METAPNEUMOVIRUS (HMPV): COMPARISON WITH INFLUENZA A AND RSV, INSIGHTS FROM THE UNIVERSAL STUDY

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10.1136/thorax-2025-BTSabstracts.359

**Background** In winter 2024–25, surges in hMPV activity renewed public health interest, but no targeted therapy or vaccine is currently available. Better understanding of hMPV's clinical features may aid diagnosis and treatment development.
**Objectives** To characterise clinical and biochemical features of adults hospitalised with hMPV compared with influenza A and RSV.
**Methods** The UNIVERSAL study is a UK multicentre prospective cohort investigating adults hospitalised with respiratory viral infections. Adults with acute respiratory infection underwent PCR testing. Clinical, radiological, and laboratory data were collected prospectively. Patients with confirmed hMPV, influenza A, or RSV were included.
**Results** Between August 2022 and May 2025, 186 influenza A, 110 RSV, and 27 hMPV cases were included. Median age was 71 years (IQR 60–77) in hMPV, 68 (IQR 58–75) in RSV, and 64 (IQR 55–74) in influenza A, p = 0.063. Influenza vaccination was reported in 88/185 (48%) influenza A, 80/110 (73%) RSV, and 22/26 (85%) hMPV patients, p < 0.001. Five COVID-19 vaccines were recorded in 16/26 (62%) hMPV versus 47/108 (44%) RSV and 48/184 (26%) influenza A, p = 0.001. Median Charlson Comorbidity Index was highest in RSV 7 (IQR 4–13) versus influenza A 4 (IQR 4–10) and hMPV 5 (IQR 4–12), p = 0.047.

Median neutrophil-to-lymphocyte ratio was lowest in hMPV 5 (IQR 3–7) versus influenza A 8 (IQR 4–13) and RSV 7 (IQR 5–14), p = 0.040. Median lymphocyte count was highest in hMPV (1.10 $\times 10^9$/L, IQR 0.70–2.00), p = 0.032.

Radiographic consolidation was reported in 8/21 (38%) hMPV versus 21/85 (25%) RSV and 34/136 (25%) influenza A patients, (p = 0.419). Median hospital stay was shortest in hMPV, 3 days (IQR 2–5) versus RSV 4 (IQR 3–7) and influenza 4 (IQR 2–6), p = 0.039. No hMPV patients required HDU/ICU versus RSV 4/106 (3.8%) and influenza 9/174 (5.2%). Mortality at 60 days occurred in 3/101 RSV (3.0%) and 5/166 influenza (3.0%), with 0/23 (0%) in hMPV.
**Conclusions** HMPV was associated with less disruption of clinical biomarkers, shorter hospital stay, and fewer severe outcomes. These findings support virus-specific phenotyping to improve care pathways.

**Abstract P178 Table 1** Patient characteristics by virus – influenza A, RSV and hMPV

| Characteristic | Influenza A N = 186[1] | RSV N = 110[1] | hMPV N = 27[1] | p-value[2] |
|---|---|---|---|---|
| Age (years) | 64 (55, 74) | 68 (58, 75) | 71 (60, 77) | 0.063 |
| Influenza vaccine | 88/185 (48%) | 80/110 (73%) | 22/26 (85%) | <0.001 |
| Five COVID-19 vaccines | 48/184 (26%) | 47/108 (44%) | 16/26 (62%) | 0.001 |
| Charlson comorbidity score | 4 (4, 10) | 7 (4, 13) | 5 (4, 12) | 0.047 |
| Neutrophil to lymphocyte ratio | 8 (4, 13) | 7 (5, 14) | 5 (3, 7) | 0.040 |
| Lymphocyte count (x $10^9$/L) | 0.90 (0.60, 1.40) | 1.05 (0.70, 1.70) | 1.10 (0.70, 2.00) | 0.032 |
| Consolidation on CXR | 34/136 (25%) | 21/85 (25%) | 8/21 (38%) | 0.419 |
| GICU admission | 6/174 (3.4%) | 0/106 (0%) | 0/25 (0%) | 0.183 |
| RHDU admission | 3/174 (1.7%) | 4/106 (3.8%) | 0/25 (0%) | 0.577 |
| Length of stay (days) | 4.0 (2.0, 6.0) | 4.0 (3.0, 7.0) | 3.0 (2.0, 5.0) | 0.039 |

[1]Median (Q1, Q3); n/N (%)

[2]Kruskal-Wallis test for continuous variables, Chi-squared or Fisher's exact test for categorical variables, as appropriate.

## P179 ASTHMA RESHAPES THE IN-HOSPITAL PHENOTYPE OF ADULTS WITH RESPIRATORY VIRAL INFECTION: HEIGHTENED SYMPTOMATOLOGY BUT ATTENUATED SEVERITY

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**Background** Respiratory Viral Infections are common cause of asthma exacerbations. However, whether pre-existing asthma worsens, mitigates or merely changes the clinical expression of respiratory viral infections (RVIs) in hospitalised adults remains uncertain. We analysed prospectively collected data from the multicentre UK UNIVERSAL study – a prospective observational cohort spanning 12 hospitals – to compare symptom burden, inflammatory profile and outcomes in adults with and without asthma.

**Methods** Consecutive adults (≥18 years) prospectively enrolled on admission between Aug 2022 and May 2025 with PCR-confirmed RVI were stratified by clinician-diagnosed asthma. Symptom burden at admission was assessed with FLU-PRO Plus (0–100, six domains). Physiological severity was recorded daily using the WHO Ordinal Scale for Clinical Improvement (OSCI, 0–8). Primary outcomes were (i) occurrence of OSCI ≥ 5 and (ii) number of days with OSCI ≥ 5. Secondary outcomes were ICU/HDU admission and 60-day all-cause mortality. Categorical variables were compared with Fisher's exact test, continuous variables with the Mann-Whitney U test.

**Results** Of 836 patients, 306 (36.6%) had asthma. Baseline characteristics are summarised in table 1.

Asthmatics were younger (median 57 vs 67 y), more frequently female (68% vs 44%), and more often infected with rhinovirus/enterovirus (25% vs 17%; all $p<0.05$). Systemic inflammation was lower in asthmatics (CRP > 10 mg $L^{-1}$ 71% vs 86% $p<0.001$ ; lymphopenia 38% vs 50% $p<0.004$), while eosinophils $\geq 0.20\times10^9$/L was more frequent (25.4% vs 18.8%, $p=0.025$).

Asthma patients reported significantly greater symptom burden, with higher median FLU PRO Plus scores in the chest/respiratory (60.9 vs 52.2, $p<0.001$), nose (36.0 vs 29.2, $p<0.001$), and systemic domains (39.5 vs 33.8, $p=0.002$).

Despite higher symptom burden, asthma patients were less likely to reach OSCI ≥5 (61.4% vs 72.0%, $p=0.002$),

**Abstract P179 Table 1** Patient characteristics by asthma status

| Characteristic | Asthma (n = 306) | No Asthma (n = 530) | p-value |
|---|---|---|---|
| **N** | 306 | 530 | |
| **Age, median [IQR] (years)** | 57 [44–71] | 67 [60–77] | <0.001 |
| **Female sex, n (%)** | 207 (67.9%) | 235 (44.3%) | <0.001 |
| **Viruses** | | | |
| **SARS-CoV-2** | 77 (25.2%) | 140 (26.4%) | |
| **Influenza** | 59 (19.3%) | 144 (27.2%) | |
| **RhV/EV** | 77 (25.2%) | 89 (16.8%) | 0.021 |
| **RSV** | 51 (16.7%) | 60 (11.3%) | |
| **Other viruses** | 31 (10.1%) | 61 (11.5%) | |
| **Co-infection** | 11 (3.6%) | 36 (6.8%) | |
| **CRP >10 mg/L, n (%)** | 201 (71.0%) | 429 (85.8%) | <0.001 |
| **Symptom duration prior to hospitilisation, median [IQR] (days)** | 6.1 [3.0–7.0] | 6.7 [3.0–7.0] | 0.124 |
| **OSCI ≥5 during admission, n (%)** | 188 (61.4%) | 381 (72.0%) | 0.002 |
| **Calendar days OSCI ≥5** | | | |
| **0 days,** | 118 (38.6%) | 148 (28.0%) | |
| **1–2 days** | 108 (35.3%) | 208 (39.3%) | 0.015 |
| **3–4 days** | 42 (13.7%) | 87 (16.4%) | |
| **≥5 days** | 38 (12.4%) | 86 (16.3%) | |
| **60-day mortality, n (%)** | 3 (1.1%) | 24 (5.1%) | 0.006 |
| **ICU/HDU admission, n (%)** | 8 (2.8%) | 15 (3.0%) | 0.922 |

experienced fewer calendar days with OSCI ≥5 (≥5 days: 12.4% vs 16.3%, p=0.015), and had lower 60-day mortality (1.1% vs 5.1%, p=0.006). ICU/HDU admission rates were similar (2.8% vs 3.0%, p=0.922).

**Conclusion** Asthma was associated with a distinct clinical profile characterised by increased upper and lower respiratory symptom burden but lower systemic inflammation and reduced short-term mortality.

## P180 THE IMPACT OF STEROIDS & IMMUNOSUPPRESSION ON OUTCOMES IN PATIENTS WITH PNEUMOCYSTIS JIROVECII PNEUMONIA (PJP): A RETROSPECTIVE ANALYSIS AT A LARGE TERTIARY CENTRE

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10.1136/thorax-2025-BTSabstracts.361

**Background** Pneumocystis jirovecii pneumonia (PJP) is a significant opportunistic infection in immunocompromised patients. This study explores whether steroid use or underlying immunosuppression influences the likelihood of developing PJP and affects clinical outcomes, particularly morbidity and mortality.

**Methods** A 5-year retrospective analysis was conducted of patients diagnosed with PJP between 2019–2024. Data collected included age, comorbidities (Charlson Comorbidity Index), lymphocyte count, steroid use and duration, immunosuppression status, number of hospitalisations, and total days spent in hospital. Mortality data were recorded. Binary variables were derived for immunosuppression and oral corticosteroid (OCS) exposure. Correlation analysis was performed to explore associations with clinical outcomes.

**Results** 73 patients were diagnosed with PJP pneumonia in the time period and the mean age was 65 years. 58% (n=42) died during or after their admission with PCP. 35% (n=25) were oncology patients. 62% of patients received a form of immunosuppressive therapy, and 66% received OCS specifically. Immunosuppression and steroid use showed a moderate correlation (r = 0.30). A weak correlation was observed between immunosuppression and mortality (r = 0.10), with negligible correlation between steroid use and mortality (r = 0.02). No strong linear relationship was observed between steroid duration and hospital stay or mortality. moderate positive correlation (r = 0.34) was found between Charlson Comorbidity Index and mortality, suggesting that higher comorbidity may increase mortality risk. Neither steroid use nor immunosuppression was significantly associated with mortality in logistic regression (p > 0.05). Predictive modelling (random forest) indicated that age and comorbidity index were the strongest predictors of mortality.

**Conclusion** In patients with PJP, neither immunosuppressive therapy or OCS showed a strong linear association with mortality or morbidity. Comorbidity index and age were identified as stronger predictors of mortality. These findings suggest that comorbid conditions may be more critical in predicting poor outcomes in PJP than steroid therapy or immunosuppression alone. Further multivariable modelling is needed to identify independent risk factors for poor outcomes in this population.

## P181 WHAT IS THE VALUE OF REPEAT CHEST RADIOGRAPHY IN THE RECOVERY OF PATIENTS HOSPITALISED WITH COMMUNITY-ACQUIRED PNEUMONIA?

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10.1136/thorax-2025-BTSabstracts.362

**Introduction** The value of performing a follow up chest X-ray (CXR) after hospital admission with community-acquired pneumonia (CAP) is debated; currently randomised-controlled trial evidence is lacking. BTS guidelines advise those with persistent symptoms, or a higher malignancy risk warrant a CXR 6 weeks after discharge.

This single-centre study from a large UK teaching hospital describes follow up CXR outcomes from a cohort of CAP patients managed by the local Respiratory Infections Team (RIT) over a one-year period.

**Methods** A retrospective database review was conducted of consecutive patients admitted with confirmed CAP and followed up by the RIT between October 2023 and October 2024. Patient records and imaging reviewed to identify outcomes.

**Results** 1034 patients were identified, with 995 surviving from hospital discharge to CXR and RIT follow up. Of these, 163

**Abstract P181 Table 1** A summary of new respiratory diagnoses found through follow up

| New diagnosis | Number of patients |
|---|---|
| Lung cancer including adenocarcinoma, small-cell lung cancer and mesothelioma | 7 |
| Interstitial lung disease (or evidence of) | 7 |
| Pulmonary oedema/heart failure | 4 |
| Benign lung nodule | 2 |
| Bronchiectasis | 2 |
| Asbestos exposure | 1 |
| Chronic aspiration | 1 |
| Persistent empyema | 1 |
| Pneumothorax | 1 |

(16.4%) were identified as having residual radiographic changes.

48 (4.8%) did not attend follow up because of choice, area or other health reasons.

Through follow up, 26/995 (2.6%) patients were found to have a new underlying pathology requiring further investigation. 9 (0.9%) were referred on for cancer investigations with 7 confirmed lung cancers. Of these, 6 were smokers or had significant smoking history. Other new respiratory disease diagnosed included interstitial lung disease, bronchiectasis and persistent empyema (table 1).

For those with persisting CXR abnormalities, symptoms resolved in 73/163 (44.8%), and the persistent changes were deemed clinically non-significant following MDT discussion. 61/163 (37.4%) had further interval radiographic surveillance and 29/163 (17.8%) had further respiratory specialty input.

Of those that had further imaging, 15/61 (24.6%) resolved by second CXR and 21/61 (34.4%) showed clinical resolution with residual non-significant CXR change, so were discharged. Slow resolvers tended to be current smokers (42/163, 25.8%) or have significant smoking history (78/163, 47.9%), and 55/163 (33.7%) patients had COPD. Median age of slow resolvers was 73 compared to 70 of the whole cohort (p=0.004).

**Conclusion** Follow up CXR at 6 weeks for CAP identified a new significant respiratory diagnosis in 2.6% of patients, of which lung cancer was identified in 0.7%. Most patients with ongoing CXR abnormalities at 6 weeks resolve on subsequent imaging.

## P182 CHEST RADIOGRAPHIC CONSOLIDATION IN ADULTS HOSPITALISED WITH RESPIRATORY VIRAL INFECTIONS (RVI): PREVALENCE AND PREDICTORS

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10.1136/thorax-2025-BTSabstracts.363

**Background** Chest radiographic consolidation is a marker of severe viral infection and a key factor in hospital management. Rates of consolidation and risk factors across RVI in adults remain poorly defined.

**Objectives** To identify prevalence and clinical and laboratory predictors of consolidation in adults with RVI in a multicentre UK study cohort

**Abstract P182 Table 1** Characteristics of patients based on presence or absence of consolidation on chest radiograph

| Characteristic | No consolidation N = 376[1] | Consolidation N = 157[1] | p-value[2] |
|---|---|---|---|
| **Viral group** | | | 0.170 |
| Influenza | 96 (26%) | 32 (20%) | |
| SARS-CoV-2 | 109 (29%) | 52 (33%) | |
| RSV | 63 (17%) | 21 (13%) | |
| Rhino/enterovirus | 75 (20%) | 29 (18%) | |
| Other viruses | 33 (8.8%) | 23 (15%) | |
| **Age (years)** | 65 (53, 76) | 70 (58, 76) | 0.043 |
| **Male sex** | 161/376 (43%) | 88/157 (56%) | 0.006 |
| **Pneumococcal vaccination** | 144/370 (39%) | 82/153 (54%) | 0.002 |
| **GICU admission** | 0/375 (0%) | 10/156 (6.4%) | <0.001 |
| **Oxygen requirement on admission (OSCI>4)** | 205/375 (55%) | 109/157 (69%) | 0.001 |
| **Length of stay (days)** | 4.0 (2.0, 7.0) | 5.0 (3.0, 9.0) | <0.001 |
| **60-day mortality** | 8/365 (2.2%) | 10/153 (6.5%) | 0.014 |
| **Charlson comorbidity score** | 4 (4, 12) | 7 (4, 13) | 0.359 |
| **C reactive protein (mg/L)** | 33 (12, 77) | 113 (34, 198) | <0.001 |
| **Neutrophil-to-lymphocyte ratio** | 7 (4, 12) | 9 (6, 15) | <0.001 |

[1]n/N (%); Median (Q1, Q3)

[2] Wilcoxon rank sum test for continuous variables, Pearson's Chi-squared test or Fisher's exact test for categorical variables, as appropriate.

OSCI = Ordinal scale for clinical improvement

**Methods** Analysis of the UNIVERSAL UK multicentre cohort of adults hospitalised with PCR-confirmed RVI. Admission chest radiographs were reviewed by clinical radiology staff and binarised for consolidation. Univariable and multivariable logistic regression were used to identify predictors.
**Results** Between August 2022 and May 2025, 533 patients with RVI had chest radiography data. Consolidation was reported in 157/533 (29%) and was evenly distributed across viral groups (p = 0.170). Proportion with consolidation by viral group: influenza 32 (20%), SARS-CoV-2 52 (33%), RSV 21 (13%), rhino/enterovirus 29 (18%) and other viruses (Human metapneumovirus, coronaviruses, adenovirus and parainfluenza) 23 (15%).

Median age was 70 (IQR 58–76) with consolidation versus 65 (IQR 53–76) without, p = 0.043. Consolidation was more common males 88/157 (56%) versus 161/376 (43%), p = 0.006 and in those vaccinated against pneumococcus 82/153 (54%) versus 144/370 (39%), p = 0.002.

Consolidation was associated with ICU admission 10/156 (6.4%) versus 0/376 (0%), p < 0.001, oxygen requirement on admission 109/157 (69%) versus 205/375 (55%), p = 0.001, longer hospital stay (median 5 versus 4 days, p <0.001), and 60-day mortality 10/153 (6.5%) versus 8/365 (2.2%), p = 0.014. Median Charlson co-morbidity index was 9 (IQR 6–15) with consolidation versus 7 (IQR 4–12) without, p = 0.359.

Median CRP and neutrophil-to-lymphocyte ratio (NLR) were higher with consolidation, CRP 113 versus 33 mg/L, NLR 9 versus 7, both p < 0.001.

Multivariable analysis identified pneumococcal vaccination (OR 1.89, 95% CI 1.20–3.00, p = 0.006) and CRP >50 mg/L (OR 4.64, 95% CI 2.30–10.10, p < 0.001) as predictors of consolidation.

Only 47/157 (30%) with consolidation had pneumonia listed as primary diagnosis on discharge (kappa = 0.265).
**Conclusion** Consolidation was seen across RVIs and linked to inflammation and poor outcomes. Concordance with discharge diagnoses was limited, with possible implications for health economics.

## P183 ENHANCING ANTIBIOTIC EFFECTIVENESS USING THE REPURPOSED MULTIPLE SCLEROSIS DRUG, GLATIRAMER ACETATE, TO COMPROMISE MEMBRANE INTEGRITY OF CF-RELEVANT PATHOGENIC BACTERIA

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10.1136/thorax-2025-BTSabstracts.364

**Objectives** People with Cystic Fibrosis (pwCF) often develop persistent lung infections caused by Pseudomonas aeruginosa (Pa) that are hard to treat. Creating new antibiotics faces several obstacles; difficult, costly and time-consuming. Thus, finding new uses for approved medications is a priority, as highlighted by the LifeArc Translational Challenge programme. Glatiramer acetate (GA), currently used to treat multiple sclerosis, is similar in structure to antimicrobial peptides such as LL-37. Our research showed enhanced killing of Pa when combining GA with tobramycin (TOB).[1] This study aims to investigate the GA-TOB impact on other CF pathogens, with this portion of the study looking at GA's effect on membrane integrity of clinical strains of Pa as well as other CF-relevant pathogens.
**Methods** We assessed depolarisation of the cytoplasmic membrane via the release of the fluorescent dye, DiSC3(5), on a panel of CF relevant bacterial pathogens. We compared these profiles to the antimicrobial peptide LL-37, and antibiotics TOB and colistin.
**Results** We have shown that the enhanced killing of Pa reference and clinical strains when co-treated with GA (50 µg/ml) and TOB is mediated by membrane disruption. Here we show that GA is shown to have an effect on the depolarisation of cytoplasmic membrane of Pa, Gram-negative bacteria and the Gram-positive bacteria, Staphylococcus aureus. GA also depolarised the cytoplasmic membrane across the panel of 9 species including Acinetobacter baylyi, A. baumannii, and Pseudomonas protogens. Preliminary data shows that all three of the Pa clinical isolates tested achieved membrane depolarisation with GA.
**Conclusions** GA depolarised the cytoplasmic membranes of clinically relevant CF pathogens as well as reference strains and clinical isolates of Pa.

The ability of GA to disturb the membrane of other clinically relevant CF pathogens in addition to Pa suggests it will act similarly as an antibiotic resistance breaker, by allowing increased intracellular access of antimicrobials such as TOB, expanding the potential of GA to combat bacterial airway infections.

### REFERENCE

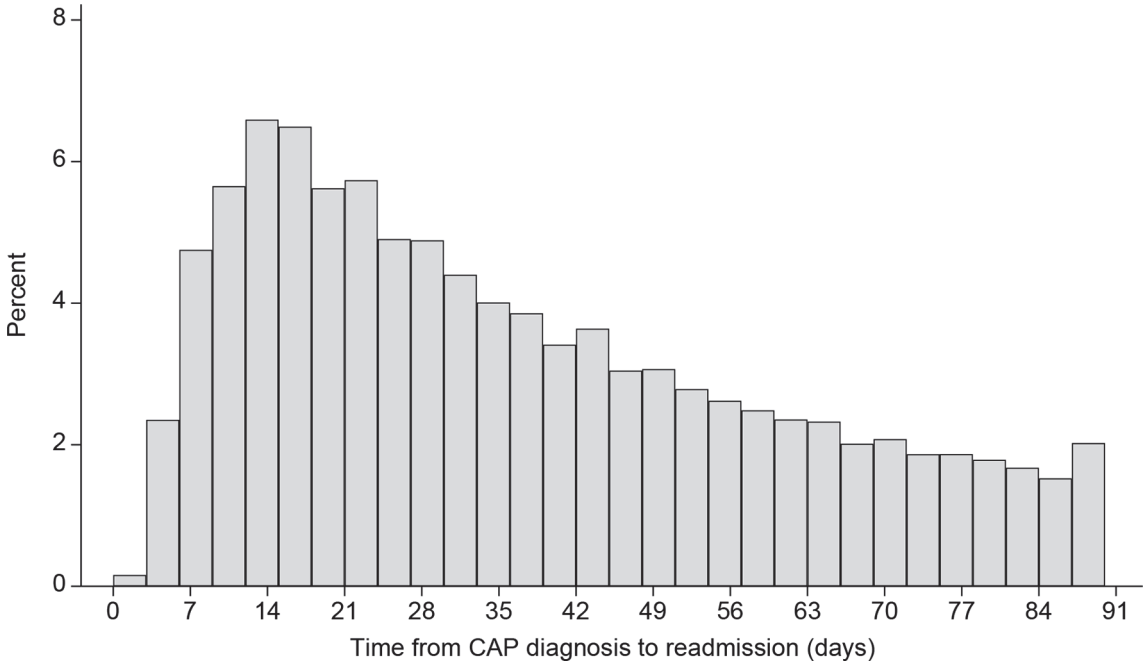
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## P184 READMISSIONS AND MORTALITY FOLLOWING INDEX HOSPITALISATION FOR COMMUNITY-ACQUIRED PNEUMONIA: A POPULATION-BASED ANALYSIS USING HES DATA

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10.1136/thorax-2025-BTSabstracts.365

**Introduction** Hospital readmission following an index admission for community-acquired pneumonia (CAP) remains a significant clinical and healthcare burden in the UK, with the BTS Pneumonia audit reporting rates of 16%.[1] However, limited data exists on risk factors and outcomes associated with readmission. This study characterises patient and disease features linked to readmission following a CAP admission in a large extract of Hospital Episode Statistics (HES) data (Health and Social Care Information Centre).[2]
**Methods** HES data were used to identify adults (≥18 years) admitted to English hospitals with a diagnosis code of pneumonia (J12-J18) in 2018. Patients who died during their index admission were excluded. All-cause readmission and in-hospital mortality within 90 days post-discharge was assessed. Baseline demographics and comorbidities were described.
**Results** Of 227,213 patients (85%) surviving an index CAP admission, 67,536 (30%) were readmitted within 90 days. The median time from CAP diagnosis date to readmission was 31 days [IQR 17–53] (figure 1). The median readmission stay was 2 days [IQR 0–9].




**Abstract P184 Figure 1** Histogram of time from index CAP diagnosis to readmission

Baseline characteristics were broadly similar between readmitted and non-readmitted patients (mean age 72 vs. 71 years, males 52% vs. 49% respectively). Minor differences in comorbidities were observed: IHD (25% vs. 20%), diabetes (24% vs. 21%), PVD (7% vs. 5%), hypertension (50% vs. 46%) and COPD (10% vs. 6%). Readmitted patients had a marginally longer index stay (median=7 days, IQR 3–13) than non-readmitted patients (median=6 days, IQR 3–15). In-hospital mortality among readmitted patients was 10%.

**Conclusion** Patients hospitalised with CAP are at increased risk of readmission within 90 days, affecting nearly one in three surviving an index admission. Readmission risk appears difficult to predict from comorbidity or demographics. Given the substantial mortality risk in this cohort, reducing readmission should be a key focus in post-discharge care for CAP patients. Moving forward, we plan to describe trends in readmission and the impact of frailty.

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2. Hospital Episodes of Statistics. Copyright © 2025, re-used with the permission of The Health & Social Care Information Centre. All rights reserved.

## P185 INCIDENCE OF DIABETES MELLITUS FOLLOWING HOSPITALISATION WITH INFLUENZA

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10.1136/thorax-2025-BTSabstracts.366

**Rationale** The incidence of diabetes mellitus (DM) after hospitalisation with influenza in England is unknown.

**Objectives** To establish the incidence of developing DM post hospitalisation with influenza using Clinical Practice Research Datalink (CPRD) linked to Hospital Episode Statistics (HES) in England.

**Methods** Adults with a first episode of hospitalised influenza as the primary cause for admission between 01/07/2004 till 01/03/2021 based on ICD-10 codes without pre-existing DM were included. Patients were followed from one day after discharge till either DM diagnosis, death or end of GP record. A randomly selected group matched for age, sex and GP practice and an un-matched group of hospitalised sepsis patients were used as comparator groups. Missing data was imputed using multiple imputation model. Hazard ratios for outcome measures were calculated using Cox regression models and adjusted for potential confounders.

**Results** 13,710 patients following influenza, 42.5% male, median age 57.6 (range 18 to 103) had a DM incidence of 12.5 per 1000 person years. Adjusted hazard ratios (aHR) for developing DM compared to non-hospitalised controls was 1.56 (95%CI 1.41–1.72, $p<0.001$) and to hospitalised sepsis comparators (1.12 (95% CI 1.02–1.24, $p=0.023$)). The greatest risk for developing DM in hospitalised influenza patients was within 90 days of discharge (aHR 2.80 (95% CI 2.01–3.89), $p<0.001$) compared to non-hospitalised controls. Risk factors for DM included males, pre-existing DM risk, obesity and critical care admission during acute illness.

**Conclusion** Patients hospitalised with influenza had a greater incidence of DM when compared to both matched controls and patients following hospitalisation with sepsis.

P186 **PROSPECTIVE STUDY OF ANTIMICROBIAL RESISTANCE IN CHRONIC LUNG DISEASE (PRESIDE) – A NEW GLOBAL REGISTRY. PROTOCOL AND PILOT PHASE RESULTS**

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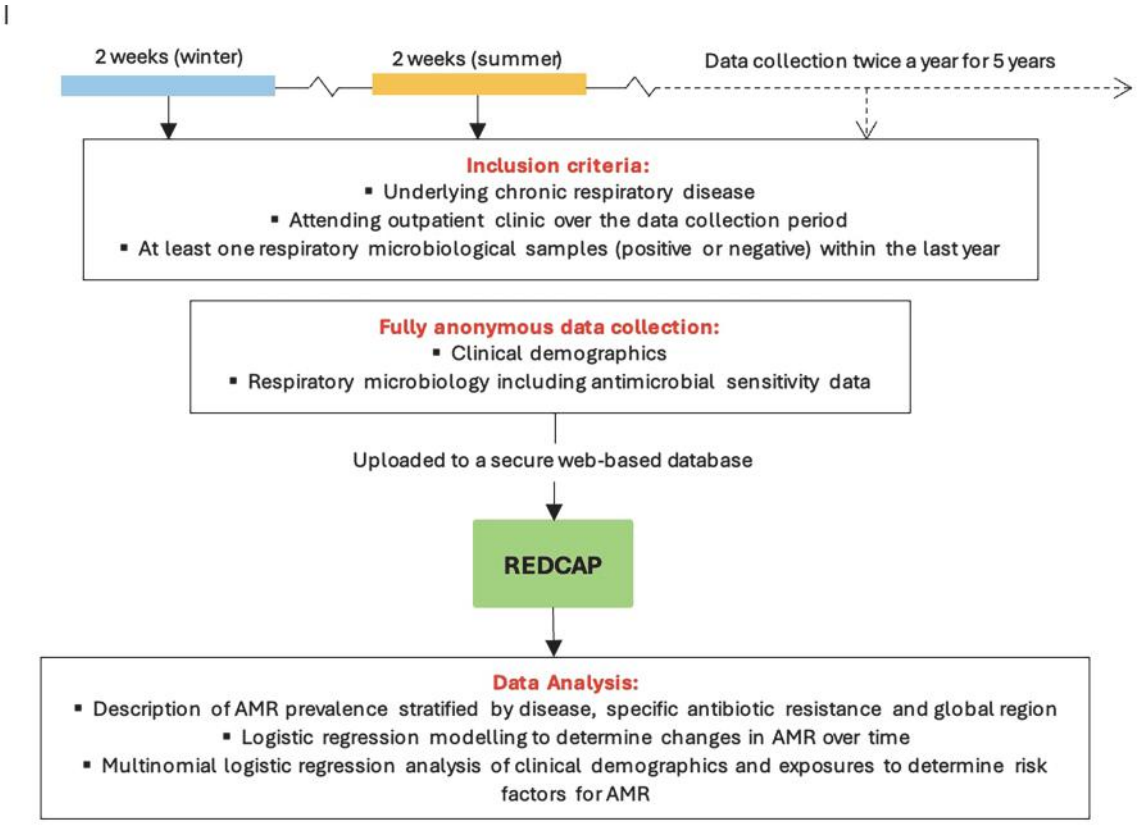
10.1136/thorax-2025-BTSabstracts.367

**Introduction** Antimicrobial resistance (AMR) is an escalating global health threat and WHO priority. Patients with chronic respiratory disease are vulnerable to AMR due to recurrent infection and colonisation with high priority pathogens. Most epidemiological data on AMR is retrospective and single centre, limiting estimations regarding prevalence, burden and impact. To tackle this problem, we have set up a prospective, global registry into AMR as part of the European Respiratory Society Clinical Research Collaboration on Antimicrobial Resistance in Lung Disease. Here we present an outline of the study protocol and pilot phase results.

**Methods** The registry is a prospective, international, multi-centre, observational study. The study design and process is shown in figure 1. Data will be captured across two-week periods biannually for five years. Primary endpoint: determine the prevalence of AMR in specific high-priority respiratory pathogens. Secondary endpoints: describe the geographical variation and susceptibility patterns of high-priority AMR pathogens, and determine any risk factors for AMR in chronic respiratory disease.

**Results** A pilot phase was recently completed. 11 centres spanning 8 countries and 4 continents participated, contributing data for 396 patients. The most common disease groups were bronchiectasis (109/396), chronic obstructive pulmonary disease (95/396) and cystic fibrosis (92/396). 208/396 (53%) patients had at least one positive bacterial sample and 33/396 (8%) had at least one positive fungal sample in the preceding 12 months. The most common bacterial pathogen was *Pseudomonas aeruginosa* (61/208 (29%)), followed by *Staphylococcus aureus* (46/208 (22%) and *Klebsiella pneumoniae* (32/208 (15%)). 41/208 (20%) grew more than one species. Resistance profiles were available for 277 bacterial isolates. 55/277 (20%) showed mono-resistance and 123/277 (44%) showed resistance to >1 antibiotic. The most common reported resistance was Ciprofloxacin.

**Conclusions** This pilot phase demonstrated the feasibility of prospective multi-centre data capture around AMR. The main registry will launch in November 2025 and is currently anticipated to involve >100 centres worldwide. The registry will address critical knowledge gaps in AMR in chronic respiratory disease through an international and collaborative approach. It has the potential to inform global strategies for antimicrobial stewardship, infection prevention, and improve our management of AMR in chronic respiratory diseases.




**Abstract P186 Figure 1** A schematic of the study design for data collection and analysis for the PRESIDE registry

## P187 DIVERSITY OF PSEUDOMONAS AERUGINOSA IN NON-CYSTIC FIBROSIS BRONCHIECTASIS: INSIGHTS FROM A NORFOLK PATIENT POPULATION

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**Background** Bronchiectasis is a chronic pulmonary condition characterised by abnormal, irreversible bronchial dilation, leading to persistent airway infection and inflammation. Pseudomonas aeruginosa colonisation is associated with frequent exacerbations and higher mortality. Previous sequencing studies have shown significant diversity between patients in P. aeruginosa sequence types.

**Methods** Adults with non-CF bronchiectasis and at least 1 positive P. aeruginosa sputum culture over 12 months were recruited between January and December 2024. Bacteria were cultured from sputum using differential agar and some incubated in 5% $CO_2$ to reflect the lung microenvironment. DNA from isolates was sequenced using Illumina NextSeq. A core SNP phylogeny was constructed to assess intra-patient strain diversity. Antibiotic minimum inhibitory concentration testing was performed on agar plates to determine the AMR profile, using EUCAST breakpoints. Existing wastewater-derived phages were screened for host range against clinical isolates.

**Results** 541 suspected P. aeruginosa isolates were cultured from 49 participants. Of the isolates tested thus far, 73% were confirmed as P. aeruginosa using whole genome sequencing. P. aeruginosa was still detected in some patients post-antibiotic treatment. Phylogenetic analysis revealed patient-specific clades, as well as notable cross-patient clustering. 69% of tested isolates were resistant to ciprofloxacin; one isolate was resistant to all frontline antibiotics used at the host institution. Notably, none of the 14 previously isolated phages lysed clinical strains, despite demonstrating activity against other clinical and reference strains.

**Conclusions** Clinical P. aeruginosa isolates from bronchiectasis patients in Norfolk display significant in vitro antibiotic resistance and patient-specific genetic diversity. These results are in keeping with or slightly higher than in previous studies which show greater diversity between patients than within individual patients. Previously isolated phages failed to lyse these strains, highlighting the need for a more targeted isolation approach to identify therapeutically viable phages for bronchiectasis patients.

## P188 WHAT DOES CARE FOR ADULTS WITH NON-TUBERCULOUS MYCOBACTERIA PULMONARY DISEASE (NTM-PD) LOOK LIKE? RESULTS FROM A UK-WIDE SURVEY

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10.1136/thorax-2025-BTSabstracts.369

**Background** Clinical outcomes for NTM-PD are often poor. The NTM Network UK Standards of Care were launched in

2024 to support consistent, high-quality practice including pharmacological and non-pharmacological treatment. We aimed to document NTM-PD management prior to this to identify opportunities to improve care.

**Methods** Centres were invited via the NTM Network UK to complete an online retrospective survey. At each site, anonymised clinical data were recorded from three consecutive adults (≥18) newly diagnosed with NTM-PD from 1 January 2022.

**Results** 48 centres participated (Northern England, n=12; Southern England, n=12; London, n=9; Wales, n=7; Midlands, n=6; Scotland, n=2) They reported having the following resources: physiotherapy for airway clearance (n=42/48, 88%), dietitian and pulmonary rehabilitation (each n=32, 67%), specialist nurse (n=30, 63%), and health psychology (n=9, 19%).

Respondents contributed data on 137 patients. 72 (53%) were female and 115 (84%) identified as White-British. 45 (34%) were aged 61–70. 62 (45%) had COPD and 55 (40%) bronchiectasis. Common radiological findings were nodular-bronchiectatic change (n=62, 45%), fibro-cavitary disease (n=40, 29%), and emphysema (n=39, 28%). *Mycobacterium avium complex* (MAC) was identified in 106 (77%), *M. kansasii*(n=14, 10%) and *M. abscessus* (n=9, 7%).

Eighty-one (59%) patients were discussed in an MDT. Treatment plans included involvement of specialist nurses (n=91, 66%), physiotherapists (n=52, 38%), dietitians (n=34, 25%), and pulmonary rehabilitation (n=16, 12%). Forty-two (31%) were advised on airway clearance using Active Cycle Breathing, 25 (18%) Positive Expiratory Pressure devices, 17 (12%) mucoactive drugs, and 12 (9%) hypertonic saline. Written resources were provided to 39 (28%) patients, 22 (16%) were directed to patient-focused websites, and 13 (10%) signposted to peer-support networks.

During a median follow-up from diagnosis of 21 (IQR 14–29) months, 124 (91%) started antibiotics for NTM-PD. Forty-five (33%) remain on follow-up and using antibiotics, 53 (39%) are not on antibiotics, 26 (19%) were discharged, and 12 (9%) died.

**Conclusion** This UK-wide assessment of NTM-PD management reveals significant variation in available services and care provided. Most patients started specific antimicrobials, though non-medical health care support and patient information provision were limited. We have identified important areas for re-audit against the 2024 Standards.

## P189 MYCOBACTERIUM CHIMAERA PREDOMINANCE AND HOST VULNERABILITY IN NON-CF BRONCHIECTASIS — A UK COHORT STUDY

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10.1136/thorax-2025-BTSabstracts.370

**Background** Non-tuberculous mycobacteria (NTM) have emerged as significant pathogens in non-cystic fibrosis (CF) bronchiectasis. While *Mycobacterium avium* and *M. intracellulare* are considered the dominant MAC species across Europe and North America, our UK-based cohort revealed a striking shift toward *M. chimaera* predominance. In addition, we observed an under-recognised burden of nutritional risk and NTM disease in patients without underlying lung conditions.

**Methods** We retrospectively reviewed all non-CF patients treated for NTM infection at a UK tertiary centre between January 2020 and December 2024. Data on demographics, NTM species, disease classification, BMI, comorbidities, immunosuppression, and lifestyle factors were collected and compared to published European and global cohort data.

**Results** Among 48 patients (67% male, mean age 66.3), *M. chimaera* was the leading species, accounting for 71% of isolates far exceeding the 10–20% typically reported in European MAC cohorts. Other species included *M. avium* (8%), *M. marinum* (6%), and several less commonly reported species such as *M. porcinum*, *M. fortuitum*, and *M. malmoense*.

COPD (44%) and bronchiectasis (23%) were the most common comorbidities, but notably, 17% of patients (n=8) had no underlying lung disease. Within this subgroup, 5 had extrapulmonary NTM (NTM-EP), 2 had pulmonary involvement (NTM-PD), and 1 had disseminated infection. Species in this group were diverse and included *M. chimaera* (2), *M. marinum* (2), *M. avium*, *M. porcinum*, *M. malmoense*, and *M. fortuitum*. Three were immunosuppressed and three had a BMI ≤21 highlighting the vulnerability of non-traditional hosts.

Nutritional risk was a common theme throughout the cohort. The mean BMI was 21.3 (range 11–32), and 33% of patients had a BMI below 20. Smoking (73%) and alcohol use (17%) were also prevalent. Most patients (85%) had NTM-PD, while 13% had NTM-EP, and 2% had disseminated infection. HIV prevalence was low (4%).

**Conclusions** This cohort highlights an evolving epidemiological trend in the UK, with *M. chimaera* emerging as the predominant MAC subspecies challenging current assumptions based on continental European data. Routine species-level identification, integrating nutritional assessments, and multi-disciplinary care could improve diagnostic precision and clinical outcomes in this growing patient group.

## P190 MANAGEMENT OF NON-TUBERCULOUS MYCOBACTERIAL PULMONARY DISEASE (NTM-PD): A EUROPEAN WIDE NTM-NET SURVEY

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10.1136/thorax-2025-BTSabstracts.371

**Introduction** NTM-PD is rare and complex. Guidelines focus on diagnosis and initial pharmacological management. There is limited guidance on drug monitoring, second-line management and access to advisory platforms. This survey aimed to explore current clinical practice across EU/EEA & UK and identify knowledge gaps to inform future guidelines.

**Methods** An online survey, including questions to determine diagnostic capability, medicines availability and treatment monitoring, was distributed via pan-European networks.

**Results** 34 centres across 20 EU/EEA & UK countries responded. The majority (79%) were from specialist NTM referral centres. Use of formal advisory platforms varied, with 53%(n=18) presenting NTM-PD cases at regional or national level. Of these, 50%(n=9) reported this being less frequently than monthly. All centres perform CT thorax in suspected cases of NTM-PD and proceed to bronchoalveolar lavage if initial sputum culture is negative. Induced sputum facilities were available in 71%(n=24) centres.

Drug-interaction monitoring relied on consultation with pharmacists (n=17,50%), national guidelines (n=16,47%) and online tools (n=9,26%). Use of injectable anti-microbials for the treatment of NTM-PD is rising. In the past two years, 82%(n=28) centres used injectables. The most frequently used were amikacin (100%), tigecycline (61%) and imipenem (50%). Second-line agents including nebulised amikacin, linezolid and clofazimine were used by 68%(n=23), 74%(n=25) and 76%(n=26) respondents, respectively. Five respondents reported that clofazimine was used in the initial treatment regimen for certain cases, such as in extensive disease or as first line for M. Abscessus. Ten (29%) centres prescribed Bedaquiline for NTM-PD. Treatment duration varied between 3–6 months (n=7,70%) and 6–12 months (n=3,30%). Indication cited was either refractory M. Abscessus or MACs.

Two thirds of respondents had patients who underwent surgery for NTM-PD in the past 5 years. Common indications included cavities, solitary lesions, haemoptysis, and failed treatment response. Follow-up of stable patients varied, 24%(n=8) did monthly reviews, while 68%(n=23) followed up every 2–4 months.

**Conclusions** There is significant variation in practice between countries within EU/EEA & UK. Use of injectable and second-line therapy is increasing with limited international/national guidance available. Ensuring access to evidence-based guidelines incorporating these anti-microbials and expert advisory groups is essential to ensure consistent, high-quality NTM-PD care.

## P191 INVESTIGATING THE RELATIONSHIP BETWEEN INTERLEUKIN-6 (IL6) AND HEALTH OUTCOMES ONE YEAR AFTER HOSPITAL DISCHARGE FOR COVID-19

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10.1136/thorax-2025-BTSabstracts.372

**Background** There is an urgent need to identify targets for therapeutic trials in patients with Long Covid. We previously described a positive association between IL-6 levels at five months and ongoing severe health impairments at one year after hospital discharge for COVID-19.[1] We therefore aimed to describe if an association persisted between IL-6 levels one year after hospital discharge for COVID-19 with health outcomes at the same timepoint.

**Methods** Adults recruited to the Post Hospitalisation Covid-19 (PHOSP-COVID) study attended a research visit one-year after discharge when the following patient reported outcomes were measured: health-related quality of life (HRQoL) using the EuroQol questionnaire (EQ5D-5L), fatigue questionnaire (FACIT), short physical performance battery (SPPB), total number of symptoms and patient-reported recovery (Yes/No). In a sub-group of participants, Olink analysis was performed with IL-6 as one of the measured proteins. We conducted an analysis to investigate the relationship between IL-6 and health outcomes using a generalized linear model (GLM) in RStudio version 4.5.0, adjusting for age, sex, body-mass index, smoking, respiratory support during acute admission, index of multiple deprivation (IMD) and number of co-morbidities as covariates. Beta coefficient (β) quantifies the change in dependent variable (IL-6) for one-unit increase in the independent variable (health outcomes).

**Findings** Data from 648 participants, who had Olink analysis performed at one year, was included: 394 (60%) men, mean (SD) age of 58.2 (12.0) years, 18.2% receiving invasive mechanical ventilation. IL-6 was associated with HRQoL ($\beta$ = -0.66, $p = 0.007$), fatigue ($\beta = 0.019$, $p < 0.001$), physical performance ($\beta$ = -0.126, $p < 0.001$) and number of symptoms ($\beta = 0.017$, $p < 0.006$) after adjustment for covariates.

**Conclusion** We report an association between IL-6 levels and a range of health outcomes at one-year after hospital discharge from COVID-19. Higher IL-6 levels at one-year were associated with lower quality of life, lower physical performance, and higher symptom count. A randomised controlled trial of anti-IL-6 in Long COVID is needed to understand if there is a causal pathway.

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## P192 PULMONARY LONG COVID IS ASSOCIATED WITH AIRWAY MICROBIAL DYSBIOSIS AND IMMUNE DYSREGULATION

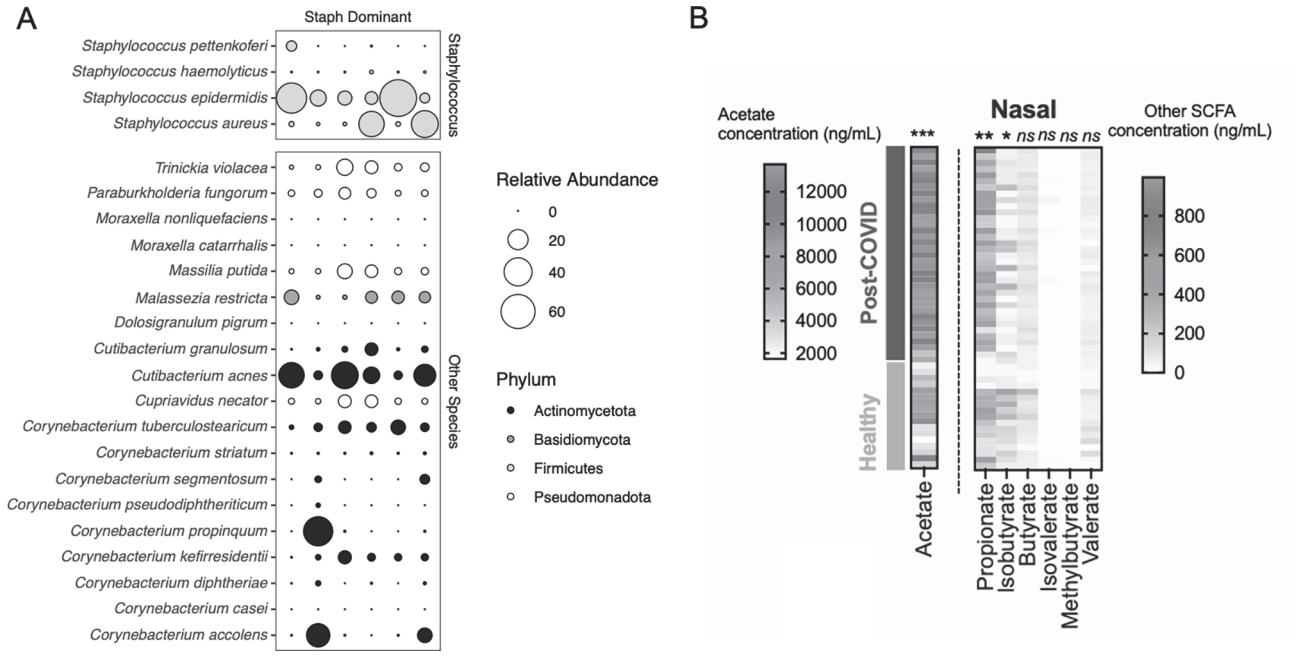
[1]M Kusumoto, [1]J McKenzie, [2]S Bamashmous, [3]H Chown, [4]CG Green, [1]MM Jackson, [1]S Laverty, [1]T Shahridan Faiez, [5]F Kamal, [1]A Reed, [1]X Xu, [1]P Kelleher, [1]PM George, [4]L Heaney, [3]T Abujamel, [1]A Shah, [1]A Singanayagam. *[1]Imperial College London, London, UK; [2]King Abudulaziz University, Jeddah, Saudi Arabia; [3]University of Glasgow, Glasgow, UK; [4]Loughborough University, Loughborough, UK; [5]Royal Berkshire Hospital, Reading, UK*

10.1136/thorax-2025-BTSabstracts.373

**Introduction** Persistent pulmonary symptoms following severe COVID-19 occurs in a subgroup of individuals, through unknown mechanisms. It has been postulated that persistent immune activation induced by microbial dysbiosis could play a potential role but data to support this theory are lacking. We hypothesised that microbial dysbiosis could be a driver of post-COVID sequelae.

**Methods** We evaluated upper airway microbiome (16S rRNA sequencing and shotgun metagenomics) and immune (transcriptomic) profiles in nasal samples taken at 3–6 months following hospitalised severe COVID-19 in the PROSAIC study. We studied associations between microbiota profiles, immune gene expression and respiratory symptom scores. We additionally quantified concentrations of the immunomodulatory metabolites short chain fatty acids (SCFAs) in nasal and plasma samples by GC-MS.

**Results** 16S sequencing revealed that nasal relative abundance of *Staphylococcus* genus was increased in post-COVID subjects (n=43) compared to healthy controls (n=19) ($P<0.001$) with a corresponding reduction in *Alloiococcus* ($P<0.05$). *Staphylococcus* relative abundance correlated with St George's Respiratory Questionnaire scores ($r=0.36$; $P<0.05$). Transcriptomic analysis of nasal brushing samples identified distinct inflammatory signatures in subjects with high (>50%) Staphylococcal abundance with significant activation of pro-inflammatory (NF-κB) and antiviral (type I interferon) pathways. Shotgun metagenomic analysis delineated that four Staphylococcal species were specifically increased in post-COVID subjects (*S. aureus, S. epidermidis, S. haemolyticus, and S. pettenkoferi*) (figure 1A). Given our prior findings that the microbiota-derived immunomodulatory metabolites SCFAs can augment pulmonary IFN signalling, we measured a range of SCFAs in these subjects and found that nasal concentrations of acetate ($P<0.001$), isobutyrate ($P<0.05$) and propionate ($P<0.01$)




**Abstract P192 Figure 1** **(A)** Metagenomic analysis of nasal microbiota in post-COVID subjects reveals Staphylococcus species expansion. Bubble plot displaying relative abundance of bacterial species, with bubble size representing relative abundance (%) and colours indicating phyla. Four Staphylococcus species (S. aureus, S. epidermidis, S. haemolyticus, and S. pettenkoferi) demonstrate increased abundance in post-COVID subjects with Staphylococcus-dominant microbiota. **(B)** Elevated nasal SCFAs in post-COVID versus healthy subjects. Heatmap comparing SCFA concentrations (ng/mL) in post-COVID versus healthy nasal samples ( *** P<0.001, ** P<0.01, *P<0.05, ns = not significant)

were all increased in post-COVID versus healthy subjects with similar increases in butyrate and propionate observed in plasma (figure 1B). Notably, Staphylococcal relative abundance correlated significantly with nasal acetate levels (r=0.44; *P*=0.0084) but not with plasma acetate concentrations, suggesting local respiratory tract, rather than systemic, production.

**Conclusion** Staphylococcal expansion and associated SCFA production are increased following severe COVID-19 and may drive persistent interferon activation and long-term symptoms. Our ongoing studies are investigating causal relationships between Staphylococcal commensals, SCFAs, and epithelial immunity using primary nasal epithelial cells.

# The logic of biologics

## P193 DEVELOPMENT OF A MULTI-MODAL MODEL TO PREDICT ASTHMA OUTCOMES USING DIGITAL MONITORING TOOLS

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10.1136/thorax-2025-BTSabstracts.374

**Introduction** Asthma is driven by inflammation and variance in lung function. While triggers including allergens worsen the condition, treatment with inhaled steroids (ICS) and beta-agonists dampen these responses. As treatment adherence is non-linear, utilizing point-in-time test results to measure inflammation and airflow obstruction, without the context of recent treatment use, makes it difficult to distinguish severe asthma from difficult to treat asthma. To address this, we developed a multi-modal model, incorporating serial lung function measures and treatment use to create a summary metric of asthma severity over time.

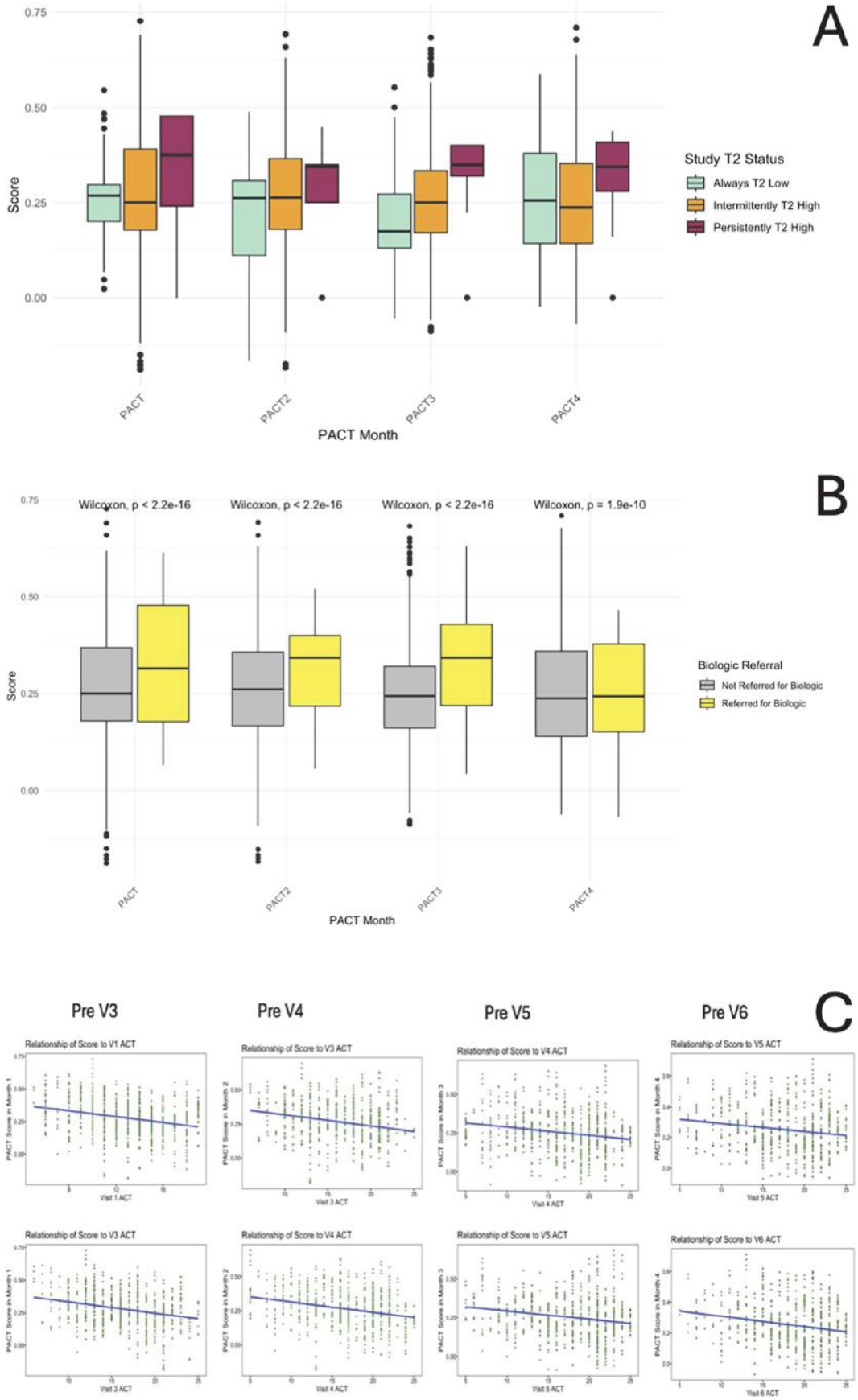
**Hypothesis** Digital monitoring of trends in lung function, in combination with treatment adherence, may provide a clinically useful diagnostic tool for asthma

**Methods** The model was developed with data from 200 patients with difficult to treat asthma, who, over an 8-month period performed twice-daily home peak flow (PEF) readings (ASMA-1 digital PEF) while both reliver and preventer bronchodilator and ICS treatment use was digitally-assessed (INCA device). A composite score for each month was created. Model performance was assessed through assessing between group differences in inflammation and requirement for biologic therapy.

**Results** The model, named the Peak flow Asthma Composite Test (PACT), demonstrates higher scores in those who remain persistently inflamed, compared to those who are only intermittently inflamed (figure 1A), with a significant positive correlation between PACT and both eosinophils and FeNO (p<0.01). The score also demonstrated that those who were referred for biologic also had statistically significantly higher scores at each time point (figure 1B). Meanwhile, a negative correlation was seen with each month's Asthma Control Test (ACT) (r = -0.26 to -0.18, all p <0.001) (figure 1C).

The model was subsequently refined, utilizing machine learning tools, and incorporating steroid exposure and T2 inflammation. Linear regression demonstrated that the refined model was significantly associated with total number of respiratory exacerbations (p<0.001).

**Conclusion** The model reliably synthesizes clinically relevant data into a single score that predicts need for biologic therapy, persistence of T2 inflammation, and patient reported




**Abstract P193 Figure 1** **A)** illustrates relatively higher scores in those who were persistently inflamed. **B)** demonstrates higher scores seen in those who ultimately required biologic referrals. **C)** demonstrates an inverse relationship between ACT and PACT score

outcomes. This tool has potential to streamline asthma diagnosis and monitoring by providing clinicians with a quantitative, longitudinal assessment of both disease presence and control.

## P194 IS T2 -GUIDED TREATMENT FEASIBLE IN ASTHMA EXACERBATIONS?

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10.1136/thorax-2025-BTSabstracts.375

**Introduction** Overuse of oral corticosteroids (OCS) is associated with long-term morbidity for patients with asthma. Recent data has shown those with little evidence of type-2 (T2) inflammation may not respond as well to OCS treatment compared with T2 high patients.[1]
**Aims** To measure outcomes of patients experiencing asthma exacerbations treated with a T2 biomarker guided strategy.
**Methods** Data were collected from patients attending a tertiary severe asthma 'rapid access' service. Patients contacted the service when they reported an increase in symptoms, were seen urgently, and underwent a comprehensive assessment with FeNO, viral swab, sputum culture, blood sampling for WBC (including eosinophil count [BEC]), CRP, and either peak flow measurement or simple spirometry. A clinical decision was then made whether or not to treat the patient with OCS. Patients were followed-up 30 days after the visit and need for further treatment was recorded.
**Results** In the period between June 2024 – May 2025, 77 patients were assessed with a mean (SD) age of 55.0 (19.2]), 76% (n=58) were female and 70% (n=54) were biologic naïve. Treating clinicians prescribed OCS for 40% (n=31) of patients. BEC were significantly lower in those not prescribed OCS (0.13 vs 0.37 x$10^9$/L, p<0.01), as were FeNO levels (35 vs 60 ppb, p=0.027). Those who received OCS had a significantly lower PEFR (percentage of best: 76.3 vs 87.8, p<0.01) and a lower CRP (5.4 vs 19.1, p=0.034). There was no difference in the proportion of patients who required further treatment at follow-up between those who received OCS and those who did not (n [%] 2 [6.5] vs 4 [8.7] respectively, p=0.87).
**Conclusions** Over 50% of patients presenting with increasing symptoms were managed without OCS and did not require a course of OCS or hospitalisation at 30-day follow-up. Those managed without OCS had significantly lower T2 biomarkers. A randomised clinical trial of T2 guided OCS is required to understand whether this strategy is non-inferior to 'blanket' OCS prescribing.

**Abstract P194 Table 1**

| Category | All | No OCS | OCS |
|---|---|---|---|
| Number – n (%) | 77 (100) | 46 (60) | 31 (40) |
| Age – mean (SD) | 55.0 (19.2) | 53.1 (17.6) | 58.9 (20.6) |
| Sex – n (%) | | | |
| - Male | 19 (24) | 12 (26) | 7 (23) |
| - Female | 58 (76) | 34 (74) | 24 (77) |
| Eos – mean (SD) | 0.22 (0.40) | 0.13 (0.15) | 0.37 (0.61) |
| Eos Categories – n (%) | | | |
| - <0.15 | 34 (44) | 22 (48) | 12(39) |
| - 0.15-0.3 | 13 (17) | 10 (22) | 3 (10) |
| - >0.3 | 15 (19) | 3 (7) | 12 (39) |
| - Missing | 15 (19) | 7 (15) | 4 (12) |
| FeNO – mean (SD) | 44.3 (46.2) | 35.0 (33.0) | 60.0 (60.3) |
| FeNO Categories | | | |
| - <20 | 24 (31) | 16 (35) | 8 (26) |
| - 20-50 | 18(23) | 11 (24) | 7 (23) |
| - >50 | 21 (27) | 10 (12) | 11 (35) |
| - missing | 14 (18) | 9 (19) | 5 (16) |
| CRP – mean (SD) | 14 (26.6) | 19.1 (32.5) | 5.4 (6.9) |

### REFERENCE

1. Celis-Preciado C, *et al. European Respiratory Journal* 2025 May.

## P195 PHENOTYPIC CHARACTERISTICS ASSOCIATED WITH FREQUENT ASTHMA EXACERBATIONS ON TEZEPELUMAB

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10.1136/thorax-2025-BTSabstracts.376

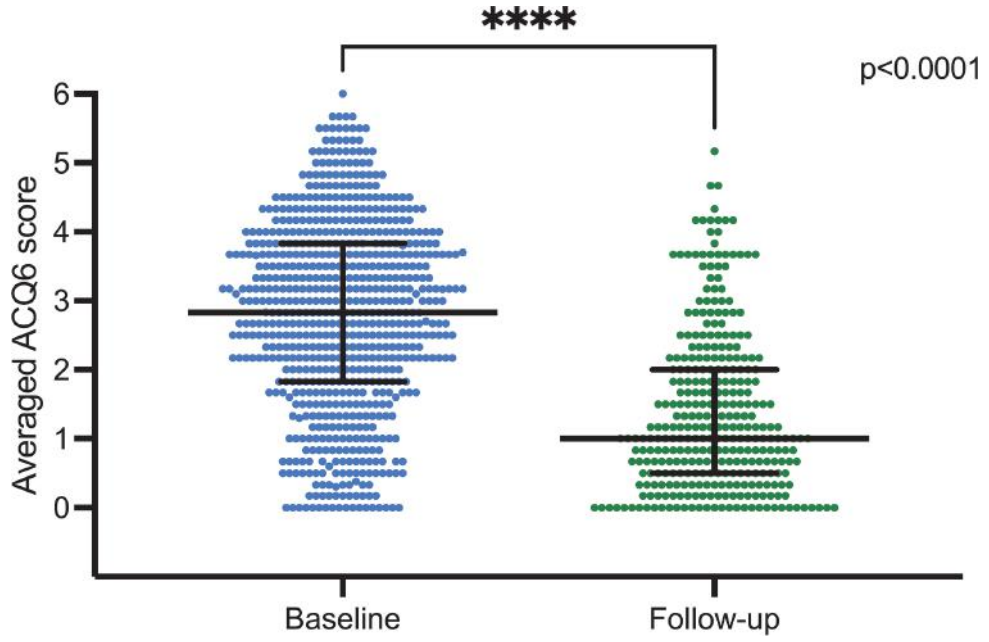
**Introduction** Tezepelumab is an anti-TSLP biologic therapy with broad anti-inflammatory effects. Clinical trials have been demonstrated its ability to significantly reduce asthma exacerbations (AE) and allow clinical remission (CR) in some patients with uncontrolled, severe asthma (SA). However, a proportion of patients do not adequately respond and continue to exacerbate on tezepelumab. The clinical characteristics of these 'non-responders' is poorly understood.
**Methods** The clinical response of individuals with SA established on treatment with tezepelumab at our regional severe asthma centre was analysed. The baseline characteristics of patients who achieved CR (defined as no AE, no maintenance OCS, ACQ6<1.5 and stable FEV1) was compared with non-responders (NR) defined as ≥2 exacerbations in the first year of tezepelumab.
**Results** Out of a total cohort of 218 patients, 103 patients had either achieved CR (n=54, 25%) or fulfilled criteria for NR (n=49, 22%) and were compared. Baseline clinical characteristics associated with NR vs CR included lower FeNO (35ppb vs 66ppb, p<0.001), higher baseline ACQ6 (2.90 vs 1.95, p<0.001), maintenance OCS requirement (p=0.03), higher baseline AE rate (3.6 vs 2.3, p=0.009), child-onset asthma (p=0.01) and the absence of comorbid nasal polyposis (p=0.006). The reduction in FeNO from baseline to treatment was significantly greater in patients achieving CR compared to NR, with median (IQR) changes of -43 ppb (-70 to -13) versus -4 ppb (-25 to 3), respectively ($p$<0.0001).
**Conclusion** Over 20% of individuals with SA continue to exacerbate frequently despite tezepelumab. Reduced efficacy in patients with lower baseline FeNO, child-onset asthma and the absence of nasal polyposis all point to an increased likelihood of non-T2 related exacerbation events in these patients.

## P196 THE IMPACT OF BIOLOGICS IN SEVERE ASTHMA: REAL-WORLD REGISTRY DATA FROM THE EAST OF ENGLAND

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10.1136/thorax-2025-BTSabstracts.377

**Background** Biologic therapies have shown great promise in asthma clinical trials over the past decade. It is important to




**Abstract P196 Figure 1**

understand their impact on patients' quality of life, lung function, steroid usage and healthcare utilisation when deployed at scale in a real-world tertiary service.

**Methods** A retrospective anonymised observational cohort study was undertaken using registry data from the East of England Severe Asthma Network from the past decade comparing patients before and after starting an asthma biologic drug. All consecutive adult patients commenced on a biologic (2013-present) were included if both a baseline (pre-treatment) and at least one follow-up (on treatment) clinic visit had occurred. Where more than one follow-up dataset was available, the most recent was used. Clinical data included steroid usage, hospital attendances, pulmonary function tests and asthma-specific patient-reported outcome data - Asthma Control Questionnaire (ACQ6).

**Results** Data was included from 806 adults: 59.8% female, 81.4% White British ethnicity, mean baseline BMI 31.3 kg/m$^2$ (SD7.5). There was a mean of 3.3 years between baseline and follow-up data. Patients were prescribed: benralizumab n=442, dupilumab n=69, mepolizumab n=146, omalizumab n=51, tezepelumab n=69, not recorded n=29. 49.3% of patients were taking maintenance oral corticosteroids at baseline compared to 25.5% at follow-up (Fisher's test; p<0.0001). There was a median of five exacerbations requiring a rescue course of oral corticosteroids in the 12-months preceding baseline compared to a median of zero annual exacerbations at follow-up (Mann-Whitney test; p<0.0001). 29.3% of patients had at least one acute hospital attendance for asthma in the 12-months prior to baseline compared to 4.4% at follow-up (Fisher's test; p<0.0001). The median averaged ACQ6 score was 2.8 (IQR 1.8–3.8) at baseline compared to 1.0 (IQR 0.5–2.0) at follow-up (Mann-Whitney test; p<0.0001) (figure 1). The median FEV1 did not differ (2.24L vs 2.27L, p=0.14).

**Conclusions** Following the initiation of asthma biologic therapies, acute hospital attendances, health-related quality of life and both acute and chronic corticosteroid usage improved. Whilst acknowledging both the limitations of this unblinded, non-controlled observational study but also the strengths of real-world clinical service data, the positive impact of asthma biologics on multiple patient-centred clinical endpoints is profound.

## P197 DUPILUMAB CAUSES IMPROVEMENTS IN SPIROMETRY RATIOS IN PATIENTS WITH MODERATE-TO-SEVERE ASTHMA, WHICH CORRELATE WITH CHANGES IN FRACTIONAL EXHALED NITRIC OXIDE AND ASTHMA CONTROL

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**Introduction and Objectives** Spirometry-derived ratios can be used to distinguish restrictive and obstructive lung disease and guide treatment decisions in patients with asthma. Knowing their relationship with type 2 biomarkers and symptom control could help assess responses to biologic treatments. Dupilumab, a fully human monoclonal antibody that blocks signaling of interleukin (IL)-4 and IL-13, improves lung function in patients with type 2, moderate-to-severe asthma. Here, we assess the effect of dupilumab on large and small airway dysfunction using spirometry-derived ratios and whether changes correlate with changes in fractional exhaled nitric oxide (FeNO) and symptom control in patients with asthma.

**Methods** In the phase 4 VESTIGE trial (NCT04400318), adults (aged 21–70 years) with type 2 (eosinophils ≥300 cells/μL and FeNO ≥25 ppb), uncontrolled, moderate-to-severe asthma were randomized to receive add-on dupilumab 300 mg (n=72) or placebo (n=37) q2w for 24 weeks. Using a mixed model for repeated measures, we evaluated changes from baseline at Weeks 4, 12, and 24 in four spirometry ratios:

**Abstract P197 Table 1** Change from baseline in spirometry-derived ratios over time in patients with type 2, moderate-to-severe asthma from VESTIGE

| | | Placebo N=37 | Dupilumab N=72 |
|---|---|---|---|
| **$FEV_1$/FVC, %** | Mean (SD) value at baseline | n=37<br>61.8 (13.9) | n=72<br>60.7 (11.5) |
| | LS mean difference* (95% CI), *P* value | | |
| | At Week 4 | n=37 | n=70<br>5.6 (2.4, 8.7), 0.0006 |
| | At Week 12 | n=36 | n=69<br>5.0 (1.9, 8.0), 0.0018 |
| | At Week 24 | n=33 | n=70<br>6.8 (3.4, 10.2), 0.0001 |
| **pp$FEV_1$/ppFVC, %** | Mean (SD) value at baseline | n=37<br>77.8 (16.8) | 76.7 (14.6) |
| | LS mean difference* (95% CI), *P* value | | |
| | At Week 4 | n=37 | n=70<br>7.0 (3.1, 10.9), 0.0005 |
| | At Week 12 | n=36 | n=69<br>6.2 (2.4, 10.1), 0.0019 |
| | At Week 24 | n=33 | n=70<br>8.5 (4.2, 12.7), 0.0001 |
| **$FEF_{25–75\%}$/FVC, %** | Mean (SD) value at baseline | n=37<br>37.8 (25.9) | 34.0 (24.0) |
| | LS mean difference* (95% CI), *P* value | | |
| | At Week 4 | n=37 | n=70<br>10.1 (3.4, 16.7), 0.0032 |
| | At Week 12 | n=36 | n=69<br>9.8 (3.6, 16.0), 0.0021 |
| | At Week 24 | n=33 | n=70<br>12.2 (5.2, 19.1), 0.0008 |
| **pp$FEF_{25–75\%}$/ ppFVC, %** | Mean (SD) value at baseline | n=37<br>49.5 (31.7) | 46.2 (31.7) |
| | LS mean difference* (95% CI), *P* value | | |
| | At Week 4 | n=37 | n=70<br>13.1 (4.7, 21.5), 0.0026 |
| | At Week 12 | n=36 | n=69<br>12.8 (4.6, 21.0), 0.0025 |
| | At Week 24 | n=33 | n=70<br>15.6 (6.3, 25.0), 0.0013 |

*LS mean difference in change from baseline for dupilumab vs placebo in percentage points. $FEF_{25–75\%}$, forced expiratory flow at 25% to 75% of pulmonary volume; $FEV_1$, forced expiratory volume in 1 second; FVC, forced vital capacity; LS, least squares; pp, percent predicted; q2w, every 2 weeks; SD, standard deviation.

absolute and percent predicted (pp) forced expiratory volume in 1 second ($FEV_1$)/forced vital capacity (FVC) and forced mid-expiratory flow at 25% to 75% of pulmonary flow ($FEF_{25–75\%}$)/FVC, which assess large and small airway dysfunction, respectively. Using Pearson's correlation coefficient (r), we assessed relationships between changes in each ratio at Week 24 and changes in FeNO level and 5-item Asthma Control Questionnaire (ACQ-5) score at Week 24.

**Results** Mean baseline values for each spirometry-derived ratio were comparable for dupilumab and placebo (table 1). Dupilumab vs placebo improved all ratios by Week 4 and through Week 24 (table 1). With dupilumab, at Week 24, change in all ratios inversely correlated with change in FeNO ($FEV_1$/FVC: r=−0.31, $P<0.01$; pp$FEV_1$/ppFVC: r=−0.31, $P<0.01$; $FEF_{25–75\%}$/FVC: r=−0.32, $P<0.01$; pp$FEF_{25–75\%}$/ppFVC: r=−0.33, $P<0.01$) and change in ACQ-5 scores ($FEV_1$/FVC: r=−0.27, $P<0.05$; pp$FEV_1$/ppFVC: r=−0.28, $P<0.05$; $FEF_{25–75\%}$/FVC: r=−0.24, $P<0.05$; pp$FEF_{25–75\%}$/ppFVC: r=−0.25, $P<0.05$). No significant correlations were observed in patients receiving placebo.

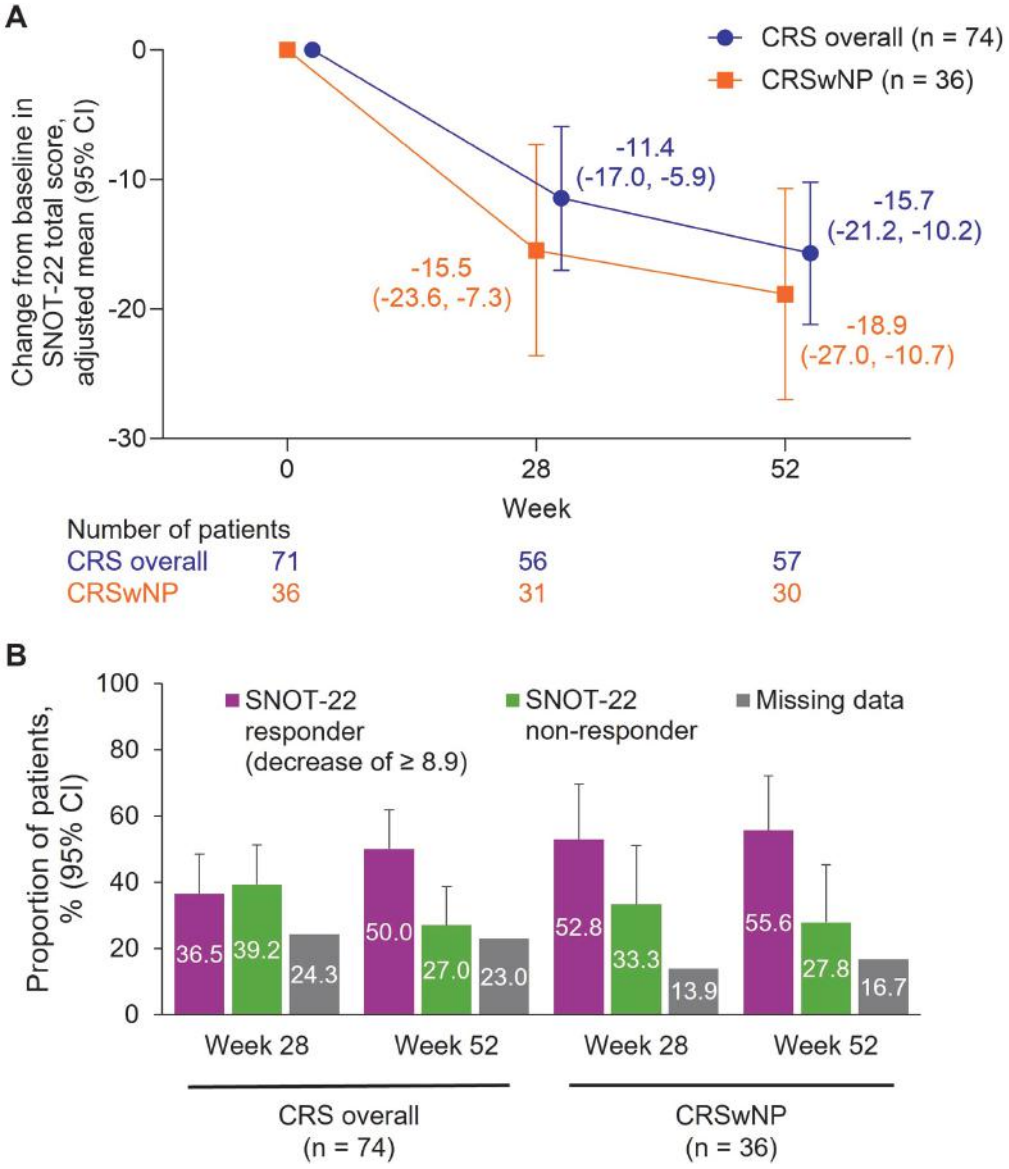
**Conclusions** In patients with type 2, moderate-to-severe asthma, dupilumab significantly increased spirometry-derived ratios, indicating improved large and small airway function. Increases in these ratios correlated with reductions in FeNO and improvements in asthma control.

P198 **TEZEPELUMAB REDUCES OCS USE AND IMPROVES SINO-NASAL SYMPTOMS IN OCS-DEPENDENT PATIENTS WITH SEVERE ASTHMA AND COMORBID CHRONIC RHINOSINUSITIS (OVERALL AND WITH NASAL POLYPS): RESULTS FROM THE PHASE 3B WAYFINDER STUDY**

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**Introduction and Objectives** Chronic rhinosinusitis (CRS), with nasal polyps (CRSwNP) or without NP (CRSsNP), is commonly associated with severe asthma. In the phase 3b WAYFINDER study of tezepelumab in oral corticosteroid (OCS)-dependent patients with severe asthma (NCT05274815), most patients achieved clinically meaningful reductions in maintenance OCS (mOCS) dose to ≤ 5 mg/day, or discontinued OCS, while maintaining asthma control. This *post hoc* analysis evaluated OCS reduction and sino-nasal symptoms in WAYFINDER participants with comorbid CRS, both overall and with NP only.




**Abstract P198 Figure 1**

**Methods** WAYFINDER was an open-label, single-arm, OCS-sparing study in adults with severe asthma receiving mOCS (prednisone/prednisolone 5–40 mg/day or equivalent) for ≥ 3 months before study entry. Patients with CRS (CRSwNP or CRSsNP) or CRSwNP only, received tezepelumab 210 mg subcutaneously every 4 weeks for up to 52 weeks. After a 4-week induction phase on a stable OCS dose, patients entered a 48-week OCS reduction and maintenance phase. The co-primary endpoints, assessed at weeks 28 and 52, were the proportions of patients who 1) reduced their daily prescribed mOCS dose to ≤ 5 mg/day without loss of asthma control; and 2) discontinued OCS without loss of asthma control. For this analysis, the co-primary endpoints, change from baseline in Sino-Nasal Outcome Test (SNOT)-22 total score, and SNOT-22 response (total score decrease from baseline of ≥ 8.9 [MCID]) were assessed at weeks 28 and 52.

**Results** Of 298 patients who received tezepelumab in WAYFINDER, 74 (24.8%) had CRS and 36 (12.1%) had CRSwNP. By week 52, 87.8% and 91.7% of patients in the CRS overall and CRSwNP groups, respectively, had an mOCS dose of ≤ 5 mg/day, while 45.9% and 50.0%, respectively, had discontinued OCS. Mean SNOT-22 total score improved by 15.7 and 18.9 in the CRS overall and CRSwNP groups, respectively, at week 52, and 50.0% and 55.6% of patients, respectively, were classified as SNOT-22 responders (figure 1).

**Conclusions** Tezepelumab enabled patients with severe asthma and CRS (with or without NP) to reduce or discontinue their mOCS use, while improving sino-nasal symptoms. These data contribute to the growing evidence of the efficacy of tezepelumab in patients with CRS, including CRSwNP.

P199

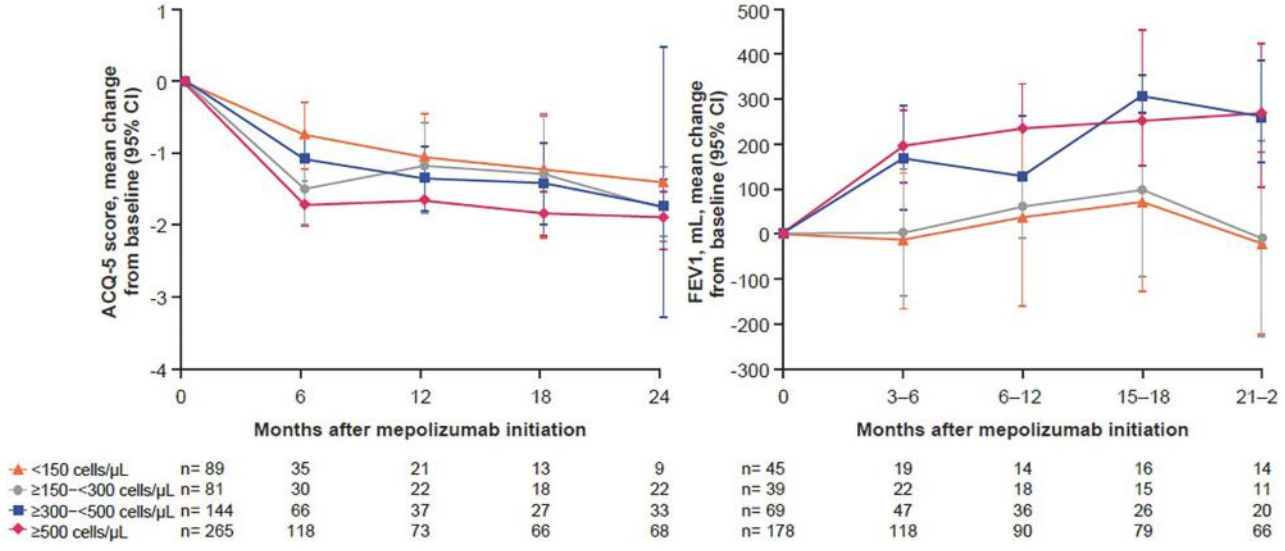
## MEPOLIZUMAB IMPROVES AND SUSTAINS ASTHMA CONTROL AND LUNG FUNCTION IN PATIENTS WITH SEVERE ASTHMA INDEPENDENT OF BASELINE BLOOD EOSINOPHIL COUNT IN THE REAL WORLD: RESULTS FROM REALITI-A AT 2 YEARS

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10.1136/thorax-2025-BTSabstracts.380

**Introduction and Objectives** Preliminary 1-year analyses from the real-world REALITI-A study demonstrated mepolizumab improvements in asthma control and lung function, a key outcome measurement, with mepolizumab treatment across blood eosinophil count (BEC) levels, in patients with severe asthma. However, additional real-world evidence on the sustained effect of mepolizumab across BEC ranges is required.

**Methods** REALITI-A was a 2-year, international, observational, prospective, single-arm study enrolling patients (≥18 years) with severe asthma, newly prescribed mepolizumab (100 mg subcutaneously). Changes from baseline in asthma control (Asthma Control Questionnaire-5 [ACQ-5] scores, minimal clinically important difference: 0.5) and lung function (forced expiratory volume in 1 second [$FEV_1$]) were assessed for baseline BEC subgroups (<150, ≥150–<300, ≥300–<500, ≥500 cells/μL) at 1-year and 2-years post-mepolizumab exposure.




ACQ-5, Asthma Control Questionnaire-5; BEC, blood eosinophil count; CI, confidence interval; $FEV_1$, forced expiratory volume in 1 second.

**Abstract P199 Figure 1** Asthma control improves and is sustained for 2 years for all BEC subgroups (left panel) and lung function improves and is sustained for 2 years for BEC ≥300 cells/uL subgroups (right panel). Error bars represent 95% CI

**Results** 822 treated patients were included. ACQ-5 scores decreased from baseline to 2-years post-mepolizumab exposure across all BEC subgroups (figure 1; mean change from baseline [95% CI]: <150 cells/μL, -1.40 [-3.27, 0.47]; ≥150–<300 cells/μL, -1.76 [-2.34, -1.19]; ≥300–<500 cells/μL, -1.74 [-2.15, -1.33]; ≥500 cells/μL, -1.88 [-2.23, -1.53]). Improvements in ACQ-5 were sustained from 1-year post-exposure reductions (mean change from baseline at 1-year [95% CI]: -1.06 [-1.68, -0.44]; -1.19 [-1.81, -0.57]; -1.37 [-1.84, -0.90]; -1.65 [-2.01, -1.28], respectively). Lung function ($FEV_1$) improved from baseline to Month 21–24 post-mepolizumab exposure for the BEC ≥300 cells/μL subgroups (figure 1; mean change from baseline [95% CI]: ≥300–<500 cells/μL, 261.5 [111.1, 411.9]) and ≥500 cells/μL, 275.6 [160.5, 390.6]), sustained from changes in $FEV_1$ at Month 9–12 post-exposure (mean change from baseline [95% CI]: 130.1 [-3.4, -263.7] and 239.4 [141.7, 337.2], respectively). Improvements in lung function were less consistent in the BEC groups <300 cells/μL.

**Conclusions** This analysis confirms treatment with mepolizumab in a real-life setting improves asthma control independent of baseline BEC, while lung function shows greater improvement with increased BEC; improvements observed at 1 year are maintained at 2 years.

## P200 REAL WORLD EXPERIENCE; AN INTERIM ANALYSIS OF EFFECTIVENESS OF TEZEPELUMAB IN PATIENTS WITH SEVERE UNCONTROLLED ASTHMA ACROSS THE UK

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10.1136/thorax-2025-BTSabstracts.381

**Introduction and Objectives** Tezepelumab is indicated in severe uncontrolled asthma (SuA) patients aged ≥12 years

**Abstract P200 Table 1** Biologics prior to index and key outcomes

| | | Overall (N=282) | Biologic-Naïve (N=104) | Biologic-experienced (N=175) |
|---|---|---|---|---|
| Clinical characteristics during the baseline period* | | | | |
| Median (IQR) highest FeNO result (ppb | | N=218<br>31.0 (17.0, 70.0) | N=86<br>28.5 (17.0, 60.0) | N=132<br>39.0 (17.5, 91.0) |
| Median (IQR) highest EOS count (cells/μL) | | N=263<br>200.0 (70.0, 400.0) | N=101<br>200.0 (100.0, 370.0) | N=161<br>150.0 (10.0, 470.0) |
| Biologics prior to index | | N=279 | N=104 | N=175 |
| Median (IQR) biologics (ever) pre-index | | 1.0 (0.0–2.0) | 0 | 2.0 (1.0–3.0) |
| No. of biologics prior to index | 0 | 104 (37.3%) | 104 (100.0%) | - |
| | 1 | 77 (27.6%) | - | 77 (44.0%) |
| | 2 | 46 (16.5%) | - | 46 (26.3%) |
| | ≥3 | 52 (18.6%) | - | 52 (29.7%) |
| Outcomes at 52 weeks post-index | | | | |
| AAER (95% CI) | | | | |
| Baseline* | | N=270<br>4.3 (4.0, 4.7) | N=100<br>4.1 (3.7, 4.6) | N=169<br>4.4 (4.0, 4.9) |
| 52 weeks | | N=274<br>2.1 (1.8, 2.5) | N=103<br>1.5 (1.1, 1.9) | N=170<br>2.6 (2.1, 3.1) |
| Rate ratio | | N=264<br>0.5 (0.4, 0.6)<br>p<0.0001 | N=99<br>0.4 (0.3, 0.5)<br>p<0.0001 | N=164<br>0.6 (0.5, 0.7)<br>p<0.0001 |
| Relative reduction (%)** | | N=264<br>49.0% | N=99<br>63.0% | N=164<br>41.0% |
| mOCS reduction | | | | |
| Proportion on mOCS at baseline | | N=274<br>111 (40.5%) | N=100<br>33 (33.0%) | N=173<br>78 (45.1%) |
| Median (IQR) mOCS dose at baseline | | N=111<br>10.0 (7.5, 15.0) | N=33<br>10.0 (5.0, 10.0) | N=78<br>10.0 (10.0, 17.5) |
| ≥50% reduction vs baseline*** | | N=109<br>52 (47.7%) | N=31<br>15 (48.4%) | N=78<br>37 (47.5%) |
| Mean (SD) ACQ-6 | | | | |
| Baseline | | N=211<br>3.3 (1.2) | N=79<br>3.2 (1.1) | N=132<br>3.3 (1.2) |
| 52 weeks | | N=107<br>1.9 (1.4) | N=46<br>1.4 (1.2) | N=61<br>2.2 (1.4) |
| % achieving MCID improvement | | N=95<br>73.7% (N=70) | N=41<br>85.4% (N=35) | N=54<br>64.8% (N=35) |

Abbreviations: FeNO; fractional exhaled nitric oxide, EOS; eosinophil, IQR; interquartile range, AAER; annualised asthma exacerbation rate, mOCS; maintenance oral corticosteroids, SD; standard deviation, ACQ-6, asthma control questionnaire-6, MCID; minimally important clinical difference.
Where N for analyses do not add to the overall sample, this is due to missing data.
*Baseline is defined as the 52 weeks prior to initiation of tezepelumab.
**Calculated as 1-RR
***Denominator is patients on mOCS at baseline (dose of ≥1 mg and reason for mOCS prescription was for asthma control, or dose of ≥5mg and the reason was missing)

inadequately controlled despite high-dose inhaled corticosteroids plus maintenance treatment. Clinical trials demonstrated favourable efficacy of tezepelumab; real-world data are needed to explore effectiveness. This study evaluates characteristics, treatment patterns and outcomes of adult SuA patients who received tezepelumab in the patient access programme and post-reimbursement populations.

**Methods** A retrospective, observational, chart review is ongoing in 11 SuA UK centres. SuA patients with an index date (tezepelumab first dose) between 01/01/2023 and 31/03/2024 were included. Data were extracted from medical records. This interim analysis (N=282) describes characteristics and outcomes during baseline (52 weeks pre-index) and follow-up (52 weeks post-index), stratified by prior biologic status, biologic-naïve [0 prior biologics, N=104] or biologic-experienced [≥1 prior biologic, N=175]).

**Results** At index, mean (SD) age was 51.0 (14.5) years, 66.0% were female, 98.2% had ≥1 comorbidity, 43.4% (N=89) had positive atopic status. Overall, 55.5% (146/263) had a blood eosinophil count (cells/μL) during baseline of ≥150.0; this was 64.4% (65/101) in biologic-naïve and 50.0% (81/161) in the biologic-experienced. 62.7% and 18.6% had ≥1 or ≥3 prior biologics, respectively.

Key outcomes are summarised in table 1. Median (IQR) forced expiratory volume in 1 second ($FEV_1$) post-bronchodilator (ml) increased from 1965.0 (1360.0, 2560.0) to 2220.0 (1530.0, 2890.0) over follow-up (absolute change of 125.0 [-20.0, 480.0]). Annualised asthma exacerbation rate (AAER) relative reduction at 52 weeks vs baseline were 49.0% overall; 63.0% and 41.0% for biologic-naïve and biologic-experienced patients, respectively. In patients on maintenance oral corticosteroids (mOCS) at baseline (N=118), 43.1% reduced dose by ≥50% by 52 weeks. Overall, 73.7% had a minimally important clinical difference (MCID) improvement in Asthma Control Questionnaire-6 (ACQ-6) at 52 weeks.

**Conclusions** Interim analyses demonstrate this cohort of SuA patients receiving tezepelumab had a high burden of mOCS use and comorbidities, with approximately two-thirds failing on a previous biologic, and almost a fifth failing on ≥3 biologics. Patients had marked improvements in all clinical outcomes, especially those receiving tezepelumab as their first biologic. The study is ongoing, final analyses will include approximately 350 patients.

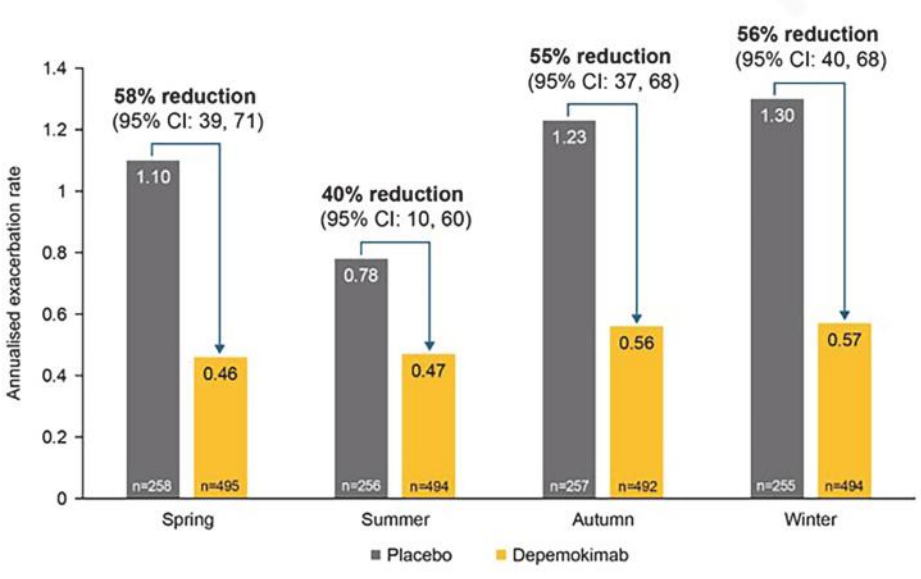
Sponsored and funded by AstraZeneca

## P201 TWICE-YEARLY DEPEMOKIMAB EFFICACY IS SUSTAINED ACROSS SEASONS IN PATIENTS WITH ASTHMA: ANALYSES OF POOLED PHASE III SWIFT-1/2 STUDIES

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**Introduction and Objectives** Depemokimab is the first ultra-long-acting biologic with enhanced interleukin-5 binding affinity, high potency and extended half-life, enabling twice-yearly dosing in patients with asthma. In the Phase III SWIFT-1/2 studies, depemokimab significantly reduced exacerbations with sustained inhibition of type 2 inflammation assessed by blood eosinophil count (BEC) observed in patients with type 2 asthma. Seasonal changes in common triggers for asthma exacerbations, including respiratory viruses, are well described; as such, this analysis of data from the SWIFT-1/2 studies aimed




Analysis performed using a generalised linear model assuming a negative binomial distribution and covariates of treatment group, baseline ICS dose (medium or high), exacerbation history (2, 3, ≥4), study (SWIFT-1 or SWIFT-2), and baseline pre-bronchodilator percent predicted $FEV_1$. Separate negative binomial models were fitted for each season.
CI, confidence interval; $FEV_1$, forced expiratory volume in 1 second; ICS, inhaled corticosteroid.

**Abstract P201 Figure 1** Annualised exacerbation rate reductions in patients receiving depemokimab versus placebo were consistent across spring, autumn and winter, with a lower magnitude of reduction in summer due to reduced exacerbation rates in the placebo group

to explore whether depemokimab efficacy in reducing exacerbations versus placebo is sustained across all seasons.

**Method** In SWIFT-1/2, patients with asthma who had ≥2 exacerbations in the past year and BEC ≥150 cells/μL (screening) or ≥300 cells/μL (past year) were randomised 2:1 to receive depemokimab 100 mg subcutaneously or placebo once every 26 weeks, plus standard of care. Analyses of pooled data from the SWIFT-1/2 studies were conducted to investigate annualised exacerbation rates (total number of exacerbations divided by total number of subject-years) by season (spring/summer/autumn/winter).

**Results** Of 762 patients included in the SWIFT-1/2 studies, 260 received placebo and 502 received depemokimab. Depemokimab was associated with reduced exacerbation rates versus placebo across all four seasons; the greatest benefit observed in spring, autumn and winter, with a 58% reduction in exacerbations in spring (rate ratio: 0.42 [95% confidence interval {CI}: 0.29, 0.61]), a 40% reduction in summer (0.60 [0.40, 0.90]), a 55% reduction in autumn (0.45 [0.32, 0.63]) and a 56% reduction in winter (0.44 [0.32, 0.60]) (figure 1). The annualised exacerbation rates (95% CI) in the depemokimab arm remained broadly consistent across all four seasons (spring: 0.46 [0.35, 0.61]; summer: 0.47 [0.36, 0.62]; autumn: 0.56 [0.44, 0.71]; winter: 0.57 [0.45, 0.72]; however, in the placebo arm exacerbation rates were, as expected, numerically lower in summer (spring: 1.10 [0.84, 1.43]; summer: 0.78 [0.58, 1.07]; autumn: 1.23 [0.97, 1.56]; winter: 1.30 [1.03, 1.63]) (figure 1).

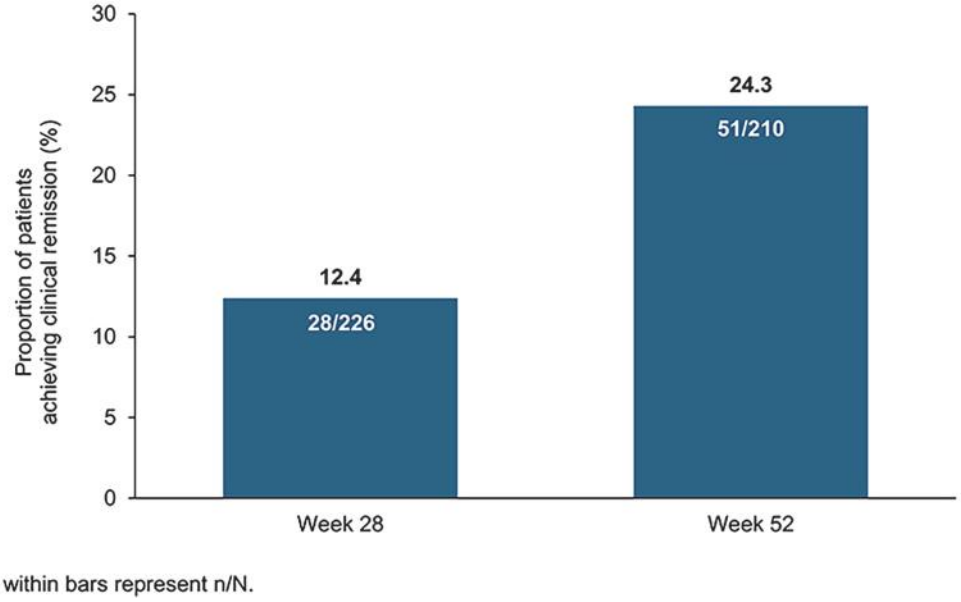
**Conclusion** Overall, twice-yearly depemokimab demonstrated a sustained clinical benefit for patients with type 2 asthma across all four seasons, regardless of additional exacerbation triggers over the autumn and winter months.

## P202 ON-TREATMENT CLINICAL REMISSION WITH TEZEPELUMAB IN OCS-DEPENDENT PATIENTS WITH SEVERE ASTHMA IN WAYFINDER

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**Introduction and Objectives** Treatment goals for severe asthma include disease remission. In the phase 3 NAVIGATOR study in severe uncontrolled asthma (NCT03347279), 28.5% of tezepelumab recipients achieved on-treatment clinical remission over weeks 0–52 (placebo, 21.9%). In the phase 3b WAYFINDER study in OCS-dependent severe asthma




**Abstract P202 Figure 1** Proportion of WAYFINDER participants who achieved on-treatment clinical remission at week 28 and week 52

(NCT05274815), OCS discontinuation without loss of asthma control was achieved by 32.2% of tezepelumab recipients at week 28 and 50.3% at week 52. This *post hoc* analysis assessed the proportion of WAYFINDER participants who achieved on-treatment clinical remission at weeks 28 and 52.

**Methods** WAYFINDER was an open-label, single-arm, OCS-sparing study in adults with severe asthma on maintenance OCS (mOCS). Patients received tezepelumab 210 mg subcutaneously every 4 weeks for 52 weeks. Clinical remission was defined as an Asthma Control Questionnaire-6 score <1.5, a post-bronchodilator forced expiratory volume in 1 second ≥95% of baseline and no use of mOCS at the end of the time period and no exacerbations over the time period.

**Results** Overall, 298 patients received tezepelumab. The proportion of patients achieving all clinical remission criteria was 12.4% (28/226) at week 28 and 24.3% (51/210) at week 52 (figure 1).

**Conclusions** About a quarter of OCS-dependent patients with severe asthma achieved clinical remission by 52 weeks of tezepelumab treatment.

## P203 UNIFORM CLINICAL BENEFIT OF BIOLOGIC THERAPY ACROSS A REGIONAL SEVERE ASTHMA NETWORK

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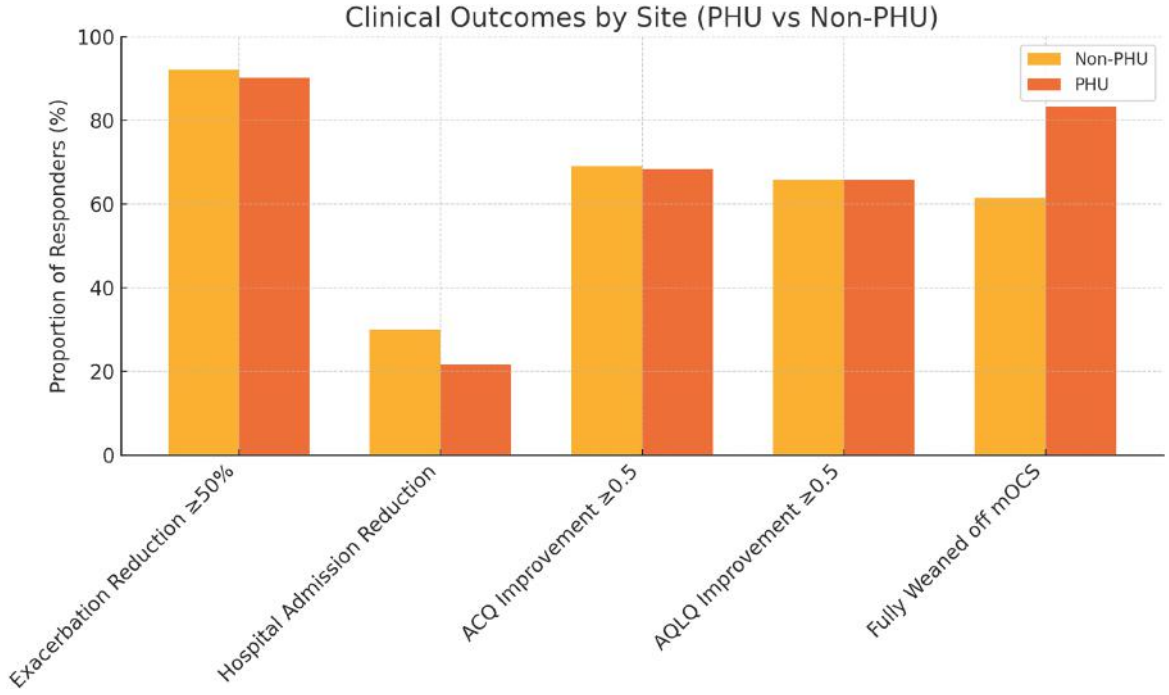
10.1136/thorax-2025-BTSabstracts.384

**Background** Biologic therapies have transformed the management of severe asthma, offering new hope to patients with uncontrolled disease. However, the real-world consistency of clinical benefit across network treatment centres remains poorly characterised. This study aimed to evaluate the magnitude of response to biologics and assess variation in outcomes across both hub and spoke sites within a regional asthma service.

**Methods** We conducted a retrospective cohort study of 101 patients with severe asthma who commenced biologic therapy between April 2022 and March 2023 across The Portsmouth Severe Asthma Service regional network comprising Portsmouth Hospitals University NHS Trust (PHU) and affiliated non-PHU sites. Paired data from baseline and 12 months were analysed for annual exacerbations, hospital admissions, asthma control (ACQ), asthma-related quality of life (AQLQ), and maintenance oral corticosteroid (mOCS) use. Clinically meaningful improvement was defined as: ≥50% reduction in exacerbations or admissions, ≥0.5-point reduction in ACQ, ≥0.5-point increase in AQLQ, or ≥50% reduction in mOCS dose. Statistical comparisons were made between sites using chi-square tests.

**Results** Among evaluable patients, 91.1% achieved ≥50% exacerbation reduction, 68.7% improved ACQ, 65.8% improved AQLQ, 25.7% reduced hospital admissions, and 72.2% of those on mOCS at baseline were completely weaned off by Year 1. Comparisons between PHU and non-PHU patients revealed no significant differences across any outcome. For example, exacerbation response rates were 90.2% vs 92.0% (PHU vs non-PHU), ACQ improvement 68.3% vs 69.0%, and AQLQ improvement 65.7% vs 65.8% respectively (all $p > 0.05$). Additionally, site-level analyses across individual hospitals demonstrated no statistically significant variation in responder rates, despite small patient numbers at some locations.

Exacerbation reduction, ACQ and AQLQ improvements, and mOCS weaning were consistently high across sites, demonstrating uniform biologic efficacy throughout the network.




**Abstract P203 Figure 1** Proportion of patients achieving key clinical outcomes at PHU and non-PHU sites. Exacerbation reduction, ACQ and AQLQ improvements, and mOCS weaning were consistently high across sites, demonstrating uniform biologic efficacy throughout the network

**Conclusion** Biologic therapies confer substantial clinical benefit in real-world severe asthma, with high rates of symptom control, steroid reduction, and quality of life improvement. Importantly, these outcomes were achieved consistently across the Portsmouth network of hospitals, including both specialist and district general settings. This reinforces the effectiveness and equity of biologic delivery across decentralised asthma care models, supporting their continued integration across regional and national severe asthma services.

## P204 SUPPRESSION OF FENO BUT NOT EOSINOPHILS WITH TEZEPELUMAB IS ASSOCIATED WITH IMPROVEMENTS IN SMALL AIRWAYS IN UNCONTROLLED SEVERE ASTHMA

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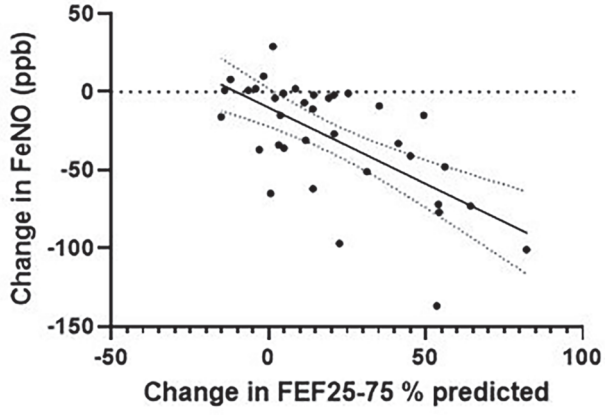
10.1136/thorax-2025-BTSabstracts.385

**Background** Tezepelumab inhibits TSLP thus acting on downstream IL 4,5 and 13, in turn reducing respective levels of IgE, eosinophils and FeNO (Niox Vero). There is a high degree of expression of IL13 but not IL5 in human small airways smooth muscle. Here we assessed the effects of tezepelumab on spirometry outcomes of large (FEV1) and small (FEF25–75) airways in relation to type 2 biomarkers in patients with uncontrolled severe asthma.

**Methods** We reviewed our severe asthma MDT identifying 38 patients who were commenced on tezepelumab. Their data were reviewed prior to commencing tezepelumab and at subsequent clinic review.

**Results** Mean baseline values (SEM) were: age 52.1 years (2.5); BMI 29.6 kg/m2 (0.9); duration on tezepelumab 7.8 months (0.4); eosinophils 745cells/uL (81); FeNO 56.9ppb (7.4); total IgE 641.6 kU/L (167.9); FEV1% predicted 74.3% (3.4); FEF25–75 % predicted 54.2% (5.9); ACQ 2.7 (0.3); AER 3.9 (0.5).

The mean changes (95% CI) in response to tezepelumab were as follows: total IgE 207.3 kU/L (49.9, 364.7) $p<0.05$, eosinophils 592cells/uL (427, 757) $p<0.001$, FeNO 29.3ppb (16.9, 41.7) $p<0.001$, FEV1% predicted 9.6% (3.2, 15.9) $p<0.01$, FEF25–75 % predicted 15.7 (5.7, 25.7) $p<0.01$, ACQ 1.0 (0.4, 1.6) $p<0.01$, AER 2.9 (2.0, 3.7) $p<0.001$.




**Abstract P204 Figure 1** Scatter plot of the change in FEF25–75% against change in FeNO showing linear regression line and 95%CI

A better correlation was found between change in FeNO and change in% predicted FEF25–75 ($r=-0.65$, $p<0.001$) (figure 1), than change in% predicted FEV1 ($r=-0.41$, $p<0,05$). However the change in eosinophils in response to tezepelumab did not significantly correlate with change in either% predicted FEF25–75 or FEV1.

**Conclusions** The response to tezepleumab was relatively greater in small than large airways. Moreover, suppression of FeNO but not eosinophils with tezepelumab was associated with improvements in FEF25–75, in keeping with expression of IL13 but not IL5 in small airways smooth muscle.

## P205 REAL WORLD OUTCOMES OF TEZEPELUMAB IN OBESE AND NON-OBESE SEVERE ASTHMATICS

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10.1136/thorax-2025-BTSabstracts.386

**Introduction** Obese patients with severe asthma have a distinct clinical phenotype. These patients are more likely to have low T2 biomarkers (T2low) so are less likely to meet criteria to be treated with most biologics. Use of Tezepelumab does not require raised eosinophils or FeNO so it may be able to reach this population. Here we present real world outcomes for patients treated with Tezepelumab comparing obese and non obese groups.

**Methods** Data from our service registry were used to study asthma outcomes from Tezepelumab comparing the obese (BMI$\geq$30 kg/m$^2$ ) and the non obese population.

**Results** A total of 142 patients were analysed (60% were obese). The cohort median BMI was 31.0 (27.2–37.2). At baseline, obese patients had non-significant trend of higher exacerbation frequency (5 vs 4.5 $p=0.371$), median ACQ (3.83 vs 3.5 $p= 0.16$) and mOCS use (8.4% vs 19.7% $n=40$ $p =0.124$) compared to non obese. 31.2% of patients had been treated with another biologic previously. Overall, 34.5% of patients were T2low.

Outcomes for patients who completed 12 month trials are described in table 1.

Trials were discontinued for adverse effects in 9.2% ($n=13/142$ including rash and joint pains. 2.8% ($n=4/142$) of patients felt no improvement in their symptoms and so trials were discontinued early at their request. A further 2.1% ($n=3$) of patients did not attend (DNA) multiple appointments and so were unable to complete trials. There were no significant differences in the responses of obese and non obese in this regard.

**Conclusion** Tezepelumab improves asthma outcomes for both obese and non obese patients. However, obese patients still have significantly higher exacerbation frequency. Symptoms remain uncontrolled in the obese group though this did not reach statistical significance. However, this may be affected by the number of patients who did not complete a 12-month trial because of adverse effects or non improvement in

**Abstract P205 Table 1** Outcomes for patients who completed 12 month trials

| | Total | BMI<30 | BMI ≥30 | p-value |
|---|---|---|---|---|
| Age (years) | 52.5 (42.5-59.5) | 48 (34-59) | 53 (45-60) | P=0.240 |
| Female Gender (frequency) | 72 | 25 | 47 | P=0.211 |
| BMI (kg/m$^2$) | 30.91 (27.00-36.75) | 25.8 (23.20-27.50) | 35.53 (31.2-40.9) | - |
| Annualised Asthma Exacerbation rate | 1 (0- 2) | 0 (0-2) | 1 (0-2) | P=0.0261 |
| Change in exacerbation frequency | -4.0 (-5 to -3) | -4 (-5 to -2) | -4 (-5 to -3) | P=0.690 |
| ACQ (median) | 2.0 (0.83 – 2.83) | 1.34 (0.33 – 2.83) | 2.09 (1.0-3.1 | P=0.131 |
| Change in ACQ | -1.5 (-2.52 to -0.83) | -1.17 (-2.74 to -0.83) | -1.5 (-2.51 to -0.83) | P=0.612 |
| Eosinophil count (x10$^9$/L) | 0.14 (0.08-0.22) | 0.14 (0.08-0.28) | 0.13 (0.07-0.21) | P=0.489 |
| Change in Eosinophil count | -0.08 (-0.20 to -0.02) | -0.09 (-0.25 to -0.02) | -0.08 (-0.18 to -0.02) | P=0.818 |
| Total IgE (kU/L) | 62.0 (18.5-140.00) | 27.00 (13.25 – 100.50) | 89 (29.5 – 157.00) | P=0.460 |
| Change in Total IgE | -177.50 (-483.00 to -14.00) | -177.50 (-726.00 to -14.00) | -122.00 (-432.00 to -29.00) | P=0.803 |
| FeNO (ppb) | 17.50 (11.00-27.00) | 17.00 (11.00 – 31.25) | 18.0 (11.25 – 26.00) | P=0.956 |
| Change in FeNO | -8.0 (-23.0 to +3.0) | -7.0 (-43 to + 3.0) | -8 (-19.0 to +3.0) | P=0.523 |
| FEV1 (L) | 2.41 (1.72-3.00) | 2.38 (1.59 to 3.12) | 2.44 (1.78 – 2.86) | P=0.964 |
| Change in FEV1 | +0.16 (0.01 – 0.45) | +0.21 (+0.008-to 0.463) | +0.14 (-0.005 to 0.443) | P=0.353 |
| Number of patients on mOCS | 13 | 6 | 7 | P=0.496 |
| Change in number of patients on mOCS | -22 | -9 | -13 | P=0.7 |
| Positive trials (frequency) | 94 | 35 | 59 | P=0.324 |
| Negative trials (frequency) | 14 | 7 | 7 | P=0.430 |
| Discontinued trials (frequency) | 22 | 12 | 10 | P=0.143 |
| 6 month extension (frequency) | 14 | 3 | 11 | P=0.134 |

symptoms. There is a need for other management options in obesity related severe asthma.

## P206 EFFECTIVENESS OF TEZEPELUMAB TREATMENT IN PATIENTS WITH SEVERE ASTHMA: REAL WORLD OUTCOMES FROM A SEVERE ASTHMA NETWORK

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**Introduction** Randomised clinical trials confirmed efficacy of Tezepelumab in treatment of severe asthma, but real-world data on its performance remains limited. This study evaluated its effectiveness in routine clinical practice across our network

**Methodology** This retrospective, observational study was conducted across nine hospital sites in central England. Eligible patients under NICE guidance were initiated on Tezepelumab following regional MDT approval. Treatment outcomes at 12 months were classified as *positive* (≥50% reduction in exacerbation frequency or maintenance oral corticosteroid [mOCS] dose) or *negative*, with early discontinuation of treatment recorded. Clinical remission was defined as: no severe exacerbations, off mOCS, and asthma control questionnaire (ACQ-6) score <1.5. Data was extracted from structured proformas. Standard and descriptive statistics were performed.

**Results** A total of 145 patients with severe asthma initiated on Tezepelumab were included in the analysis. Baseline clinical and phenotypical characteristics are summarised in table 1. Positive outcomes were seen in 67% (n=97/145), 7% (n=10/145) had borderline responses requiring review at 18 months, and 26% (n=38) discontinued due to negative response, which included adverse effects such as rash, arthralgia and myalgia, neurological symptoms, and fatigue (10%, n=13/145).

In patients with paired baseline and 12-month data (n=125), median annual exacerbations fell from 5.0 (4.0–7.0) to 1.0 (0.0–2.25) (p<0.0001), and maintenance OCS from 10.0 (7.50–15.0) to 5.0 (0.0–10.0) mg/day (p<0.0001) among those on OCS at baseline (n=44). ACQ-6 significantly improved from 3.67 (2.67–4.64) to 2.17 (1.00–4.05) (p<0.0001), and proportion of well-controlled asthma (ACQ-6

**Abstract P206 Table 1** Baseline clinical and phenotypical characteristics. *Data missing for 3 patients

| | **n=145** |
|---|---|
| **Female n (%)** | 98 (67.6%) |
| **Age mean (SD)** | 50.2 (13.3) |
| **BMI median (IQR)** | 31.0 (27.2-37.2)* |
| **Current or ex-smokers n(%)** | 63 (43.4%) |
| **Atopy n (%)** | 110 (76%) |
| **Co-morbidities n (%)** | |
| Gastroesophageal reflux disease | 92 (63.4%) |
| Rhinosinusitis | 74 (51%) |
| Underlying psychology (e.g. anxiety, depression) | 54 (37%) |
| Breathing pattern disorder | 26 (18%) |
| Obstructive sleep apnoea | 24 (16.5%) |
| Nasal polyposis | 21 (14.5%) |
| Inducible laryngeal obstruction | 21 (14.5%) |
| Atopic dermatitis | 21 (14.5%) |
| **Severe exacerbation rate in the preceding 12 months median (IQR)** | 5.0 (4.0-7.0) |
| **Maintenance OCS requirement n (%)** | 44 (30%) |
| Baseline mOCS dose (mg) median (IQR) | 10.0 (7.50-15.0) |
| **High dose ICS (2000mcg BDP) n (%)** | 87 (60%) |
| **Previous biologic(s) ever, n (%)** | 47 (32%) |
| Switch from anti-IL-5/5R | 23 (49%) |
| Switch from anti-IgE | 19 (40%) |
| Switch from anti IL-4R | 5 (11%) |
| **Baseline ACQ-6 median (IQR)** | 3.67 (2.67-4.64) |
| **Baseline blood eosinophil count in the previous 12 months ($x10^9$/L) median (IQR)** | 0.23 (0.11-0.45) |
| **Baseline FeNO (ppb) median (IQR)** | 25.0 (12.0-59.8) |
| **Baseline total IgE (kU/L) median (IQR)** | 187.0 (33.3-468.3) |
| **FEV1 (L) median (IQR)** | 2.22 (1.44-2.71) |
| **FEV1% predicted mean (SD)** | 69.5 (22.5) |

<1.5) rose from 3.5% (n=4/116) to 38% (n=44/116) (p=0.03). $FEV_1$significantly increased from 2.22 (1.44–2.71) to 2.44L (1.76–3.02) (p<0.0001). FeNO declined from 25.0 (12.0–59.8) to 18.0 (11.0–29.0) ppb (p<0.0001), blood eosinophil count from 0.23 (0.11–0.45) to 0.14 (0.07–0.24) $x10^9$/L (p<0.0001), and total IgE (n=26) from 192.0 (34.0–410.1) to 67.5 (18.0–169.0) (p=0.0002).

The proportion who achieved clinical remission at 12 months were 22% (n=27/125).

**Conclusions** Tezepelumab led to significant improvements in OCS use, asthma control, and T2 markers in a real-world setting. Two-thirds of patients had a positive response, with a subset (22%) achieving clinical remission, demonstrating clinical benefits of Tezepelumab in managing severe asthma in routine care.

## P207 PATH TO CONSENSUS: PATIENT AND PHYSICIANS PERSPECTIVES ON REDUCING BACKGROUND MEDICATION IN PATIENTS WITH SEVERE ASTHMA CONTROLLED ON BIOLOGIC THERAPY IN THE UK

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**Abstract P207 Table 1** Selected key questions and results from the asthma specialist HCP and severe asthma patient surveys

| Asthma Specialist Healthcare Professional (HCP) survey | | | | | |
|---|---|---|---|---|---|
| **Question** | **% of total survey responses** | | | | |
| When escalating therapeutic management, is one of the considerations whether you will be able to reduce background medication? | **Always** | **Often** | **Sometimes** | **Rarely** | **Never** |
| | 31 | 34 | 23 | 12 | 0 |
| When seeing patients with asthma who are receiving biologic treatment, do you assess their suitability for background medication reduction? | **Always** | **Often** | **Sometimes** | **Rarely** | **Never** |
| | 42 | 35 | 19 | 4 | 0 |
| How do you involve patients who are receiving a biologic therapy in decisions about reducing background asthma medication? Select all that apply | **Full discussion and shared decision-making** | **Follow-up consultations** | **Patient education materials** | **Other** | |
| | 24 | 17 | 7 | 0 | |
| Do you believe that reducing background asthma medication for patients with well-controlled asthma who are receiving a biologic therapy can have a positive impact on long-term asthma control? | **Yes** | **No** | **Unsure** | | |
| | 58 | 15 | 27 | | |
| | **% of responses suggesting this approach** | | | | |
| What additional evidence or guidance would help you feel more confident in reducing background medication for patients with asthma who are controlled | **On-going real world evidence** | **Protocols or guidelines** | **None** | **Clinical trials** | **Review capacity challenges** |
| | 35 | 23 | 12 | 8 | 8 |
| How do/would you address concerns from patients about reducing their background asthma medication while on a biologic therapy? | **Patient reassurance** | **Explain option to adjust/titrate treatment** | **Patient education and discussion** | **Shared decision-making** | **Patient reviews** |
| | 35 | 35 | 35 | 15 | 8 |
| **Severe Asthma Patient survey** | | | | | |
| | **% of total survey responses** | | | | |
| Following the initiation of biologic therapy, your asthma has been well controlled as a result. How important do you think it is to reduce other asthma medications (e.g. inhalers, oral steroids)? | **Very important** | **Somewhat important** | **Neutral** | **Somewhat unimportant** | **Very unimportant** |
| | 51 | 32 | 15 | 2 | 0 |
| Following the initiation of biologic therapy, your asthma has been well controlled as a result. How confident are you that reducing other asthma medications (e.g. inhalers, oral steroids) is good for your health? | **Very confident** | **Somewhat confident** | **Neutral** | **A little unsure** | **Very unsure** |
| | 34 | 44 | 12 | 7 | 2 |
| When your asthma is well controlled on biologic medication, have you lowered the dose (taken less) of your other asthma medications yourself independent to medical advice (without your Doctor having advised you to)? | | **Yes** | **No** | **Unsure** | |
| | **All patients** | 46 | 54 | 0 | |
| | **18–35 year olds** | 69 | 31 | 0 | |
| | **36+ year olds** | 32 | 68 | 0 | |

**Introduction and Objectives** Biologic therapies have proven to be effective at reducing the need for background medications.[1] GINA recommend stepping down maintenance treatment in patients with well-controlled asthma[2]; however, there remains a lack of consensus on how to safely taper background medications, including ICS, in patients with severe asthma on biologic therapy.

**Methods** A targeted literature review (TLR) evaluated evidence on reducing background medication in adults with controlled severe asthma on biologic therapy. Two surveys were conducted to gain further insights from UK respiratory healthcare professionals (HCP) and patients.

**Results**

TLR: data from 24/25 of the publications extracted supported a reduction of background medications for patients with controlled asthma after biologic treatment. A step-wise reduction in ICS dose was recommended in 18 publications, however, standard tapering criteria of medication or discontinuation sequences and monitoring requirements were not established.

HCP survey: Of the 26 participants, 77% (20) assess the patient's suitability for background medication reduction and 65% (17) always or often consider tapering background medication when escalating therapeutic management. To support the clinical application of background medication reduction in asthma management plans, 66% (17) of HCPs highlighted the need for evidence generation, protocols and guidance.

Severe Asthma Patient survey: Of the 41 patients surveyed, 83% (34) believe that reducing background medication is very or somewhat important when asthma is well-controlled on biologic therapy; 46% (19) of patients lower the dose of other asthma medication independently of medical advice. Two key barriers to reducing background medications were fear of asthma deterioration and other undesirable effects. Commonality in the need for guidance in treatment titration and monitoring was seen across the two surveys (table 1).

**Conclusion** The TLR supports background medication reduction in well-controlled asthma patients receiving biologic treatment; however, current guidance on the optimal reduction strategy is sparse. Consensus recommendations and protocols, based on the available evidence and expert opinion, could support HCPs in deciding when and how to consider background medication reduction.

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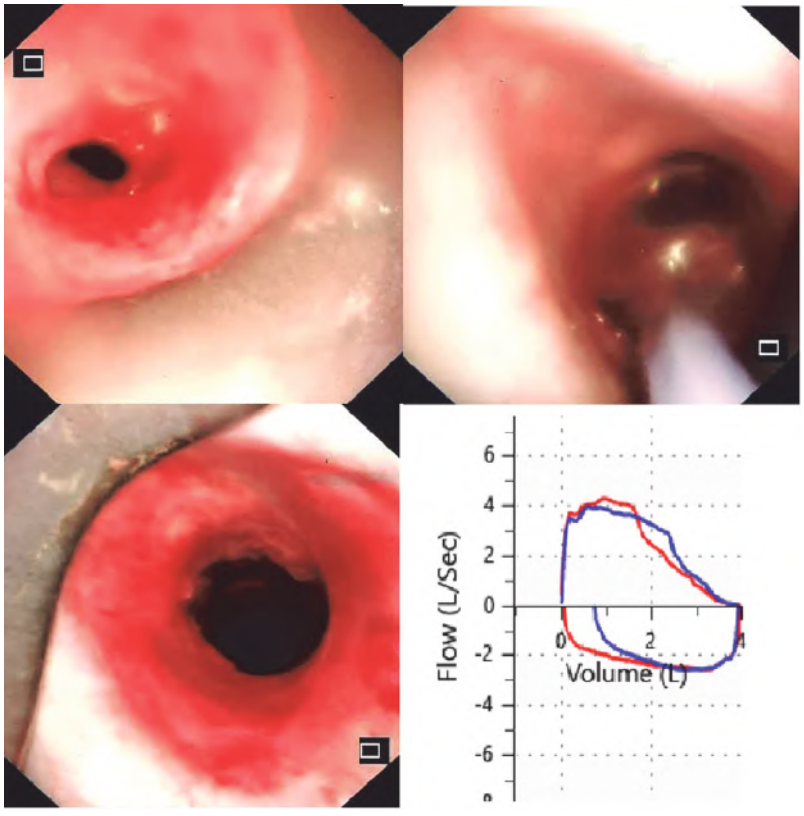
# The interventionalist's toolkit: bronchoscopy to thoracoscopy

## P208 SUB-GLOTTIC STENOSIS: A RARE BUT CLINICALLY SIGNIFICANT LARGE AIRWAY DISEASE – A SINGLE CENTRE EXPERIENCE

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**Background** Subglottic stenosis (SGS) is a rare condition with symptoms ranging from exertional breathlessness to severe airway compromise. Bronchoscopic intervention is typically the




**Abstract P208 Figure 1**

first-line intervention, with surgical resection considered in selected cases.

**Aims** To review the prevalence of SGS in our region and our clinical experience in managing this condition, including complications, recurrence, and the outcomes of bronchoscopic interventions.

**Methods** Retrospective analysis of 27 subglottic stenosis patients referred to adult respiratory service, in a large tertiary centre in UK between 2018 – 2025.

**Results** The mean age was 48.8 (range 17 – 73) years with 24 (89%) female patients. 16 (59%) patients had idiopathic SGS, 6 (22%) were associated with granulomatosis with polyangiitis (GPA), and 5 (19%) were secondary to trauma or previous intubation. Excluding trauma, prevalence of SGS in our region is approximate 1/200,000. Most were never smokers, 4 had high BMI and 4 were diagnosed as having asthma.

Diagnosis of SGS is made on bronchoscopic assessment, complemented by flow volume loops on spirometry. Tracheal biopsies during first procedure distinguishes idiopathic SGS from GPA. Extent of stenosis and distance from vocal cords are also recorded.

Under general anaesthesia using rigid and flexible bronchoscope, graded balloon dilatation was performed in 13/16 (81%) idiopathic SGS, 4/6 (67%) GPA and 2/5 (40%) post traumatic patients. 7/19 (37%) required single dilatation and 12/19 (63%) experienced recurrence requiring multiple dilatations (range 2 – 5). Time to recurrence ranged from 2 months to 10 years. No serious complications or mortality were observed. Stenosis of >70% at presentation was associated with higher risk of recurrence. All patients were discussed in the regional multidisciplinary meeting and 3 patients underwent laryngo-tracheal resection for symptom relief.

**Conclusions** Majority of SGS patients are females with no significant comorbidities. Combined rigid and flexible bronchoscopy with graded balloon dilatation is a safe minimally invasive procedure for the management of SGS, with no significant complications and avoids the need for invasive intervention in most cases. The recurrence rate is similar in patients with idiopathic SGS and those with GPA. Despite its rarity, SGS should be considered in patients presenting with unexplained exertional breathlessness.

## P209 SAFETY PROFILE IN BRONCHOSCOPIC SAMPLING OF BRONCHIAL CARCINOID TUMOURS: A RETROSPECTIVE REVIEW FROM A SINGLE TERTIARY CENTRE

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**Introduction and Objectives** Concerns about bleeding complications, driven by the vascular nature of some bronchopulmonary carcinoid tumours (BPCs) and previous data, can lead to hesitation in performing bronchoscopic biopsies in some secondary care settings. We retrospectively reviewed our tertiary centre experience over a 13-year period to determine the safety profile of bronchoscopic sampling in BPCs.

**Methods** A retrospective review of patients with BPCs undergoing diagnostic bronchoscopic biopsy between 2009–2021 was conducted using electronic patient records from a large tertiary referral bronchoscopy unit. Bronchoscopy reports were reviewed to determine complications across three diagnostic bronchoscopic procedures: standard flexible bronchoscopy, navigational bronchoscopy and endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA). Patients undergoing rigid bronchoscopy or bronchoscopy under general anaesthetic were excluded. Bleeding severity was classified based on the British Thoracic Society bronchoscopy guidelines.

**Results** The study included 59 patients (62.7% female) with a mean age of 57.6 years. Most had a performance status of 0 (57.6%) or 1 (33.8%) at diagnosis. Typical carcinoids were most commonly diagnosed (74.5%), followed by carcinoid not otherwise specified (15.2%). The primary tumour was predominantly right-sided (66.1%), most frequently affecting the right lower lobe (27.1%). The mean tumour size was 28.8 mm (IQR: 6–75 mm). There were no reports of severe bleeding (0%). Bleeding was most commonly reported as mild (16.9%) or moderate (11.8%), (see table 1), all safely managed with cold saline (n=11) or adrenaline (n=7) as necessary. The incidence was similar across all bronchoscopic procedures. Pre-biopsy interventions included topical adrenaline (27.1%) and cold saline wash (8.4%). Post-procedure, two patients (3.3%) reported chest discomfort within 48 hours, but chest X-rays in both showed no pneumothorax. No patients required blood transfusion, fluid resuscitation, acute admission or any management beyond standard bronchoscopy care.

**Conclusions** Our findings demonstrate bronchoscopic sampling of PBCs is safe with no evidence of severe bleeding across all bronchoscopic procedures, challenging the perceived need for tertiary referral for sampling. Careful reconsideration of current referral pathways could streamline care by expediting diagnosis locally, without compromising safety, whilst also alleviating pressure on tertiary services.

**Abstract P209 Table 1** Bleeding complications across all bronchoscopic procedures, pre- and post-biopsy interventions, immediate postoperative complications, and subsequent management interventions used in 59 patients identified in this study

| Bleeding complications and associated interventions | |
|---|---|
| Total bleeding cases | n = 17, (28.8%) |
| Severity | |
| No bleeding | n = 42, (71.1%) |
| Mild | n = 10, (16.9%) |
| Moderate | n = 7, (11.8 %) |
| Severe | n = 0 |
| Total Pre-biopsy interventions | n = 21, (35.5%) |
| Adrenaline | n = 16, (27.1%) |
| Cold saline wash | n= 5, (8.4%) |
| Total Post-biopsy interventions | n = 17, (28.8%) |
| Suctioning | n = 3, (5%) |
| Adrenaline | n= 7, (11.8%) |
| Cold saline wash | n= 11, (18.6%) |
| Post Procedure complications | n = 2, (3.3%) |
| Chest discomfort/pain | n= 2, (3.3%) |
| Pneumothorax | n = 0 |

## P210 THE IMPACT OF HETEROGENEITY AND FISSURE INTEGRITY ON THE TREATMENT RESPONSE OF AIRWAY SCAFFOLDS FOR EMPHYSEMA-RELATED HYPERINFLATION

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**Introduction** The Apreo BREATHE Airway Scaffold is a self-expanding nitinol implant deployed via bronchoscopy to release trapped air. The effect of emphysema distribution and collateral ventilation (CV) on treatment response is unknown. Fissure integrity (FI) can be used as a non-invasive surrogate for CV.

**Aim** To determine the impact of heterogeneity and FI on the treatment response of the BREATHE Airway Scaffold.

**Method** In the first-in-human studies in Australia (BREATHE-1) and in Europe (BREATHE-2), up to three scaffolds were implanted per lung in 50 patients with severe hyperinflation bilaterally. Heterogeneous disease was defined as a ≥15% difference between upper and lower lobes at -910HU. Unilateral homogeneous included one homogeneous and one heterogenous lung. Complete fissures included a FI >95% in at least one lung. Changes in lung function tests, questionnaires and 6-minute walk distance (6MWD) were measured 6-months post treatment.

**Results** All groups showed improvement in the outcomes at 6-months post treatment (mean±SEM).

Those with at least one heterogenous lung had a greater improvement. FI did not have a significant impact on outcomes.

**Conclusion** The BREATHE Airway Scaffold may be an effective treatment for emphysema-related hyperinflation, including in those with bilateral incomplete fissures and homogenous emphysema; groups typically with limited options.

**Abstract P210 Table 1**

| | All patients (n=50) | Emphysema heterogeneity | | | Fissure integrity | |
|---|---|---|---|---|---|---|
| | | Bilateral Heterogeneous (n=12) | Unilateral Homogeneous (n=13) | Bilateral Homogeneous (n=25) | Complete Fissure (n=27) | Incomplete Fissure (n=23) |
| RV (L) | -0.78 (0.12) | -0.95 (0.26) | -0.95 (0.26) | -0.60 (0.16) | -0.77 (0.18) | -0.79 (0.17) |
| RV/TLC (%) | -11.6 (1.9) | -15.0 (3.3) | -14.5 (4.1) | -8.4 (2.6) | -11.2 (2.8) | -12.1 (2.5) |
| $FEV_1$(L) | 0.12 (0.03) | 0.12 (0.04) | 0.19 (0.09) | 0.07 (0.02) | 0.14 (0.05) | 0.09 (0.03) |
| $FEV_1$(%) | 18.08 (3.66) | 18.74 (6.10) | 26.88 (10.48) | 13.19 (3.91) | 20.24 (5.82) | 15.55 (4.20) |
| FVC (L) | 0.51 (0.10) | 0.56 (0.21) | 0.69 (0.28) | 0.39 (0.11) | 0.49 (0.15) | 0.53 (0.14) |
| SGRQ-C (points) | -28.2 (3.3) | -23.2 (5.9) | -39.0 (5.2) | -24.9 (5.1) | -28.9 (4.3) | -27.3 (5.2) |
| CAT score | -7.5 (1.2) | -5.2 (3.3) | -11.5 (1.7) | -6.5 (1.6) | -7.4 (1.8) | -7.6 (1.7) |
| mMRC | -1.5 (0.2) | -1.3 (0.4) | -2.0 (0.3) | -1.3 (0.3) | -1.2 (0.2) | -1.7 (0.3) |
| 6MWD (meters) (n completed test) | 71.3 (11.4) (n=47) | 114.8 (20.6) (n=12) | 86.2 (16.6) (n=12) | 41.0 (16.9) (n=23) | 69.5 (15.9) (n=26) | 73.6 (16.7) (n=21) |

## P211 INTRAOPERATIVE REAL-TIME THORACIC ULTRASOUND TO PREDICT LOBAR COLLAPSE AND PNEUMOTHORAX DURING ENDOBRONCHIAL VALVE INSERTION FOR LUNG VOLUME REDUCTION

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**Background** Bronchoscopic lung volume reduction (BLVR) using endobronchial valves (EBVs) is associated with post-procedure pneumothorax in up to 30% of cases. Immediate lobar collapse is desirable but not always radiologically evident intraoperatively. This pilot case series evaluates the utility of intraoperative bedside thoracic ultrasound in identifying lobar collapse and ruling out pneumothorax in real time during EBV procedures.

**Methods** Eight patients underwent BLVR with Zephyr valves. Intraoperative bedside thoracic ultrasound was performed to assess diaphragm elevation and pleural integrity. All ultrasound findings were confirmed with post-procedure chest radiographs. Chartis assessments were negative in all cases.

**Results** The mean age was 67.1 ± 10.1 years; 4 patients (50%) were female. A median of 4 valves (range 3–5) were implanted per patient. Lobar collapse, indicated by diaphragmatic elevation or visible lung sliding loss, was observed in 6 of 8 patients (75%) via ultrasound. The remaining 2 showed no significant sonographic changes, which was confirmed by

CXR. No pneumothorax was detected intraoperatively or post-procedure. One case showed blunting of the costophrenic angle.

**Conclusion** Intraoperative bedside thoracic ultrasound is a feasible, non-invasive adjunct to EBV procedures, enabling immediate evaluation of lobar collapse and exclusion of pneumothorax. Its integration into BLVR workflow may improve procedural safety and clinical decision-making.

## P212 THE ROLE OF EBUS-TBNA WITH/WITHOUT EUS-B-FNA IN UNDIAGNOSED ISOLATED MEDIASTINAL LYMPHADENOPATHY

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**Introduction and Objective** EBUS-TBNA/TBNB is the gold-standard for real-time evaluation of mediastinal and/or hilar lymph nodes. The EBUS bronchoscope can be inserted through the esophageal route for needle aspiration of lymph nodes close the esophagus (EUS-B-FNA). A meta-analysis involving lymph node staging of lung cancer demonstrated a higher diagnostic yield of EUS-B-FNA in combination with EBUS-TBNA than using EBUS-TBNA alone (91% vs. 80%, p = 0.004). Regarding patients with undiagnosed mediastinal with/without hilar lymphadenopathy and without parenchymal lesion suspected for lung cancer, there is no similar study in the literature regarding the comparison of EBUS-TBNA/EUS-B-FNA with EBUS-TBNA alone.

The aim of the study is to demonstrate the diagnostic value of the combined approach compared with EBUS-TBNA alone in undiagnosed mediastinal lymphadenopathy.

**Methods** Prospective single-center study of patients with mediastinal with/without hilar lymphadenopathy on imaging (CT, PET/CT) including at least 2 lymph node stations with at least one of them involving the subcarinal or the left inferior paratracheal lymph node (EUS-accessible stations). Patients with intraparenchymal lesion suspicious for lung cancer are excluded from the study.

Patients are randomized into 2 groups (EBUS-TBNA/TBNB alone and combination of methods) according to two

**Abstract P212 Table 1**

| Characteristics | | EBUS (n = 22) | EBUS + EUS (n = 19) | p-value |
|---|---|---|---|---|
| **Gender**, n (%) | Female | 9 (40,9%) | 8 (42,1%) | 0.93 |
| | Male | 13 (59,1%) | 11 (57,9%) | |
| **Age**, mean (range), years | | 51,5 (36 – 76) | 57,6 (42 – 74) | 0.09 |
| **Smoking status**, n (%) | Smokers | 6 (27,3%) | 5 (26,3%) | 0.94 |
| | Ex-smokers | 9 (40,9%) | 10 (52,6%) | 0.45 |
| | Non-smokers | 7 (31,8%) | 4 (21,1%) | 0.43 |
| **Short axis of smaller biopsied lymph node**, n (%) | ≤ 15 mm | 13 (59,1%) | 13 (68,4%) | 0.53 |
| | > 15 mm | 9 (40,9%) | 6 (31,6%) | |
| **Sensitivity of procedure**, % (n) | In total | 90% (18) | 93,8% (15) | 0.86 |
| | Sarcoidosis | 84,6% (11) | 92,9% (13) | 0.49 |
| | Tuberculosis | 100% (3) | 100% (1) | 1.00 |
| | Berylliosis | 100% (1) | - | - |
| | Lymphoma | 100% (3) | 100% (1) | 1.00 |
| | Reactive | 100% (2) | 100% (3) | 1.00 |
| **Specificity of procedure**, % (n) | In total | 50% (2) | 75% (3) | 1.00 |
| **Diagnostic yield**, % | In total | 90,9% | 94,7% | 1.00 |
| **Major Complications**, n (%) | | - | - | - |

parameters of randomization. These are gender (male or female) and the short axis of the smallest lymph node (7 and/or 4L) from which the biopsy will be taken (≤ 15 mm or > 15 mm).

We defined samples as diagnostic when a definitive diagnosis (malignancy or benign disease) has been made. Patients with non-diagnostic sample results underwent other invasive procedures or follow-up of at least six months.

**Results** So far, 47 patients have been enrolled in the study. Six patients are in follow-up. The diagnostic yield for EBUS and EBUS/EUS is 90,9% and 94,7% respectively (table 1).

**Conclusions** At first glance, the combination of EBUS-TBNA/TBNB with EUS-B-FNA/FNB does not seem to be superior to EBUS-TBNA/TBNB in terms of diagnostic accuracy in undiagnosed mediastinal lymphadenopathy, however it is worth noting that 3 patients in the EBUS/EUS-B group were diagnosed only with the EUS-B-FNA samples, adding 16.7% to the diagnostic performance of the test.

## P213 IMPACT OF NODULE SIZE ON DIAGNOSTIC YIELD IN SHAPE-SENSING ROBOTIC-ASSISTED BRONCHOSCOPY

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**Background** Lung cancer screening programmes have significantly reduced mortality through earlier detection of malignancy. However, they have also increased the identification of small pulmonary nodules, which poses a challenge for traditional sampling techniques. CT-guided transthoracic needle biopsy (CT-TTNB) is the gold standard for sampling peripheral lesions, achieving high diagnostic yields (~90%) overall. However, this yield decreases for smaller nodules, with yields as low as 66% reported in nodules smaller than 15 mm.[1] Shape-sensing robotic-assisted bronchoscopy (ssRAB) offers precise navigation to peripheral lung nodules and may improve diagnostic performance, particularly for smaller lesions.

**Methods** We conducted a retrospective analysis of prospectively collected data at a tertiary referral centre. Consecutive patients who underwent ssRAB between 3rd September 2023 and 20th February 2024 were included. All procedures were performed under general anesthesia using the ION shape-sensing robotic endoluminal system (Intuitive Surgical) supported by cone-beam CT, fluoroscopy and radial endobronchial ultrasound. Nodules were sampled using transbronchial needle aspiration (TBNA), cryobiopsies and/or forceps biopsies. Lesions were stratified by size into three groups: <15 mm, 15–30 mm and >30 mm. Diagnostic yield was calculated per nodule using the strict definition outlined in the American Thoracic Society (ATS) Delphi consensus.

**Results** A total of 251 nodules from 204 procedures were analysed. The mean age was 70.3 years, and 60% were female. Of the nodules, 124 measured < 15 mm, 108 were 15–30 mm and 17 were > 30 mm. The strict diagnostic yields were 86.3%, 87% and 89.5% for the respective sizes.

**Conclusion** SsRAB achieved a high strict diagnostic yield across all nodule sizes, including those <15 mm. These findings highlight the potential of ssRAB as an effective tool for the sampling of small pulmonary nodules, a growing need as the screening programme expands. Prospective randomised control studies are warranted to directly compare different sampling modalities and determine the optimal approach.

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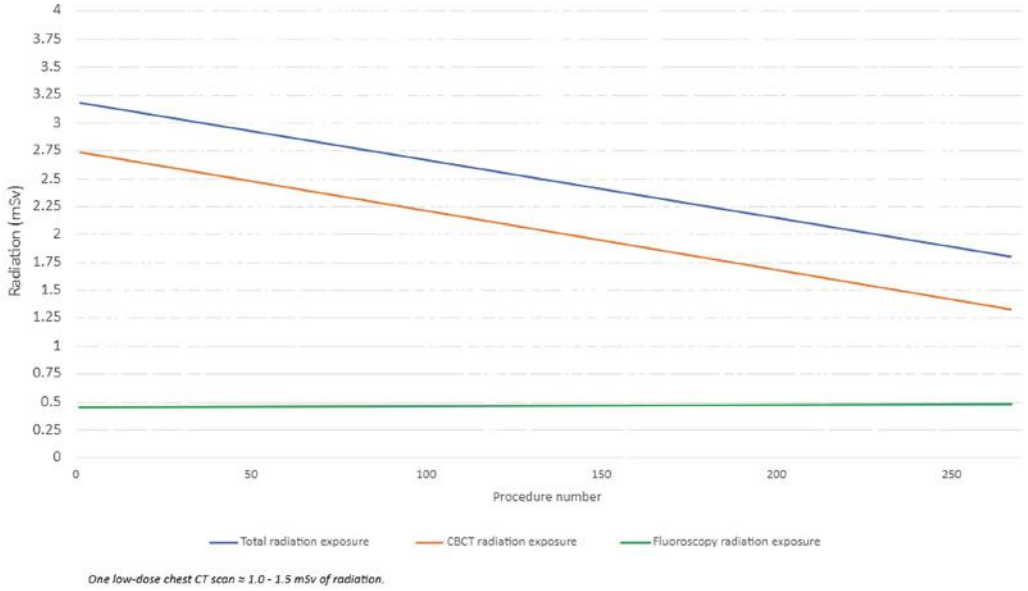
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## P214 IMPACT OF PROCEDURAL EXPERIENCE ON RADIATION EXPOSURE IN ROBOTIC-ASSISTED BRONCHOSCOPY

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**Background** Shape-sensing robotic-assisted bronchoscopy (ssRAB) is increasingly used for the diagnosis of pulmonary




**Abstract P214 Figure 1** Trends in radiation exposure over sequential procedures

nodules. Cone-beam computed tomography (CBCT) and fluoroscopy are common adjuncts, used alongside ssRAB to enhance nodule localisation and improve diagnostic yield. However, this inevitably introduces additional radiation exposure. Understanding whether procedural experience influences patients' radiation exposure is important for optimising safety. We report on the radiation exposure trends at the UK's first ssRAB centre.

**Method** We conducted a retrospective analysis of prospectively collected procedural data from patients undergoing robotic assisted biopsy of lesions at a tertiary referral centre, from 21st November 2023 to 20th February 2025. Data collected included total radiation dose, CBCT radiation and fluoroscopy radiation. To assess trends over time the dataset was divided into three equal, consecutive cohorts based on procedure date. Patients undergoing sampling of multiple lesions or additional interventions were excluded.

**Results** 267 out of 268 patients had complete radiation dosage and CT spins documented and were included in the analysis. The mean age was 69.4 years and mean BMI was 25.67. 56.6% were female and 28% had underlying lung disease (inc. COPD and ILD). Mean total radiation dose, CBCT radiation, and fluoroscopy radiation was 2.49 mSv, 2.03 mSv, and 0.46 mSv respectively.

In the three consecutive cohorts, mean total radiation was 3.12 mSv, 2.34 mSv, and 2.0 mSv, respectively. Mean CBCT radiation was 2.65 mSv, 1.87 mSv, and 1.56 mSv, respectively. Mean fluoroscopy radiation was 0.47 mSv, 0.47 mSv, and 0.44 mSv, respectively.

**Conclusion** As procedural experience with ssRAB increased, there was a notable reduction in total radiation dose and CBCT radiation dose. These findings support the development of highly experienced, high volume specialised centres for ssRAB. Further studies are required to understand other factors impacting radiation exposure and the broader impact on service development.

## P215 IMPLEMENTING ROBOTIC-ASSISTED BRONCHOSCOPY WITHIN THE NHS: OUTCOMES FROM THE FIRST 100 CASES PERFORMED AT A LARGE UK DIAGNOSTIC CENTRE

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**Introduction** Robotic-assisted bronchoscopy (RAB) enhances navigation, reach, and diagnostic accuracy for peripheral lung nodule biopsies. Prospective studies have demonstrated a diagnostic yield of 86–93% with low complication rates. Whilst available in >500 USA centres, it's adoption across Europe is at an early stage. In this abstract we report outcomes from the first 100 RAB cases performed at a UK trust.

**Methods** Our RAB procedures are performed between two interventional bronchoscopists, under general anaesthetic, in a hybrid theatre space. The team uses the Ion® Endoluminal Robotic Bronchoscopy System (*Intuitive Surgical*), integrated with the Cios-Spin mobile imaging system utilising cone-beam CT (*Siemens*). A prospective database is used to collect patient, nodule, procedural details and clinical outcomes. Here, we report outcomes from the first 100 cases performed between September 2024 and April 2025.

**Results** Mean age was 68 (range 47–88; 50% female). Median lesion size was 22 mm, reducing from 24.5 mm in the first 50 cases to 17.5 mm in the second 50 cases. 87% of lesions were in the middle-to-outer thirds of the lung.

Diagnostic accuracy was 94%, with a diagnostic yield of 86% (strict criteria). Tool-in-lesion was achieved in 97% of cases. There have been no major complications and no pneumothoraces. 9 patients had minor bleeding, managed endobronchially, and no acute clinical admissions.

Two thirds (n=65/100) of cases did not have a safe alternative biopsy option, defined as either not amenable, or extremely challenging and high risk for a CT-guided biopsy. Of these, 49/65 had a lung cancer diagnosed, from which 32/49 had surgical resections. 14/65 patients had a true negative diagnosis, all of whom had suspected lung cancer radiologically. 96% of samples requiring further molecular analysis had adequate material.

25% of cases also had concurrent staging EBUS-TBNA, of which 96% (n=24/25) confirmed adequate N0 disease.

**Conclusion** RAB can be implemented within NHS services in a safe and effective manner. It can be focussed to target lesions not amenable to other forms of biopsy and be combined with staging EBUS-TBNA when indicated. Further prospective work is needed to define its exact role in optimal lung cancer pathways and determine cost-effectiveness within the NHS.

## P216 ROBOTIC ASSISTED BRONCHOSCOPY WITH CONE-BEAM CT IMAGING FOR MULTIPLE NODULES: DIAGNOSTIC AND SAFETY OUTCOMES

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**Introduction** The introduction of lung screening programmes and more widespread use of CT imaging has led to a rise in the number of identified pulmonary nodules. It has been reported that 48% of baseline scans in the NELSON study had multiple nodules.[1] This raises the question about potential management of these nodules especially if there is no evidence of lymph node involvement and when multiple nodules can upstage primary lung malignancy. While CT-guided transthoracic biopsy remains the gold standard method, the role of shape-sensing robotic assisted bronchoscopy (ssRAB) in this cohort of patients has not been fully explored. We report here the diagnostic and safety outcomes for multiple nodule biopsy using ssRAB with cone-beam CT (CBCT) imaging.

**Methods** Between April 2023 and February 2025, a retrospective analysis was performed for prospectively collected data on patients undergoing ssRAB with CBCT for two or more suspicious nodules. All patients underwent a procedure under general anaesthesia with adjunctive radial EBUS. Demographics, nodule characteristics, diagnostic and safety outcomes were collected.

**Results** A total of 611 nodules were biopsied in 500 patients. There were 95 patients who underwent procedures for multiple nodules, of which 15 were excluded due to lesion size over 30 mm. The final analysis included 80 patients with 171 biopsied nodules. 70 patients had 2 nodules; 9 had 3 nodules

and 1 had 4 nodules biopsied. 50% of the procedures were bilateral. The diagnostic yield was 92.5%. Synchronous tumours were identified in 15 patients and metastatic disease in 10 patients. There were 8 pneumothoraces (10%) with 4 (5%) requiring a chest drain.

**Conclusion** The use of shape-sensing robotic-assisted bronchoscopy with CBCT to biopsy multiple nodules is feasible with an excellent diagnostic yield and safety profile. There is additional evidence that biopsying multiple nodules can impact management through the identification of synchronous lesions and potentially increase staging accuracy for cancer diagnoses.

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## P217 DOES AUGMENTED FLUOROSCOPY WITH LUNG VISION NAVIGATION ENHANCE THE DIAGNOSTIC YIELD OF RADIAL EBUS MINI PROBE GUIDANCE FOR PERIPHERAL PULMONARY LESIONS?

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**Introduction and Background** Accurate diagnosis of peripheral lung lesions can be challenging and requires multiple bronchoscopy techniques. The Lung Vison Navigation system (LV) utilises augmented fluoroscopy to facilitate localization of lesions. This study compares diagnostic outcome of mini probe Radial EBUS (MP) alone versus with combination of MP with LV.

**Methodology** This retrospective study was performed on 71 patients who underwent sampling of peripheral lung lesions before and after introduction of LV in June 2023. MP group included 39 patients who underwent sampling before introduction of LV and LV group included 32 patients who underwent sampling during the first few months after introduction of LV.

**Results** All the patients underwent procedures under general anaesthesia. Diagnostic yield was higher in LV group compared to MP group (81% vs. 64%, OR 1.23, 95% CI 0.45–3.37, $p = 0.682$). Significantly higher numbers of samples were adequate for molecular markers in LV group ((78% vs. 54%, $\chi^2 = 4.54$, $p = 0.033$). Lesser number of patients from LV group required further diagnostic procedures compared to MP group, although there was no statistical significance for this observation. There was one pneumothorax, and a minor bleeding reported from MP group, both of which were conservatively managed. There was one case of atrial fibrillation reported from LV group which was manged medically.

**Conclusion** This study was conducted during the period immediately after introduction of Lung Vision Navigation. Combining LV with REBUS mini probe guided biopsy led to improved diagnostic yield, reduction of need for repeat procedures as well as significant increase in sample adequacy for molecular testing, with minimal complications. Lung Vision system is a safe and effective adjunct to mini probe guided biopsy.

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## P218 HIGH FREQUENCY BRONCHOSCOPIC MICROWAVE ABLATION FOR PATIENTS WITH LUNG CANCER: INTERIM RESULTS FROM THE UNITED KINGDOM

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**Introduction** High frequency bronchoscopic microwave ablation (MWA) is a novel alternative to conventional lung cancer treatments.

**Aims** To investigate the feasibility of high frequency bronchoscopic MWA in patients with lung cancer.

**Methods** We report the interim results of a single arm prospective study enrolling subjects with confirmed malignant or highly suspicious pulmonary nodules ≤20 mm, who are not surgical candidates.

**Abstract P217 Table 1** Comparison of mini probe R-EBUS group and lung vision navigation group

| | Mini probe group (n=39) | Mini probe with Lung Vision Navigation (n=32) |
|---|---|---|
| Mean age | 74.2 years | 70.5 years |
| Male: Female | 17:22 | 15:17 |
| Mean lesion size | 2.7cm | 2.6cm |
| Diagnosis achieved | 25 (64%) | 26 (81%) |
| Sample adequacy for molecular markers | 21 (54%) | 25 (78%) |

**Results** 19 nodules were ablated in 19 subjects. Median age was 73 years. 68.4% were female. 58% of subjects had previous thoracic surgery, radiotherapy, or percutaneous ablation. 17 out of 19 high frequency MWA were delivered robotically.

58% of the nodules were solid; 42% were part-solid. Median baseline long axis nodule length was 9.9 mm. Median distance was 15.1 mm from pleura and 43.1 mm from central zone. Median procedure time was 60 mins, with a median hospital stay length of 1 day.

Median length of ablation zone was 27.1 mm at Day1 (n=19), 21.6 mm at 1-month (n=16), 17.1 mm at 3-month (n=15), 16.2 mm at 6-month (n=12) and 14.5 mm at 12-month (n=6).

Median patient-reported outcomes are as follows (*p<0.05, **p<0.001 compared to baseline):

**Abstract P218 Table 1**

| Time post-ablation | Baseline (n=19) | Day 1 (n=19) | Month 1 (n=16) | Month 3 (n=15) |
|---|---|---|---|---|
| EQ-5D Index (IQR) | 0.8 (0.2) | 1.0 (0.2)** | 0.8 (0.3) | 0.8 (0.2) |
| EQ-VAS Score (IQR) | 70 (25) | 80 (20)* | 80 (17.5) | 80 (25) |
| Pain score (IQR) | 0 (3.0) | 0 (1.0) | 0 (2.0) | 0 (0) |

Disease progression occurred in 1 patient at 6 months, and 1 at 12 months.

**Conclusion** Interim data suggests high frequency bronchoscopic MWA may be a feasible alternative for treating patients with lung cancer who are not suitable for surgery, and is associated with low morbidity.

## P219 TEN YEAR AUDIT OF ENDOBRONCHIAL ULTRASOUND (EBUS) BIOPSIES: DIAGNOSTIC YIELD AND CLINICAL INDICATIONS (2014–2024)

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**Introduction** Endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) is a minimally invasive, highly effective tool for evaluating mediastinal and hilar lymphadenopathy. While commonly used in lung cancer staging and diagnosis, EBUS also plays a diagnostic role in patients with unexplained mediastinal lymphadenopathy.

**Aims** Understand the outcome of patients undergoing EBUS for mediastinal lymphadenopathy of uncertain cause.

**Methodology** A retrospective audit was conducted of all EBUS-TBNA procedures performed between 2014 and 2024. Data were extracted from University Hospitals Plymouth NHS Trust EBUS database. Routinely, indication for EBUS is pre-categorised into 6 categories by the operator at the time of the procedure: These categories include: suspected lung cancer, suspected metastatic disease, isolated mediastinal lymphadenopathy of uncertain cause, cancer recurrence, research, and other. Histopathological findings were reviewed to assess diagnostic yield and disease distribution.

**Results** During the audit period, 2343 EBUS were performed with 3891 samples taken, including suspected lung cancer (n=2463), suspected metastatic disease (n=376), isolated mediastinal lymphadenopathy of uncertain cause (n=923), cancer recurrence (n=86), research (n=2), and other (n=27).

Of the 923 EBUS for mediastinal lymphadenopathy of uncertain cause, 463 (50%) were diagnosed with granulomatous inflammation consistent with sarcoidosis, 17(2%) had lymphoma, and 19(2%) had malignant diagnoses (e.g., NSCLC or SCLC). In contrast, among those with suspected lung cancer or metastatic disease, the diagnostic yield was dominated by malignancy (e.g., NSCLC adenocarcinoma: 543 cases; SCLC: 289 cases).

**Conclusion** In patients with mediastinal lymphadenopathy of unclear aetiology, most patients were diagnosed with sarcoidosis or had normal lymph node histology. The risk of lymphoma was <2%.

## P220 A SERVICE EVALUATION OF THE ENDOBRONCHIAL ULTRASOUND SERVICE (EBUS), INCLUDING MOLECULAR DIAGNOSES AND PATIENT SATISFACTION AT UNIVERSITY HOSPITAL SERVICE (UHS)

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**Background** EBUS-Transbronchial Needle Aspiration (TBNA) is a minimally invasive procedure which collects biopsies in

**Abstract P219 Table 1**

| Diagnosis Category | Suspected Lung Ca | Suspected Metastasis | Uncertain Lymphadenopathy |
|---|---|---|---|
| NSCLC (all variants) | 867 (35.2%) | 40 (10.6%) | 10 (1.1%) |
| SCLC | 289 (11.7%) | 7 (1.9%) | 3 (0.3%) |
| Metastatic Cancer (all origins) | 20 (0.8%) | 246 (65.4%) | 4 (0.4%) |
| Lymphoma | 11 (0.4%) | 7 (1.9%) | 17 (1.8%) |
| Carcinoid/Neuroendocrine | 13 (0.5%) | 0 (0%) | 2 (0.2%) |
| Granulomatous/TB/Fungal | 38 (1.5%) | 36 (9.6%) | 469 (50.8%) |
| Non-diagnostic | 11 (0.4%) | 4 (1.1%) | 7 (0.8%) |
| Negative | 1209 (49.1%) | 146 (38.8%) | 406 (44.0%) |

Table of final diagnoses in the top 3 categories for undergoing EBUS.

**Abstract P220 Table 1** Number and ages of different EBUS-TBNA diagnoses

| Condition Diagnosed | Percentage (N) | Age in Years (median±IQR) |
|---|---|---|
| Primary Lung Cancer | 47.12% (131) | 68.00±17.00 |
| Sarcoidosis | 23.38% (65) | 57.00±23.50 |
| Tuberculosis | 1.80% (5) | 35.00±18.50 |
| Metastatic Cancer | 9.71% (27) | 73.00±16.00 |
| Reactive Lymph Node | 0.72% (2) | 73.50±0.00 |
| No Atypical Cells | 17.27% (48) | 71.50±17.25 |
| Total | 100% (306) | 66.00±21.00 |

patients with mediastinal and/or hilar lymphadenopathy, often in lung cancer or sarcoidosis. Cancerous biopsies can undergo molecular testing to identify mutations, which may be targeted by immunotherapy. The role of EBUS-TBNA in molecular outcomes has not been studied previously in the UK. This study also explores patient demographics and satisfaction to evaluate EBUS-TBNA comprehensively.

**Aim** To evaluate the EBUS-TBNA service at UHS, focusing on patient demographics, satisfaction, diagnostic outcomes, procedural statistics, and identifying targetable mutations for immunotherapy.

**Method** 306 patients were studied retrospectively, and 39 were prospective, using a questionnaire. Data on their characteristics, reports, diagnoses, and molecular tests were recorded and analysed using SPSS.

**Results** 47.12% were diagnosed with primary lung cancer, 23.38% with sarcoidosis, 9.71% with metastatic cancer and 1.95% with other diagnoses, such as tuberculosis. Primary lung cancer was categorised as adenocarcinoma (52.31%), squamous cell carcinoma (SCC) (25.38%) and small cell carcinoma (SCLC) (16.92%).

Of the primary cancer cases, 60.31% underwent molecular testing. 29.41% of adenocarcinoma tested positive for a targetable mutation: 85% were KRAS G12C, and 15% were the EGFRE19del. Overall, 15.27% of EBUS-TBNA procedures for primary lung cancer revealed a clinically actionable variant.

Of the 39 patients surveyed, there was a significant difference between pain experienced (median = 2/10, IQR = 4) and expected pain (median = 5/10, IQR = 6) using a Wilcoxon-signed ranked test (z = -2.91, p=0.004). The average experience with staff during the EBUS was 9.87/10 (SD = 0.47), and 55.85% waited <1 week for the procedure.

**Conclusions** EBUS-TBNA's role in diagnosing various conditions, especially primary lung cancer, is clear.

Clinically, EBUS-TBNA provides genetic diagnoses, which can enable immunotherapy. Patient satisfaction is high, with patients expressing relief after the procedure and finding the staff exceptional.

## P221 FEASIBILITY AND SAFETY OF SINGLE-PORT THORACOSCOPIC PLEURODESIS WITH SAME-SITE INDWELLING PLEURAL CATHETER INSERTION FOR MANAGEMENT OF MALIGNANT AND UNEXPLAINED EXUDATIVE PLEURAL EFFUSION

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**Background** Thoracoscopic talc pleurodesis remains a mainstay in the management of malignant pleural effusions (MPE). Integrating same site indwelling pleural catheter (IPC) insertion during thoracoscopy has been shown in recent studies to facilitate early discharge, improve pleurodesis success, and support outpatient care. However, real-world evidence on the safety and feasibility of this single-port combined approach remains limited.

**Methods** This was a retrospective single-centre study of all consecutive patients who underwent single-port thoracoscopic talc pleurodesis with same-site IPC insertion between January 2019 and December 2024. Patients were followed for a maximum of 6 months. Data were collected on demographics, known malignancy at the time of the procedure, IPC duration (days), pleurodesis success (defined as IPC removal without recurrence of effusion), and procedure-related complications.

**Results** A total of 194 patients were included (67.0% male; mean age 69 ± 11.5 years). Thirty-seven patients (19.1%) had a known malignancy at the time of thoracoscopy. IPCs remained in situ for more than 182 days in 11 patients (5.7%), and 18 patients (9.3%) died within 6 months with the IPC in place. Among those with IPC duration ≤182 days, the mean duration was 34.4 ± 34.8 days, with a median of 20 days (IQR 14–47).

Although minor surgical emphysema involving the IPC port site or ipsilateral chest wall was common, significant surgical emphysema, defined as extension to at least the neck and subpectoralis major region was observed in 1 patient (0.5%). This

**Abstract P221 Table 1** Summary table of key results

| Parameter | Value |
|---|---|
| Total patients | 194 |
| Gender distribution | 130 Male (67.0%), 64 Female (33.0%) |
| Mean age ± SD | 69 ± 11.5 years |
| Known malignancy at procedure | 37 (19.1%) |
| IPCs in situ >182 days | 11 (5.7%) |
| Deaths with IPC in situ | 18 (9.3%) |
| Mean IPC duration (≤182 days) | 34.4 ± 34.8 days |
| Median IPC duration (≤182 days) | 20 days (IQR: 14–47) |
| Significant surgical emphysema | 1 (0.5 %) |
| IPC-related complications | 10 total (5.2%) — Severe Pain: 4 (40.0%), Infection: 4 (40.0%), Leakage: 1 (10.0%), Dislodged: 1 (10.0%) |

patient required hospitalisation the following day for symptom management.

IPC-related complications occurred in 10 patients (5.2%). Severe pain requiring early IPC removal was noted in 4 patients (2.1%). Infection was seen in 4 patients (2.1%): 3 (1.5%) had localised infections managed with a short course of antibiotics, while 1 (0.5%) developed malignant empyema necessitans. Leakage and catheter dislodgement were each observed in 1 patient (0.5%). Early IPC removal due to pain did not compromise pleurodesis success.

**Conclusion** Single-port thoracoscopic pleurodesis with same-site IPC insertion is a feasible and generally safe option for managing MPE and unexplained exudative effusions. The technique offers high pleurodesis success with manageable complications, supporting its use in routine clinical practice.

## P222 EVALUATION OF DRAINAGE AND CLOSURE METHODS FOLLOWING LOCAL ANAESTHETIC THORACOSCOPY (LAT): A MULTI-CENTRE ANALYSIS OF COMPLICATIONS AND OUTCOMES

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10.1136/thorax-2025-BTSabstracts.403

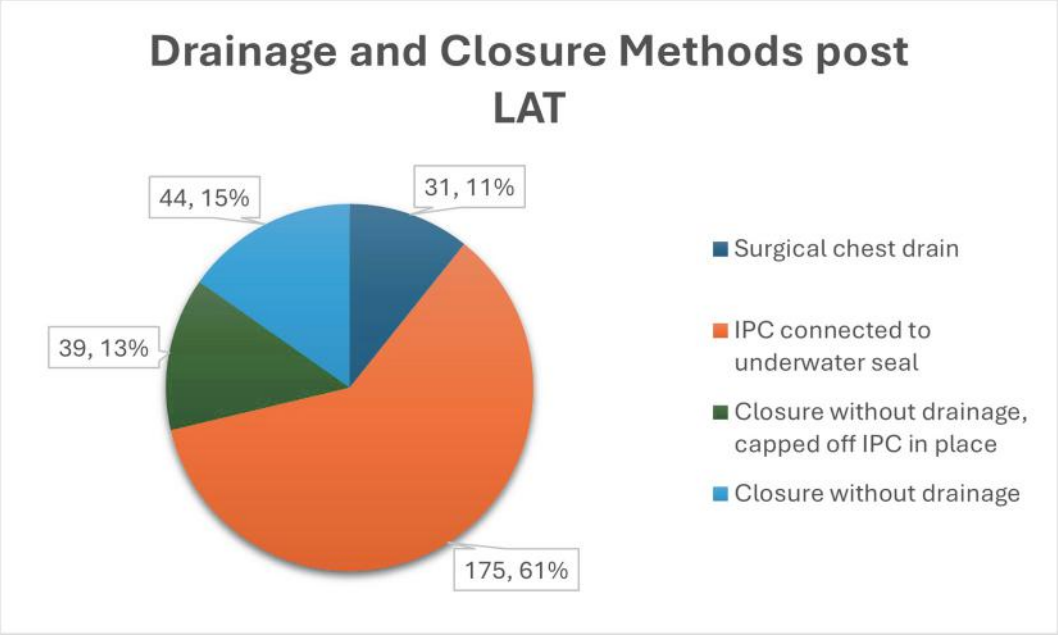
**Introduction** Local Anaesthetic Thoracoscopy (LAT) is a well-established procedure for evaluating unexplained exudative pleural effusions. The standard approach involves the insertion of a chest drain post-LAT, connected to an underwater seal to facilitate lung re-expansion and reduce the risk of surgical emphysema (SE).

Alternative techniques include placement of a long-term indwelling pleural catheter (IPC) when indicated, connected to an underwater seal or capped, as well as primary closure of the LAT incision without drainage.

**Aim** To evaluate different methods of incision closure and drainage following LAT, and to assess associated complications.

**Methods** Retrospective review of 289 patients undergoing LAT between 2022 and 2024 across three UK centres.

**Results** Mean age 71.5 years (range 33–92); 222 (76.8%) males. LAT was performed as a day-case procedure in 246 patients (85.1%). 31 (10.7%) had large bore drain insertion removed prior to discharge; 175 (60.6%) had IPC connected to an underwater seal until discharge; 39 (13.5%) had a capped IPC with no drainage and 44 (15.2%) had primary incision closure without drainage.




**Abstract P222 Figure 1**

6 (2.1%) intraoperative complications, none related to drainage or closure methods. 19 (6.6%) had post LAT complications within 30 days.

Of the 175 with post LAT IPC and underwater seal, 3 (1.7%) had local infection, 1(0.57%) had pneumonia, 4 (2.3%) had ongoing air leak or clinically significant surgical emphysema requiring admission; 3 (1.7%) had blocked IPC and 2 (1.1%) had complications unrelated to drainage method.

Of the 83 who had primary closure with or without capped IPC, 3 (3.6%) had clinically significant surgical emphysema, 1 (1.2%) slow to re-expand lung,1 (1.2%) local infection and 1(1.2%) complication unrelated to drainage method. There were no deaths related to LAT.

**Conclusions** Our study demonstrates that various drainage and incision closure techniques following LAT, performed as day-case procedures, are generally safe and associated with low complication rates. The less conventional approach - primary incision closure and no drainage – does not appear to increase the risk of clinically significant surgical emphysema (SE) compared to post LAT drainage with either a short-term chest drain or IPC. Further prospective studies are warranted to compare these techniques.

# Ventilatory support in focus

## P223 LONG-TERM ADHERENCE TO NON-INVASIVE VENTILATION IN PATIENTS WITH MYOTONIC DYSTROPHY

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10.1136/thorax-2025-BTSabstracts.404

**Background** Myotonic dystrophy (MD) is a neuromuscular disease that may affect the respiratory muscles contributing to the development of chronic hypercapnic respiratory failure. Long-term non-invasive ventilation (LT-NIV) is indicated for these patients; however the adherence to LT-NIV has been commonly described to be suboptimal. The aim of this project was to investigate the factors associated with adherence to LT-NIV in an attempt to provide interventions to improve LT-NIV use.

**Methods** We analysed the clinical data of patients with MD referred to two large tertiary long-term ventilation centres in Liverpool and Manchester. Factors associated with adherence were analysed with Cox-regression and Cox-Mantel test.

**Results** Out of 46 patients identified in this project, 26 were selected for the analysis (10 were not on positive airway pressure, 6 were on CPAP and in 4 patients no long-term follow up data was available). 20% of patients completely stopped LT-NIV by 28 days, 31% by 6 months, 40% by 1 year and 61% by 5 years. Patients with bulbar symptoms had significantly worse adherence to LT-NIV ($p=0.02$); whilst patients with atrial fibrillation were more likely using LT-NIV ($p=0.03$). Adherence was not related to age, sex, body-mass index, other comorbidities, the presence of respiratory symptoms, pre-setup nocturnal oximetry results, pre-setup blood gases, the mode of NIV or NIV settings (all $p>0.05$).

**Conclusions** The adherence to LT-NIV is variable in MD with a significantly high percent of patients stopping LT-NIV early. Bulbar symptoms may prevent effective NIV use. Further studies are warranted to identify treatable traits to improve adherence to LT-NIV.

## P224 'NIV? NOT FOR ME.' – A PILOT RETROSPECTIVE 'SERVICE IMPROVEMENT' QUALITATIVE STUDY EXAMINING THE PSYCHOLOGICAL IMPACT OF HOSPITAL-BASED NON-INVASIVE VENTILATION (NIV) WHEN TREATING ACUTE TYPE 2 RESPIRATORY FAILURE

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10.1136/thorax-2025-BTSabstracts.405

Non-invasive ventilation (NIV) has proven clinical benefits in treating acute type 2 respiratory failure (T2RF), but can contribute to distress in the acute setting (Demoule *et al.*, 2018). This anxiety can result in poor NIV compliance - impacting on clinical outcomes such as length of stay, mortality, and morbidity (Yıldız *et al.*, 2022).

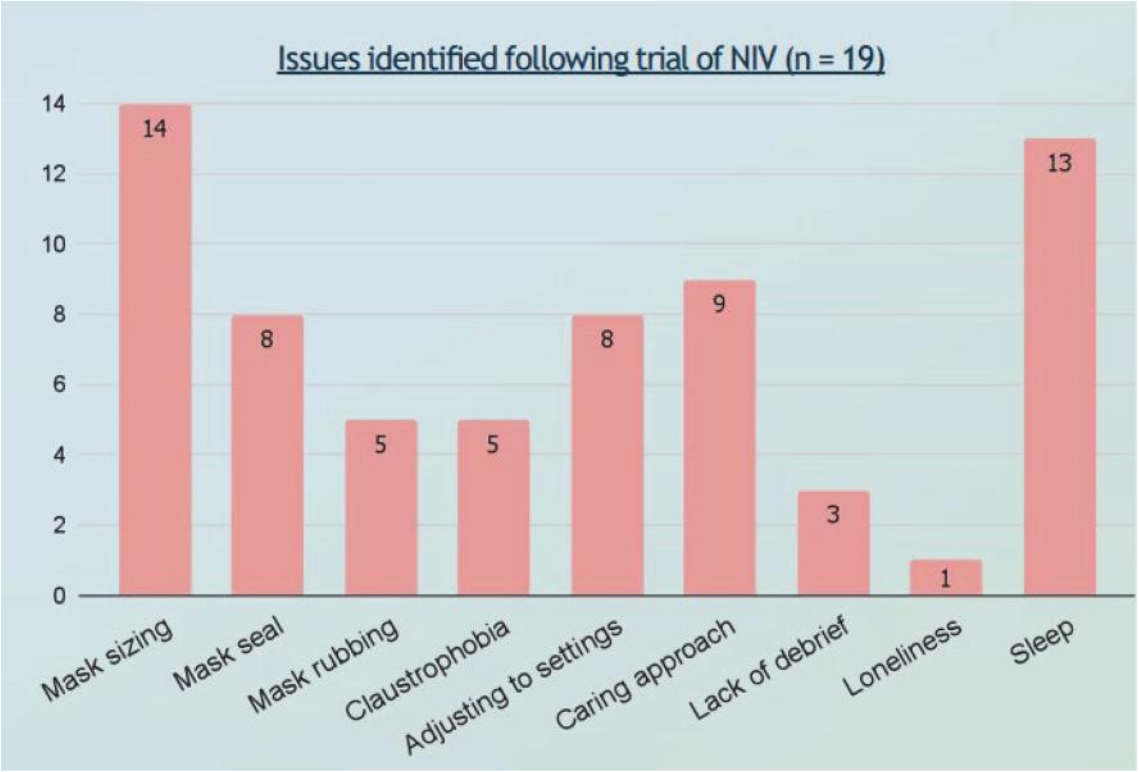
This study sought to identify the contributory factors to distress when acute NIV is initiated, thus illuminating key actions to alleviate anxiety and promote patient compliance.

All patients who received acute NIV for T2RF within a three-month period were approached with written information to obtain study consent. A semi-structured standardised telephone interview was conducted two weeks post-discharge with participants to minimise recall bias. Interviews were audio recorded, transcribed, and analysed by trained members of the study team. Patients were given the option to opt out at any time.

42 patients were identified as eligible to participate. Amongst these, the study recruited 19 patients from 1 December 2024 to 1 March 2025. 4 patients consented were lost to follow up with 1 patient withdrawing initial consent. 8 patients declined to participate. A number of patients were deemed inappropriate due to frailty (n=4), being end of life (n=8) and lacking capacity (n=3). BTS NIV Audit was conducted parallel to assess confounding factors.

Qualitative thematic analysis identified aspects of patient care pivotal to compliance and patient anxiety. Key themes included difficulties with mask fitting and claustrophobia, interrupted sleep for monitoring, and communication from clinical staff to reassure and convey important clinical information on progress (see figure 1).

A targeted action plan to focus on mask fitting, seal, comfort and adjustment, and communication was implemented. Healthcare staff involved in NIV provision attended advanced communication and interdisciplinary simulation training. Twice daily prompts were included in the NIV care plan to support communication on weaning and clinical progress. Open dialogue from clinical staff to assess comfort and sleep quality and pressure sore areas were standardised. A variety of masks with adjustable straps and interfaces were introduced.




**Abstract P224 Figure 1**

A repeat analysis of patient satisfaction and anxiety/distress levels is planned to be conducted in the autumn.

## P225 BRONCHIECTASIS IS ASSOCIATED WITH A HIGHER RISK FOR INFECTIVE EXACERBATIONS IN PATIENTS WITH CHRONIC HYPERCAPNIC COPD RECEIVING LONG-TERM NON-INVASIVE VENTILATION

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10.1136/thorax-2025-BTSabstracts.406

**Introduction** Patients with chronic obstructive pulmonary disease (COPD) and chronic hypercapnia are at a higher risk of acute exacerbations (AEs) compared to those without hypercapnia. Long-term non-invasive ventilation (LT-NIV) can reduce this risk, but its effectiveness may be diminished in patients with coexisting bronchiectasis. It remains unclear whether bronchiectasis independently increases the risk of AEs in patients with chronic hypercapnic COPD receiving LT-NIV.

**Methods** We retrospectively identified 335 patients with chronic hypercapnic COPD who were initiated on LT-NIV at our tertiary ventilation centre between September 2011 and September 2023, with follow-up continuing until January 2025. Bronchiectasis was diagnosed based on chest CT findings, and severity was quantified using the Bronchiectasis Severity Index (BSI) and the FACED score. Clinical data at LT-NIV initiation and during follow-up were analysed and compared between patients with and without bronchiectasis. A Cox-regression analysis was conducted to evaluate the impact of bronchiectasis on AEs.

**Results** The median follow-up time was 26 months. Ninety-five patients (28.4%) were diagnosed with bronchiectasis. Prior to LT-NIV initiation, patients with bronchiectasis experienced more exacerbations than those without (median (IQR): 4 (2–4) vs. 3 (2–5); p=0.02). However, bronchiectasis was not associated with exacerbation-free survival after LT-NIV initiation (p=0.24). Notably, patients with bronchiectasis had significantly shorter infective exacerbation-free survival (p=0.04). A higher BSI score prior to LT-NIV initiation was significantly correlated with increased total exacerbations ($\rho$=0.51, p<0.01) and infective exacerbations ($\rho$=0.34, p<0.01), but not with exacerbation-free survival after LT-NIV initiation (both p>0.05). The FACED score showed no correlation with exacerbations before or after LT-NIV initiation.

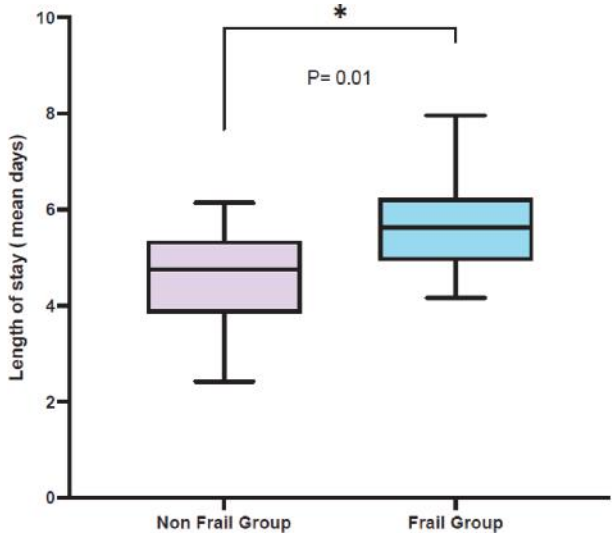
**Conclusion** Coexisting Bronchiectasis increases the risk of infective exacerbations in patients with chronic hypercapnic COPD receiving LT-NIV. This highlights the importance of actively screening for and managing bronchiectasis in this patient population.

## P226 UTILISING ROCKWOOD CLINICAL FRAILTY SCORE (CFS) IN EVALUATING THE IMPACT OF PRE- ADMISSION FRAILTY ON CLINICAL OUTCOMES IN PATIENTS ADMITTED TO ACUTE RESPIRATORY CARE UNIT (ARCU)

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10.1136/thorax-2025-BTSabstracts.407

**Introduction** Frailty is a complex, multidimensional syndrome characterised by loss of physiological reserves that increase patients' susceptibility to adverse health outcomes. Rockwood Clinical Frailty Score (CFS), a semi quantitative tool used in




**Abstract P226 Figure 1** Showing the length of stay between non frail and frail group of patients

the BTS adult respiratory support audit in 2023 showed that patients in our ARCU ranged from moderate to very severely frail and this was higher compared to national average. Inpatient respiratory admissions increase the burden of frailty and less is known about the impact on various clinical outcomes in patients admitted to ARCU and we have evaluated this further.

**Methods** This was a single centre retrospective observational cohort study, and patients admitted to ARCU acutely for respiratory support (NHFT, CPAP or NIV) based on pre-set admission criteria were included over a 12 month period (May 2023-April 2024). Patients not needing respiratory support and elective admissions were excluded. Baseline demographics and CFS ranging from score 1–9 were obtained (score 1= very fit to score 9= terminally ill). The CFS score was divided into two groups; non frail (CFS score 4 or less) and frail (CFS score of 5 or more). Length of stay (LOS), 30 day readmission rate to ARCU, inpatient and 90 day mortality between the two groups were compared.

**Results** Patients (n=735, 56% females, mean age-67± 2) were included. No patients had a CFS score of 9 (terminally ill). As compared to non-frail group, patients in frail group had increased LOS (mean days= 4.5 ±1 v/s 5.6 ±1; P= 0.01, 95% CI 0.19- 1.92, figure 1) and were more likely to get readmitted to ARCU within 30 days (11.4% v/s 24.5%; P= < 0.0001, OR= 2.5). The inpatient and 90 day mortality was higher in the frail group as compared to no frail group (30.4% v/s 19.4%; P= 0.002, OR =1.8) and (13.4% v/s 3.68%; P= 0.001, OR= 4.0) respectively.

**Conclusion** Our data suggests that higher CFS score predict adverse outcomes and may identify the high-risk patients with guarded prognosis. When incorporated into acute assessment, this may be a potential tool to help prognosticate, to aid effective communication with patients/relatives and better patient flow.

## P227 ETHNIC DIVERSITY IN REFERRAL AND ACCEPTANCE PATTERNS TO A UK TERTIARY COMPLEX VENTILATION AND SPECIALIST WEANING UNIT

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10.1136/thorax-2025-BTSabstracts.408

**Background** Healthcare disparities across ethnic groups are well-documented, yet little is known about ethnic diversity in tertiary care ventilation referrals and acceptance decisions. As gatekeepers to specialised interventions, tertiary units play a crucial role in healthcare equity. We characterised the ethnicity of patients referred to and accepted by a tertiary specialist weaning and complex ventilation unit to identify potential healthcare inequities.

**Methods** This was a retrospective analysis of consecutive referrals (October 2020 – February 2024) made via a secure online interface to a tertiary complex ventilation and specialist weaning unit. Referrals were categorised as level 2 (non-invasive ventilation review/domiciliary setup), level 3 (tracheostomy

**Abstract P227 Table 1** Patient demographics, referral patterns and acceptance rates by ethnicity compared to census data. Data presented as median (IQR), n (%) and % for census data

| Ethnicity | Age (years) (Median (IQR) | Sex (n Male (%)) | Total referred (N (%)) | Total accepted (N (%)) | English Census 2021 (%) | London Census 2021 (%) |
|---|---|---|---|---|---|---|
| Asian | 63 (45 - 70) | 24 (60.0) | 40 (5.3) | 25 (5.1) | 9.6 | 20.7 |
| Black | 58 (48 - 70) | 30 (41.1) | 73 (9.8) | 46 (9.4) | 4.2 | 13.5 |
| White | 66 (52 - 74) | 299 (51.9) | 576 (77.0) | 384 (78.5) | 81.0 | 53.8 |
| Mixed or Multiple | 67 (47- 67) | 3 (37.5) | 8 (1.1) | 5 (1.0) | 3.9 | 5.7 |
| Other | 62 (52 - 72) | 30 (58.8) | 51 (6.8) | 29 (5.9) | 2.2 | 6.3 |

ventilation review/domiciliary setup) or remote non-invasive ventilation setup. Patient ethnicity was categorised by Office for National Statistics Standards, recorded by referring clinicians using 19 categories (classification 20b) and then grouped into 5 grouped classifications (classification 6a). Chi-square tests were used to assess statistical significance of categorical variables.

**Results** Of 748 referrals received (Age: 63 (±20), 52% male), 489 (65%) were accepted. Referrals originated from 59 hospitals across 24 of England's 42 Integrated Care Boards (ICBs) (57.1%), with the majority (95%) from the South East region, including 43.9% from London.

Ethnic distribution of referred patients was: White (576, 77%), Black (73, 9.8%), Asian (40, 5.3%), Mixed (8, 1.1%) and Other (51, 6.8%). Diagnostic patterns varied significantly by ethnicity ($p<0.05$). A higher proportion of White patients had COPD (22.7%) compared to Asian (7.5%), Black (8.2%), Mixed (0%), and Other (13.7%) groups. Conversely, a higher proportion of Asian patients had a diagnosis of neuromuscular conditions (57.5%) than in White (35.9%), Black (28.8%), Mixed (25%), and Other (39.2%) groups.

Despite these diagnostic differences, acceptance rates were equitable across all ethnic groups, with patients accepted in the same proportions as they were referred ($p=0.66$).

**Conclusion** This single-centre study demonstrates equity of acceptance to a tertiary ventilation service across ethnic groups despite diagnostic case-mix differences between ethnic groups. Continued monitoring and clinical pathway review are essential to ensure sustained equitable acceptance and a clear understanding of the ethnic diversity of referrals for patients requiring ventilatory support.

## P228 EVALUATING CAPILLARY BLOOD GAS MONITORING AS A PATIENT-CENTERED ALTERNATIVE TO ARTERIAL BLOOD GAS IN ACUTE NIV PATIENTS – A PILOT PROJECT IN THE RESPIRATORY SUPPORT UNIT AT A DISTRICT GENERAL HOSPITAL

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10.1136/thorax-2025-BTSabstracts.409

**Introduction** Blood gas monitoring is crucial in patients on acute non-invasive ventilation (NIV) for acute hypercapnic respiratory failure (AHRF). Performing an arterial blood gas (ABG) sampling demands an experienced doctor or a specialist nurse. Capillary blood gas (CBG) can be done by any nursing staff or physiologist with appropriate training, with similar efficacy to monitor the pH and PaCO2.

**Aim** To evaluate the feasibility, patient experience, and effectiveness of CBG monitoring compared to ABG in AHRF patients requiring acute NIV.

**Method** Data were collected in two phases. Initially, three months retrospective data collected from June to August 2024 of patients who received acute NIV for AHRF in the Respiratory Support Unit (RSU) to understand the median duration on acute NIV and the average number of ABG done during inpatient. Then, as a pilot project, three advanced nurse practitioners were trained to do CBG for blood gas monitoring after 24 hours of acute NIV initiation. We collected one-month prospective data on CBG-guided NIV support and patients' pain score using the visual analogue scale with a questionnaire.

**Result** Total 60 patients' data were analysed retrospectively. The mean age was 67 years. Affected males and females were 33 and 27, respectively. Median duration on acute NIV was 4 days. The average number of ABG done during admission was 12 per patient. In the prospective study, we provided a questionnaire to total15 patients to get the patient reported experience. 80% (13) patients described ABG as 'severe to worst possible pain' and 100% (15) patient described CBG as 'no pain to mild pain'. All patients preferred CBG as a method for blood gas sampling. No observable difference identified in CBG-guided NIV support in contrast to ABG.

**Conclusion** CBG monitoring aligns with BTS NIV and RSU standards by meeting the core recommendations for frequent blood gas assessment and continuous patient monitoring and review. As a less invasive alternative to ABG, CBG offers a patient-centred approach that minimizes discomfort. Our gradual introduction of CBG monitoring into the unit has led to significant improvements in patient comfort without compromising the clinical rigor required for effective NIV management.

## P229 SINGLE-CENTRE EXPERIENCE OF ACUTE NIV OUTCOME FOR ACUTE HYPERCAPNIC RESPIRATORY FAILURE

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10.1136/thorax-2025-BTSabstracts.410

**Introduction** Non-invasive ventilation (NIV) is a key intervention for acute hypercapnic respiratory failure (AHRF), with outcomes influenced by multiple factors like timing, care location, and underlying diagnosis.

**Aim** To assess factors associated with outcomes of acute NIV in a district general hospital.

**Methods** We retrospectively analysed data from patients who received acute NIV for AHRF between June and August 2024. Key variables included timing of NIV initiation, arterial blood gas results, setting, and diagnosis.

**Results** 63 patients (mean age 67 years, 54% male) were included. Inpatient mortality was 16% (n=10). NIV was started in the emergency department in 68% and in the respiratory unit in 32%, with higher mortality in the latter (20% vs 14%). Median door-to-mask time was 3 hours. Early NIV (within 1 hour of decision) occurred in 73% and was associated with higher weaning rates (46%). Patients with pH <7.26 (46%) had higher mortality (21%). COPD was the most common diagnosis (63%). Patients with pulmonary oedema had the highest mortality (33%). The OSA/OHS group had a high rate of discharge with domiciliary NIV.

**Conclusion** Our centre's mortality rate was lower than that reported in the BTS 2017 audit. However, delayed initiation and lower initial pH were linked to poorer outcomes. Improving early recognition and reducing door-to-mask times may enhance patient outcomes.

**Abstract P229 Table 1** NIV Outcome by clinical variables

| Variables | Number of patients | Inpatient mortality | Weaned from NIV | Domiciliary NIV on discharge |
|---|---|---|---|---|
| Total patients | 63 | 16% (10) | 41% (26*) | 43% (27**) |
| COPD exacerbation | 63% (40) | 15% (6) | 45% (18*) | 38% (15) |
| OSA | 14% (9^) | 0% | 11% (1) | 89% (8) |
| OHS | 14% (9^) | 11% (1) | 11% (1) | 78% (7) |
| Pulmonary oedema/ HF | 10% (6^^) | 33% (2) | 33% (2) | 33% (2) |
| Initial pH <7.26 | 46% (29) | 21% (6) | 52% (15) | 24% (7) |
| Door-to-mask time >2hours | 67% (29) | 3% (1) | 41% (12) | 55% (16) |
| Decision to mask time >1 hour | 27% (17) | 12% (2) | 29% (5) | 59% (10) |

*1 patient died, **8 patient already on domiciliary NIV previous to admission, ^6 patients had OSA and OHS, ^^1 patients had OSA, and 1 patient had OHS.

## REFERENCE

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## P230 IS THERE ANY VALUE? A 1 YEAR PREVALENCE STUDY OF PATIENTS REQUIRING VENITLATORY SUPPORT AT A SPINAL CORD INJURY CENTRE

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10.1136/thorax-2025-BTSabstracts.411

Spinal Cord Injury (SCI) is often complicated by ventilation. SCI centres may fully/partially wean 50–80% of ventilated patients. This charts the ventilatory, weaning, communication and swallow outcomes over 1 year in a UK SCI centre. Do we offer value to patients and the NHS?

**Method** Consecutive adult patients requiring domicilary set-up or ventilation or tracheostomy status changes were included. Legacy patients, CPAP/oxygenation only requirements, those admitted for other reasons were excluded.

**Results** 23 patients were included. Most were male, aged 50+ and had traumatic, motor complete cervical injuries. 25% had no tracheostomy cuff deflation pre-admission, mean 85 days. 56% had partial deflation (range 45 mins to 12 hours per day). Once admitted, all had plain tubes inserted in 1–7 days.

16 required SLT investigations. 2 commenced normal oral intake before admission. 56% recieved FEES and 1 recieved videofleuroscopy (VF). After admission, 88% had FEES, VF, or both. Silent aspiration improved, but laryngeal pathologies and dysphagia persisted. 11/14 progressed to taking normal oral intake with targeted treatment and chest care, likely resulting in cost savings compared to enteral intake. All demonstrated improvements.

Time to admission averaged 71 days. 6 patients who subsequently weaned spent 92 days average in another facility costing approximately £173,052 per patient. They weaned in 16 days average and decannulated in 35 days at a cost of £1,533.61 per patient in the SCI centre. Tracheostomised only patients were decannulated in 20 days average, leading to significant cost savings.

17/21 had signs of infection at admission and 5 had failed weans or decannulations. All of these showed improvements in ventilation status except 1 apnoeic high tetraplegia patient. Mental health conditions were most common comorbidity.

A total of 12/23 were weaned and decannulated.1 remained fully ventilator dependent. 2 died during their admission.

**Conclusions** SCI rehabilitation produced advantages for ventilated patient outcomes and health economics. There was increased prospect of ventilator weaning and decannulation, rapid return to vocalisation and progression of oral intake without negative chest consequences due specialised and integrated team working. There are long waits for beds. Improvement could further enhance cost and clinical outcomes.

## P231 HIGH FLOW NASAL OXYGEN: IS IT SUITABLE FOR EVERYONE WITH RESPIRATORY FAILURE?

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10.1136/thorax-2025-BTSabstracts.412

**Introduction** High flow nasal oxygen (HFNO) is often deployed in patients with severe respiratory failure. It can deliver oxygen at rates of up to 60 litres per minute. It also provides some positive pressure which prevents the alveoli from collapsing. Studies have shown that it is comfortable, effective and may reduce the need for invasive ventilation in some patients.[1] We are looking to identify patients that are most likely to benefit from this intervention.

**Methods** We retrospectively reviewed the case notes of patients requiring HFNO in our hospital over a 3 month period. Data such as demographics, diagnosis, co-morbidities, care escalation status and outcomes including death were collected.

**Results** 63 patients were identified. Median age was 77 years. 34 (54.0%) had an underlying respiratory co-morbidity. 59

(93.7%) received HFNO in a ward (level 1) environment, the remainder in intensive care (level 2–3). Overall mortality was 39.7%.

Aspiration pneumonia (22.2%) and community-acquired pneumonia (20.6%) were the most common diagnoses. Mortality rates were lower in patients with aspiration pneumonia (35.7%), community acquired pneumonia (30.8%), viral pneumonia (0%) and exacerbation of chronic airways disease (20.0%).

Higher mortality rates were observed in patients with hospital acquired pneumonia (75.0%), interstitial lung disease (50.0%) and pulmonary embolism (100%).

24 patients (38.1%) were for escalation to ICU level care. Of these, 20 (83.3%) survived to discharge without requiring ICU admission and 3 (12.5%) were admitted to ICU but did not require invasive ventilation. 1 (4.2%) patient declined invasive ventilation and died.

There were no complications associated with starting HFNO in a ward setting.

**Conclusion** High flow nasal oxygen is an effective, non-invasive method of providing respiratory support in patients with severe respiratory failure. It should be considered in all patients with community acquired pneumonia, aspiration pneumonia and exacerbation of COPD. It is likely to prevent the need for intubation and ventilation in these patients. It seems less effective in conditions such as hospital acquired pneumonia, interstitial lung disease and pulmonary embolism.

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## P232 IMPACT OF ACETAZOLAMIDE IN REFRACTORY HYPERCAPNIC RESPIRATORY FAILURE: EXPERIENCE FROM A RESPIRATORY SUPPORT UNIT

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10.1136/thorax-2025-BTSabstracts.413

**Introduction** Hypercapnic respiratory failure (HRF) is associated with prolonged inpatient hospital stay and has impact on morbidity and mortality. Most patients with HRF can be controlled by non-invasive ventilation (NIV) however, some patients continue to have refractory HRF despite optimization of NIV. It is thought that acetazolamide can reduce metabolic alkalosis which then may reduce hypercapnia,[1] however there are limited literature with mixed outcomes about benefit of acetazolamide in these cases.[2] A retrospective review of patients prescribed acetazolamide for this purpose was performed.

**Method** Patients with HRF who were in Respiratory Support Unit (RSU) from June 2019 to February 2025 were screened and patients who received acetazolamide for 3 days were identified.

**Results** 16 patients were identified, 11 patients were excluded due to reasons of unable to find exact prescription, already on acetazolamide for other reasons and non-HRF. 5 patients (4 Females) were identified for analysis. Reasons for HRF and RSU admission were Obesity hypoventilation syndrome/ Obstructive Sleep Apnoea and Exacerbation of COPD. Mean NIV days till acetazolamide trial was 14 (8–21). There was a mean reduction of serum bicarbonate by 13.3 mmol/L and CO2 by 2 kPa post-trial. Mean RSU-need bed days was 19. The duration of hospital admission was confounded by other factors like social care needs. All 5 patients were discharged back to their homes. 3 patients are still alive and 2 has passed away at different times during further admissions with different reasons.

**Conclusion** There was improvement of serum bicarbonate and CO2 levels for refractory HRF patients with good NIV compliance who received acetazolamide. No evidence of harm demonstrated. If instituted sooner, it might reduce RSU bed stay. Further randomized studies are needed to see its impact on RSU stay and mortality.

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## P233 COMPARISON OF NIV OUTCOMES IN ACUTE HYPERCAPNIC RESPIRATORY FAILURE: DOES INITIATION PH AFFECT THE OUTCOME?

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10.1136/thorax-2025-BTSabstracts.414

**Background** The Joint British Thoracic Society/Intensive Care Society (BTS/ICS) guidelines[1] recommend prompt initiation of

**Abstract P232 Table 1**

| | Age | BMI (kg/m²) | pH | Bicarb (mmol/L) | CO2 (kPa) | pH | Bicarb (mmol/L) | CO2 (kPa) | Duration of RSU need (days) | Total Hospital Admission duration (days) | Days of NIV till Acetazolamide trial (days) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Pre Acetazolamide | | | Day 3 Post Acetazolamide | | | | | |
| Range (Min-Max) | 55-75 | 15.5 - 44 | 7.37-7.51 | 42 - 48 | 7.56-11.30 | 7.27-7.44 | 27 - 38.5 | 6.9 - 10.2 | 6 - 32 | 14 - 88 | 8 - 21 |
| Mean | 68 | 27.1 | 7.42 | 43.2 | 10.2 | 7.36 | 29.9 | 8.2 | 19 | 44 | 14 |
| Median | 70 | 23 | 7.41 | 42 | 10.9 | 7.36 | 28 | 7.8 | 21 | 42 | 15 |

**Abstract P233 Table 1**

| Variables | pH 7-7.15 | pH7.16-7.25 | pH7.26-7.35 |
|---|---|---|---|
| Total number of patients | 15 | 42 | 42 |
| Median Age | 70 | 74.5 | 72 |
| Male: Female | 6:9 | 20:22 | 17:25 |
| Discharged following Index admission | 8 | 26 | 34 |
| Died in hospital during Index admission | 7 | 16 | 8 |
| Weaned off NIV during index admission | 4 | 10 | 8 |
| Discharged with Domiciliary NIV (Newly started NIV) | 2 | 3 | 4 |
| Pre-admission NIV patients (Discharged on NIV) | 1 | 13 | 22 |
| Discharged from ITU | 1 | 3 | 2 |
| Outcome - No. of patients alive at 3 months | 6/8 | 23/26 | 28/34 |
| Outcome - Dead in 3 months | 2/8 | 3/26 | 6/34 |
| Re-admission in 3 months | 3/6 | 4/23 | 5/28 |
| Outcome - No. of patients alive at 12 months | 6/6 | 21/23 | 23/28 |
| Outcome - Dead in 12 months | 0/6 | 2/23 | 5/28 |
| Re-admission in 12 months | 1/6 | 4/21 | 5/23 |
| Discharge rate | 15 | 42 | 42 |
| | 8 | 26 | 34 |
| | 53.33% | 61.91% | 80.95% |
| Odds Ratio | 0.27 | 0.38 | |
| 95% CI | [0.08, 0.96] | [0.14, 1.03] | |
| p value | 0.04 | 0.06 | |
| Time Specific Mortalities | | | |
| In-Hospital Mortality | 15 | 42 | 42 |
| | 7 | 16 | 8 |
| | 46.67% | 38.10% | 19.05% |
| Odds Ratio | 3.72 | 2.62 | |
| 95% CI | [1.04, 13.29] | [0.97, 7.04] | |
| p value | 0.04 | 0.06 | |
| 3-month mortality | | | |
| No. of patients discharged | 8 | 26 | 34 |
| No. of patients dead within 3 months | 2 | 3 | 6 |
| Mortality rate at 3 months | 25.00% | 11.54% | 17.65% |
| Odds Ratio | 1.56 | 0.61 | |
| 95% CI | [0.25, 9.67] | [0.14, 2.71] | |
| p value | 0.64 | 0.51 | |
| 3-Months to 12-months mortality | | | |
| No. of patients alive at 3 months | 6 | 23 | 28 |
| No. of patients dead from 3 to 12 months | 0 | 2 | 5 |
| Mortality rate at 12 months | 0.00% | 8.70% | 17.86% |
| Odds Ratio | 0.33 | 0.44 | |
| 95% CI | [0.02, 6.75] | [0.08, 2.50] | |
| p value | 0.47 | 0.35 | |
| Cumulative Mortalities | | | |
| (In-hospital mortality + Mortality following discharge) | | | |
| 3-month mortality (cumulative) | 15 | 42 | 42 |
| | 9 | 19 | 14 |
| | 60.00% | 45.24% | 33.33% |
| Odds Ratio | 3 | 1.65 | |
| 95% CI | [0.89, 10.12] | [0.68, 4.00] | |
| p value | 0.08 | 0.27 | |
| 12-months (cumulative) | 15 | 42 | 42 |
| | 9 | 21 | 19 |
| | 60.00% | 50.00% | 45.24% |
| Odds Ratio | 1.82 | 1.21 | |
| 95% CI | [0.55, 6.02] | [0.51, 2.85] | |
| p value | 0.33 | 0.66 | |
| Weaned off NIV during index admission | 15 | 42 | 42 |
| | 4 | 10 | 8 |
| | 26.67% | 23.81% | 19.05% |
| Re-Admission | | | |
| Re-admission in 3 months | 6 | 23 | 28 |
| | 3 | 4 | 5 |
| | 50.00% | 17.39% | 17.86% |
| Odds Ratio | 4.6 | 0.97 | |
| 95% CI | [0.71, 29.84] | [0.23, 4.12] | |
| p value | 0.11 | 0.97 | |
| Re-admission in 12 months | 6 | 21 | 21 |
| | 1 | 4 | 5 |
| | 16.67% | 19.05% | 23.81% |
| Odds Ratio | 0.64 | 0.75 | |
| 95% CI | [0.06, 6.85] | [0.17, 3.31] | |
| p value | 0.71 | 0.71 | |

acute non-invasive ventilation (NIV) for patients presenting with acute hypercapnic respiratory failure (AHRF) that does not respond to initial optimal medical management. We analyzed the short, medium, and long-term survival and in-hospital mortality for patients treated with acute NIV for AHRF with different levels of acidosis on initiation. Current evidence suggests poor outcome in patients with lower pH (especially pH of <7.25).[2]

**Aim** To evaluate the clinical outcomes in patients initiated on NIV for AHRF and whether the level of acidosis predicts short, medium, and long-term outcomes.

**Methods** Retrospective analysis of data from electronic case records of a tertiary hospital between January and December 2024. Patients were categorized into 3 groups according to their pH (Group 1 = pH 7 to 7.15, Group 2 = 7.16 to 7.25 and Group 3 = 7.26 to 7.35).

**Results** 99 patients were ventilated for various etiologies. 15 patients presented with severe acidosis (group 1) with a median age of 70 years. In-hospital mortality was 46.7% (n = 7). Group 2 comprised 42 patients with a median age of 74.5 years. In-hospital mortality was 38.1% (n= 16). Group 3 included 42 patients with a median age of 72 years. The inpatient mortality was lowest at 19% (n = 8). Mortality was higher in Group 1 at 3 months and in Group 3 at 12 months. The mortality rate in Group 2 was lower than both groups at 3 months and lower than Group 3 at 12 months.

**Conclusion** Admission pH predicts in-hospital mortality in patients presenting with AHRF. The 3-month and 12-month mortality is comparable, but the p-value is likely skewed due to a low sample size. There were no significant differences in the overall long-term success and re-admission rates among the various groups. In conclusion, although lower pH can predict poor outcome, a trial of NIV in AHRF can be considered in various pH categories if the patient population is carefully selected and the care provided on a dedicated ventilation unit.

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## P234 PREDICTING IN-HOSPITAL MORTALITY IN A REAL-WORLD POPULATION OF WARD-BASED NON-INVASIVE VENTILATION (NIV) IN ACUTE EXACERBATIONS OF COPD


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**Background** Accurate tools for patient stratification by likely outcome are needed to support complex decision-making and improve acute non-invasive ventilation (NIV) delivery. We assessed the potential of an emerging NIVO score tool to predict in-hospital mortality, to aid its validation in a real-world

UK hospital population of ward-based NIV for acute exacerbations of COPD (AECOPD).

**Methods** This was a retrospective observational cohort analysis on all consecutive admissions of patients with AECOPD, managed with NIV for acute hypercapnic respiratory failure (AHRF) at an acute teaching hospital. Clinical parameters were collected within our ongoing quality improvement project. Patients were split according to survival or in-hospital mortality. NIV failure and hospital mortality were modelled using logistic regression. NIV failure was defined as NIV withdrawal or intubation requirement due to clinical worsening clinical with NIV therapy.

**Results** There were 249 unique patients and 314 AECOPD admissions with ward-based NIV. Across all admissions, NIV failure rate was 32.6%, in-hospital mortality 23.3%, and 1-year mortality 46.6. NIVO score was significantly associated with both NIV failure and in-hospital mortality, odds ratio (95% Confidence intervals) of 1.32 (1.14–1.53, $p<0.001$) and 1.54 (1.29–1.85, $p<0.001$), respectively. Older age was also associated with in-hospital mortality 1.07 (1.03–1.11, $p<0.001$) and current smoking associated with reduced risk, 0.41, (0.20–0.79, $p=0.008$).

**Conclusions** We demonstrate the prospect of a novel NIVO tool to predict in-hospital mortality in a real-world population of ward-based NIV COPD patients. We show its potential utility to aid decision making and NIV delivery in new clinical pathways to meet the burden of COPD exacerbations.

## P235 MULTIDISCIPLINARY NON-INVASIVE VENTILATION OUTPATIENT SERVICE FOR PATIENTS WITH MOTOR NEURONE DISEASE AT A TERTIARY COMPLEX VENTILATION CENTRE

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**Background** Chronic respiratory failure (CRF) due to Motor Neurone Disease (MND) presents a significant clinical challenge due to variability in speed of the progressive respiratory muscle weakness. Non-invasive ventilation (NIV) may improve survival and quality of life but requires timely specialised support to initiate effectively. Traditional inpatient pathways can delay treatment. An outpatient NIV set-up service (OPSUS) has been established to provide structured assessment and ventilation initiation for patients with respiratory failure including a cohort of patients with MND. It provides joint multidisciplinary assessments by a Respiratory Physician, Clinical Nurse Specialist, and Senior Physiotherapists. Comprehensive evaluation incorporates arterial blood gases, overnight oximetry (prior to appointment) and measures of respiratory muscle function (FVC, SNIP). NIV initiation is protocolised, with outpatient titration using pressure control or pressure support modes based on individual patient need. Patients receive a

**Abstract P235 Table 1** Characteristics of referrals at baseline for all MND patients assessed and those set up on NIV with follow up data where available. Data are presented as n (%), median (IQR) or mean (SD). Oximetry data prior to appointment available for 24 patients (44%)

| | Baseline entire cohort (n =55) | Baseline not set up on NIV (n=18) | Baseline set up on NIV (n=37) | Follow up (n=35) |
|---|---|---|---|---|
| Age (years) | 70 (64-76) | 72 (67-77) | 69 (63 – 73) | |
| Gender | 57% female | 61% female | 54% female | |
| BMI | 25.6 (20.6-28.0) | 25.3 (22.2-26.6) | 25.3 (22.2 – 26.6) | |
| Epworth sleepiness scale | 7.48 (±5.19) | 5.66 (±4.14) | 7.55 (±5.47) | |
| FEV1 (L) | 1.47 (±0.71) | 1.55 (±0.95) | 1.42 (±0.57) | |
| FVC (L) | 1.88 (±0.91) | 1.91 (±1.14) | 1.87 (±0.81) | |
| SNIP (cmH2O) | 35 (±20) | 40.6 (±22.5) | 32.5 (±17.7) | |
| SpO2 (%) | 96.6 (±2.1) | 96.9 (±1.43 | 96.4 (±2.29) | |
| Oximetry ODI | 9.02 (±8.47) | 13.00 (±12.90) | 7.52 (±5.79) | |
| Oximetry mean SpO2 | 93.3 (91.4 – 94.8) | 93.3 (92.8 – 95.9) | 93.5 (91.1-94.8) | |
| Oximetry time below 90% | 5.01 (1.23 – 14.79) | 1.04 (0.67 -12.6) | 6.85 (1.68 – 15.84) | |
| $CO_2$ (kPa) | 5.5 (±0.7) | 4.9 (±5) | 5.7 (±0.7) | 5.3 (±0.5) |
| Adherence at last follow-up (hours) | | | | 6.5 (1.5-8) |

phone call 2 weeks after set up to troubleshoot and aid adherence.

**Methods** We sought to characterise the MND cohort seen via the OPSUS pathway at a tertiary ventilation unit between January 2024 and May 2025. Clinical characteristics, and adherence outcomes were assessed.

**Results** Between January 2024 to May 2025, 55 MND patients attended OPSUS; (Age mean 69 ± 7.7 years), 57% female). Median time from referral to first appointment was 27 days (IQR 9 – 51 days). 37 (67%) were set-up on NIV (16 pressure control mode, 21 pressure support mode). Baseline characteristics are provided in table 1. 13 of NIV patients (36%) were electively admitted within 3 months of set-up, 4 (11%) for respiratory review and 9 (24%) for PEG assessment. At last follow up (median time to follow up 111 days (IQR 33–209)), 24/35 (68.5%) patients had adherence at >4 hours per 24 hours. The service enabled prompt identification of nocturnal hypoventilation and hypercapnia, facilitating timely intervention through optimised mask fitting, patient education and adherence strategies.

**Conclusion** Outpatient NIV set-up constitutes an effective, multidisciplinary outpatient model for managing chronic respiratory failure in MND patients, achieving high levels of NIV adherence while reducing the demand for inpatient initiation.

## P236 ACUTE HYPERCAPNIC RESPIRATORY FAILURE: ASSESSING THE INCIDENCE IN THE EMERGENCY DEPARTMENT

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10.1136/thorax-2025-BTSabstracts.417

**Background** Blood gas (BG) analysis is frequently used to detect Acute Hypercapnic Respiratory Failure (AHRF), a serious disease. National audits on AHRF primarily focus on patients referred to respiratory medicine or critical care, leaving the true incidence of AHRF in the Emergency Department (ED) largely underreported. This audit was aiming to analyse BG tests conducted in ED to determine the occurrence of AHRF in critically ill adult patients.

**Methods** A single-center audit was conducted in December 2024, involving 500 consecutive adult patients in the Majors & Resuscitation areas of ED, 187 of whom underwent BG testing. Demographic, clinical, and BG data were reviewed to determine the incidence of AHRF based on the British Thoracic Society (BTS) and Intensive Care Society (ICS) criteria.

**Results** Among 500 patients, 187 (37.4%) had blood gas (BG) test done, and respiratory acidosis was detected in 25 (13%) patients. 84% of all BG samples were venous, and 60% were taken within 60 minutes of ED admission. The mean age of these patients was 68.8 years, and a mean body mass index was 26.5 kg/m$^2$. The patients with respiratory acidosis had a greater referral rate to general medicine compared to others (68% vs. 64%, $p < 0.05$), treatment time was significantly shorter for these patients (91 minutes vs. 137 minutes, $p<0.05$). Four patients in the respiratory acidosis group needed ventilatory support; two of them were intubated and two of them got non-invasive ventilation (NIV).

**Conclusion** AHRF was promptly identified and referred for treatment in the ED, with ventilatory support initiated in a timely manner despite department pressures. Incidentally, our hospital's BG testing rate was considerably higher than international benchmarks, likely due to the frequent use of venous samples. Further multicentre surveys are needed to better understand the true incidence of AHRF in ED.

## P237 CLOSING THE GAP IN ACUTE NIV: A SCALABLE NURSE-LED APPROACH TO OPTIMISING TIME-CRITICAL CARE

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**Introduction** The 2023 BTS RSU audit demonstrated a lower all-cause mortality for hospitals with an RSU and enhanced staffing ratios. At Sherwood Forest Hospitals (SHFT), an acute NIV nurse-led outreach and retrieval service, based on an RSU with enhanced staffing and central monitoring of observations, has been in place for over 8 years. However, data demonstrated our median gas and door-to-mask times were 03:29hrs, and 04:15hrs respectively, with delays noted in performing and interpretation of blood gases.

**Methods** We created a digital tracker focusing on gas and door-to-mask times as our improvement measures. Following process mapping and root cause analysis, we redesigned our referrals process and developed an early assessment model for all acute patients. Acute NIV nurses perform an early clinical review at the initial identification of possible NIV patients, optimising patient care, ensuring timely blood gases and earlier ringfencing of RSU beds. Adopting and supplementing the BTS care bundle,[2] we added 3 additional locally conceived standards to match our new delivery model and created a checklist to ensure consistency and completeness. We audited the utilisation of our standards before and after the intervention.

**Results** Audit data demonstrated that pre-intervention, only 10% of sampled patients from 2024 received all elements of the care bundle, improving to 74%. Delivery of acute NIV improved with a reduction in both gas and door-to-mask times of 43 minutes and 1 hour 09 minutes, respectively.

**Conclusion** Early results demonstrate an improvement in treatment times. The development of the locally enhanced care bundle enabled the reduction in variation of care and empowered the team to utilise their skills, experience and knowledge. We believe the continuous development of our acute NIV nurse model to now include earlier optimisation and identification of patients is sustainable, cost-effective and applicable to similar services.

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# New insights from the fundamental sciences

## P238 SPATIAL TRANSCRIPTOMIC PROFILING AT SINGLE-CELL RESOLUTION REVEALS LUNG RESPONSES TO STERILE INJURY

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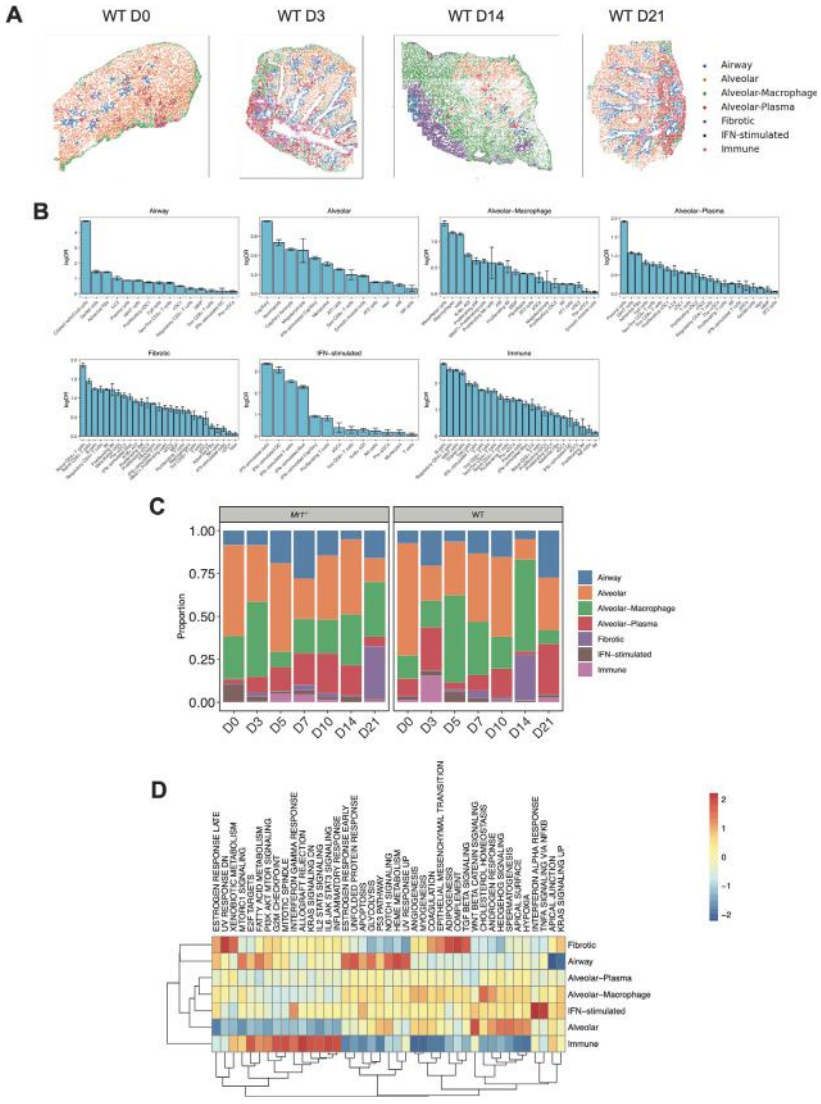
10.1136/thorax-2025-BTSabstracts.419

**Introduction** Mucosal-associated invariant T (MAIT) cells protect against lung injury, but their spatial interactions and mechanisms of action remain poorly defined.

**Methods** Using single-cell resolution spatial transcriptomics (MERSCOPE), we profiled over 1.8 million cells from 14 lung tissue sections collected across seven time points from bleomycin-treated wild-type and MAIT cell-deficient *Mr1*$^{-/-}$ mice.

**Results** We identified seven conserved cellular niches, with alveolar niches reducing, immune and fibrotic niches expanding after injury. Fibrotic niches featured fibroblast-macrophage co-localisation, with interstitial macrophages (IM) expressing both pro-fibrotic and reparative signatures. B cell follicles emerged in immune niches, showing *CXCL13* expression in B cells, goblet cells and CD4$^{+}$ T cells. cDC1, regulatory T cells, and γδ T cells were spatially enriched near MAIT cells. cDC1s close to MAIT cells upregulated activation markers such as *Xcr1*, *Flt3*, and *Ccr2*, while nearby T cells expressed higher levels of supression marker *Ctla4*. Spatial analysis (using *Spacia*) revealed that MAIT cell-derived *Ctla4*, *Pdcd1*, and *Tgfb1* were associated with reduced expression of pro-inflammatory genes (*Il6*, *Ccl2*, *Ereg*) in surrounding immune cells, particularly cDC1 and naïve CD4$^{+}$ T cells. Re-analysis of human pulmonary fibrosis spatial transcriptomic data confirmed the presence of *CXCL13*$^{+}$ B cell follicles and an increased MAIT cell gene signature in fibrotic lungs, mirroring murine findings.

**Conclusions** Together, this work provides the first spatially resolved map of MAIT cell interactions in lung injury and highlight their role in modulating local immune responses.




**Abstract P238 Figure 1** Figure Multi-slice cellular niche identification. **(A)** spatial distribution of MENDER-annotated cellular niche across different samples. Different colours indicate different cellular niches. **(B)** enriched cell types within each cellular niche. **(C)** the proportion different cellular niche in each sample. **(D)** gene signature score of hallmark gene sets in each cellular niche

## P239 ENDOTHELIAL HIF2A SIGNALLING MODULATES HYPOXIA DRIVEN MYELOID CELL MIGRATION INTO THE LUNG

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**Introduction and Objectives** Acute exposure of pulmonary vascular endothelial cells to pathological hypoxia initiates the secretion of pro-inflammatory mediators and increases the expression of leukocyte adhesion molecules that promotes the recruitment of myeloid-lineage cells into the lung. The infiltration of activated neutrophils into hypoxic regions of the lung has been reported in a multitude of pulmonary diseases, including Pulmonary Hypertension, resulting in the release cytotoxic proteins and proteases that cause local tissue damage and vascular remodelling. However, a complete understanding of the hypoxic signalling pathway that initiates the migration of myeloid-lineage cells into the lung remains unclear.

**Methods and Results** A time-course (1–14 days) of wild-type mice exposed to acute hypoxia (10% oxygen) confirmed the migration of myeloid cells into pulmonary tissues. Lung single cell multi-colour FACs analysis identified a significant increase in both neutrophil and monocyte derived cell populations following 3-days hypoxia, with a significant increase in the neutrophil sub-population $CXCR4^+$/$VEGFR1^+$ (pro-angiogenic). Blood neutrophil counts fluctuated with a trend towards decreasing CXCR4 expression following hypoxia exposure, suggesting an increase in neutrophil mobilisation from the bone marrow. Here, we show the normal increase in myeloid lineage cells following hypoxia exposure was absent in mice with pulmonary endothelial Hypoxia Inducible Factor (HIF)2a deletion; the total cell count for lung neutrophils and monocytes was not significantly different from wild-type animals breathing room air. This is a HIF2a specific event because loss of pulmonary endothelial HIF1a did not affect hypoxia-induced cell migration. RNA-seq analysis of human pulmonary artery endothelial cells (hPAEC) exposed to hypoxia identified a significant increase in target genes associated with leukocyte adhesion, and trans-endothelial migration. The co-culture of PT2385 (HIF2a inhibitor) with hPAEC exposed to hypoxia significantly reduced the expression of leukocyte adhesion molecules and chemokines.

**Conclusion** Our data support pre-clinical studies in pulmonary hypertension which identified that drug intervention with a HIF2a specific inhibitor significantly reduced the migration of both monocytes and neutrophils into the lung. Further investigations are required to fully elucidate the role of HIF2a in leukocyte/endothelial transmigration and understand the potential beneficial effect of inhibiting HIF2a signalling in other neutrophil-driven pulmonary diseases.

## P240 PULMONARY EXTRACELLULAR VESICLE MICRORNAS INDUCE ALVEOLAR MACROPHAGE DYSFUNCTION IN ARDS

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10.1136/thorax-2025-BTSabstracts.421

Acute Respiratory Distress Syndrome (ARDS) is a hyper-inflammatory lung disorder which develops following sepsis. Alveolar macrophages (AMs) from ARDS patients have impaired efferocytosis compared to control ventilated sepsis patients (PMID:34112730). This defect is replicated when healthy AMs are treated with ARDS patient broncho-alveolar lavage fluid (BAL) (PMID:34660643). Impaired AM efferocytosis contributes to alveolar inflammation via secondary necrosis of apoptotic neutrophils. Lung injury models implicate EVs in ARDS pathogenesis. We hypothesised BAL EVs drive AM dysfunction in ARDS.

EVs were isolated from ARDS and post-operative control patient BAL via ultracentrifugation. AMs were isolated from lung tissue resections of non-smoking patients. AMs were treated with BAL EVs 24 hours prior to assessment of efferocytosis, Seahorse XF analysis, ICC and Western Blot. ARDS BAL EV treatment impaired AM efferocytosis (fc 0.541, $p=0.0029$). AMs treated with ARDS BAL EVs had increased basal OCR ($p=0.0391$), maximal respiration ($p=0.0273$) & ATP driven by mitochondrial respiration ($p=0.0039$). Markers of mitophagy (LAMP1 & LC3B) were decreased in AMs after ARDS EV treatment ($p=0.0156$ & $p=0.0312$). Thus, accumulation of damaged mitochondria may drive the dysfunction observed.

Transcriptomics reveal enrichment of 8 microRNAs in ARDS BAL EVs. EV miR-15a & miR-26b inversely correlate with ex vivo AM efferocytosis. Transfection of healthy AMs with miR-15a & miR-26b mimics impairs efferocytosis ($p=0.0115$) and decreased LC3B & LAMP-1 expression, replicating the effects of EV treatment.

Targeting EV microRNA cargo may attenuate AM dysfunction & inflammation, offering a therapeutic strategy for ARDS patients.

## P241 DIFFERENTIAL METHYLATION ANALYSIS IN FIBROTIC VERSUS NON-FIBROTIC SARCOIDOSIS

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10.1136/thorax-2025-BTSabstracts.422

**Introduction and Aims** Sarcoidosis is a heterogeneous complex disease involving genetic susceptibility and environmental factors. Epigenetic modifications are emerging as important in regulating immune and fibrotic processes through gene expression in lung disease. We aim to assess how differential methylation can provide mechanistic insights into sarcoidosis-specific disease-regulating pathways.

**Methods** 79 patients with sarcoidosis underwent methylation analysis of 100ng of genomic DNA extracted from peripheral white blood cells. Analysis was carried out using the methylkit package in R. Clinical data, including demographics, pulmonary function tests, CXR stage, disease duration and need for treatment were analysed. We stratified patients by the presence or absence of fibrosis on plain CXR at the time of blood draw. Results were cross-referenced to stratification by presence or absence of inflammation (ESR >20, CRP >7). A further targeted approach evaluated involvement of the mTOR pathway.

**Results** The mean age of the cohort was 58.4 (SD 11.6), 66% were male, 54% required treatment, and 41% were fibrotic (n 32). Screening results for > 5 log2 fold or < -5 Log2 fold

**Abstract P241 Table 1** Differential Methylation within CPG Island in Fibrotic Sarcoid Patients

| | Gene ID | p value | q value | Meth.diff | Gene Function & Known Pathways |
|---|---|---|---|---|---|
| Up | DUSP22 | 8.41E-07 | 0.000116 | 7.090 | Involved in immune regulation and cancer. MAPK signalling pathway, JNK pathway. |
| | MCC | 1.60E-05 | 0.000775 | 6.820 | Tumour suppressor gene, negative regulator of Wnt signalling. |
| | HOXD3 | 9.38E-09 | 6.05E-06 | 6.287 | Homeobox protein involved in early embryonic development. Developmental pathways, morphogenesis |
| | INSYN1 | 1.08E-12 | 8.23E-09 | 6.248 | Inhibitory synapse-associated protein, may regulate neurotransmission. |
| | APC2 | 8.88E-05 | 0.002586 | 5.305 | Adenomatous polyposis coli protein 2, involved in Wnt signalling |
| | PTPRN2 | 0.000323 | 0.006159 | 5.005 | Protein tyrosine phosphatase involved in insulin secretion and neuronal function. |
| | | | | | |
| Down | MAPK11 | 4.14E-05 | 0.001483 | -5.417 | MAP kinase family -involved in cellular responses to stress and inflammation |
| | NBL1 | 0.000154 | 0.003655 | -5.575 | Inhibits BMP signalling. Tumour suppressor and plays a role in cell cycle |
| | GRM1 | 7.94E-10 | 1.28E-06 | -5.578 | Involved in Glutamate signalling |
| | NKX2-1-AS1 | 2.92E-09 | 2.57E-06 | -6.159 | Promotes lymphangiogensis, tumour progression and angiogenesis. |
| | TXNRD1 | 0.000156 | 0.003669 | -8.459 | Regulates inflammation and oxidative stress in Small airway Epithelium, can activate the Akt/mTOR signalling pathway |
| | UNCX | 5.64E-11 | 1.09E-07 | -8.775 | Associated with impaired cell proliferation and differentiation |
| | ZGPAT | 3.67E-05 | 0.00135 | -9.362 | Zinc finger protein act as a transcriptional repressor in EGFR signalling pathway. |
| | HDAC4 | 2.05E-09 | 2.21E-06 | -12.158 | Histones influence the expression of genes involved in extracellular matrix production and tissue remodelling |

change identified 9 targets with increased methylation, 29 with decreased methylation, and an additional 20 novel transcripts. Table 1 presents results focusing on methylation of genes found within the CPG island (promoter region), more likely to affect transcription and therefore gene expression. Initial results indicate involvement of Wnt, JN and MAP Kinase pathways. Increased expression of HDAC4 was found in fibrotic patients; these are involved in extracellular matrix production and tissue remodelling processes and may represent a therapeutic target. Targeted evaluation of mTOR revealed significant differential methylation of Forkhead box protein 01 (FOX01), a transcription factor, acting both as a regulator and being regulated by inflammatory pathways, in the inflammatory group (q 0.0126).

**Conclusions** Methylation profiles may have potential applications as biomarkers for disease identification and prognostication, and personalised treatment strategies. However, our results require validation and further work is required to understand the biological significance of the identified profiles, including their impact on gene expression and cellular processes, as well as clinical outcomes.

## P242 EPITHELIAL CELL EXTRUSION PROMOTES THE LOSS OF KRT5 WITH IMPLICATIONS FOR PULMONARY FIBROSIS

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**Background** IPF is characterised by an increase in aberrant basaloid epithelial cells which have high levels of Keratin 17 (KRT17) and low levels of Keratin 5 (KRT5) although their source is still unclear. One potential source is airway basal cells which express high levels of both KRT17 and KRT5. Cell extrusion is a process whereby individual epithelial cells are expelled from contiguous epithelium, whilst barrier integrity and tissue architecture is maintained. I hypothesise that extrusion of epithelial cells from small airways in lung leads to loss of KRT5 and is a critical first step in fibrogenesis and generation of aberrant basaloid cells.

**Methods** KRT5 and KRT17 gene expression was evaluated in ihBECs (immortalised human bronchial epithelial cells) using real time qPCR in monolayer, extruded and replated extruded cells. ihBECs were seeded at high density to induce overcrowding to promote cell extrusion. Once 95% confluent, extruded cells were collected from supernatant and replated for sub-culture. The monolayer cells remained in culture for another three days until collected along with extruded cells for RNA extraction. KRT5 and KRT17 protein in monolayer and extruded ihBECs was evaluated by flow cytometry (FACS Canto BD) and flow imaging cytometry (Imagestream MKII) using fluorescent antibodies ((Anti-Cytokeratin 5 antibody [EP1601Y] and Anti-Cytokeratin 17 antibody [EP1623]); 2ug/mL).

**Results** There was a five-fold decrease in KRT5 and two-fold increase in KRT17 mRNA in extruded ihBEC (eihBEC) cells compared ihBEC in monolayer (mihBEC). KRT5 protein was downregulated by 50% in eihBECs compared with mihBEC in both flow cytometry and flow imaging cytometry analyses. Flow cytometry of KRT17 in eihBEC compared with miHBEC showed upregulation by 35%. The gene expression pattern in extruded cells showing loss of KRT5 and increase of KRT17 was maintained upon sub-culture of replated eihBEC cells compared with mihBEC.

**Conclusion** Extrusion of ihBECs leads to loss of KRT5 and increased KRT17 consistent with the phenotype of aberrant basaloid cells seen in IPF. We propose that extrusion of small airway epithelial cells may be a critical first step in development of aberrant basaloid cells.

## P243 CHARACTERISATION OF SRC-FAMILY KINASE INHIBITORS IN LUNG FIBROBLASTS SHOWS DISTINCT SIGNALLING COMPLEXES REQUIRED FOR MATRIX SYNTHESIS AND DIFFERENTIATION

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Idiopathic pulmonary fibrosis (IPF) is the most rapidly progressive fibrotic interstitial lung disease, with a median survival of 3–5 years post-diagnosis, characterized by excessive deposition of extra-cellular matrix (ECM). Current evidence implicates differentiated myofibroblasts, responding to aberrant TGF-β1 signalling, as primary effector cells. Classically identified by α-smooth muscle actin (α-SMA) expression,

emerging evidence suggests a more complex model of distinct pathological cell sub-types. The Src-family kinases (SFKs) have been implicated in several profibrotic cellular processes, such as myofibroblast differentiation, epithelial-mesenchymal transition, and pulmonary inflammation. To define their role in fibrogenic processes in primary human lung fibroblasts (pHLFs), we conducted functional assays with small molecule inhibitors and found divergent impacts on ECM synthesis and myofibroblast differentiation. While less selective compounds showed inhibition of both processes, a novel, more selective and conformation-dependent inhibitor (NXP900) showed only inhibition of α-SMA expression, suggesting independent mechanisms for these processes. Cell-free kinome scan assays and cell-based phosphoproteomics revealed that more selective SFK inhibition does not affect TGF-β1-mediated mTOR signalling required for ECM synthesis, but does modulate cell signalling complexes associated with cytoskeletal organisation, focal adhesion, and cell-cell/substrate junctions. These results suggest specific roles for the SFKs in myofibroblast differentiation with implications for therapeutically targeting functional heterogeneity in pathological cell sub-populations in IPF.

## P244 THE RNA BINDING PROTEIN INSULIN GROWTH FACTOR 2 BINDING PROTEIN 3 (IGF2BP3) IS A NOVEL BIOMARKER OF T2 HIGH ASTHMA AND INHIBITOR OF IL-13/IL-4 SIGNALLING IN HUMAN BRONCHIAL EPITHELIUM

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10.1136/thorax-2025-BTSabstracts.425

**Background** Interleukin 13 (IL-13) and IL-4 are archetypal cytokines that drive type 2 immune responses, which are dysregulated in a large proportion of patients with asthma. The regulation of IL-13 and IL-4 signalling has focused on their downstream events (e.g. phosphorylation of STAT6 upon receptor binding) leading to transcription of type 2 mediators. Further regulators of this fundamental pathway remain poorly understood. Likewise, molecular markers of type 2 disease remain scarce.

**Methods** We used RNA immunoprecipitation, siRNA and shRNA depletion in cell lines and primary bronchial epithelium, as well as RT-qPCR and protein analyses (western blotting, MSD). We also employed Frac-seq (subcellular fractionation and RNA-sequencing) to compare steady (transcription) vs polyribosome-bound (translation) mRNA expression in response to IL-13 in an IGF2BP3-dependent manner. We explored several datasets of 'type 2 high' disease cohorts.

**Results** Using RNA immunoprecipitation, we detected IGF2BP3 directly bound to the mRNAs encoding for IL4R and IL13Ra1. Depletion of IGF2BP3 led to an increase of IL-4Ra and IL-13Ra1 cell surface expression, without affecting *IL4R* or *IL13RA1* mRNA levels, methylation or stability in human bronchial epithelial BEAS-2B cells. Concomitantly, IGF2BP3 depletion led to increased IL-13-dependent STAT6 phosphorylation and activity as determined by a STAT6-reporter and increased expression of IL-13-dependent eotaxins *CCL26*, *CCL24* and *CCL11* mRNAs, as well as the classically associated Th2 marker *GATA3*. IGF2BP3 regulation of eotaxin and *GATA3* expression was recapitulated in primary airway cells. Frac-seq demonstrated that IL-13 triggers different transcriptional and post-transcriptional responses, with IGF2BP3 levels mainly modulating steady mRNA levels. We also found IGF2BP3 upregulated in multiple cohorts of atopic and type 2 high asthmatic patients.

**Conclusions** We show for the first time that an RNA binding protein modulates IL-13/IL-4 signalling and offer a novel approach for suppressing IL-13-dependent signalling in bronchial epithelium. IGF2BP3 is increased in type 2 high disease, where it may be part of a feedback loop to halt type 2 inflammation and serve as a novel biomarker.

## P245 THE REV-ERBA CLOCK AGONIST (SR9009) PROTECTS AGAINST HOUSE DUST MITE INDUCED T2 ASTHMA

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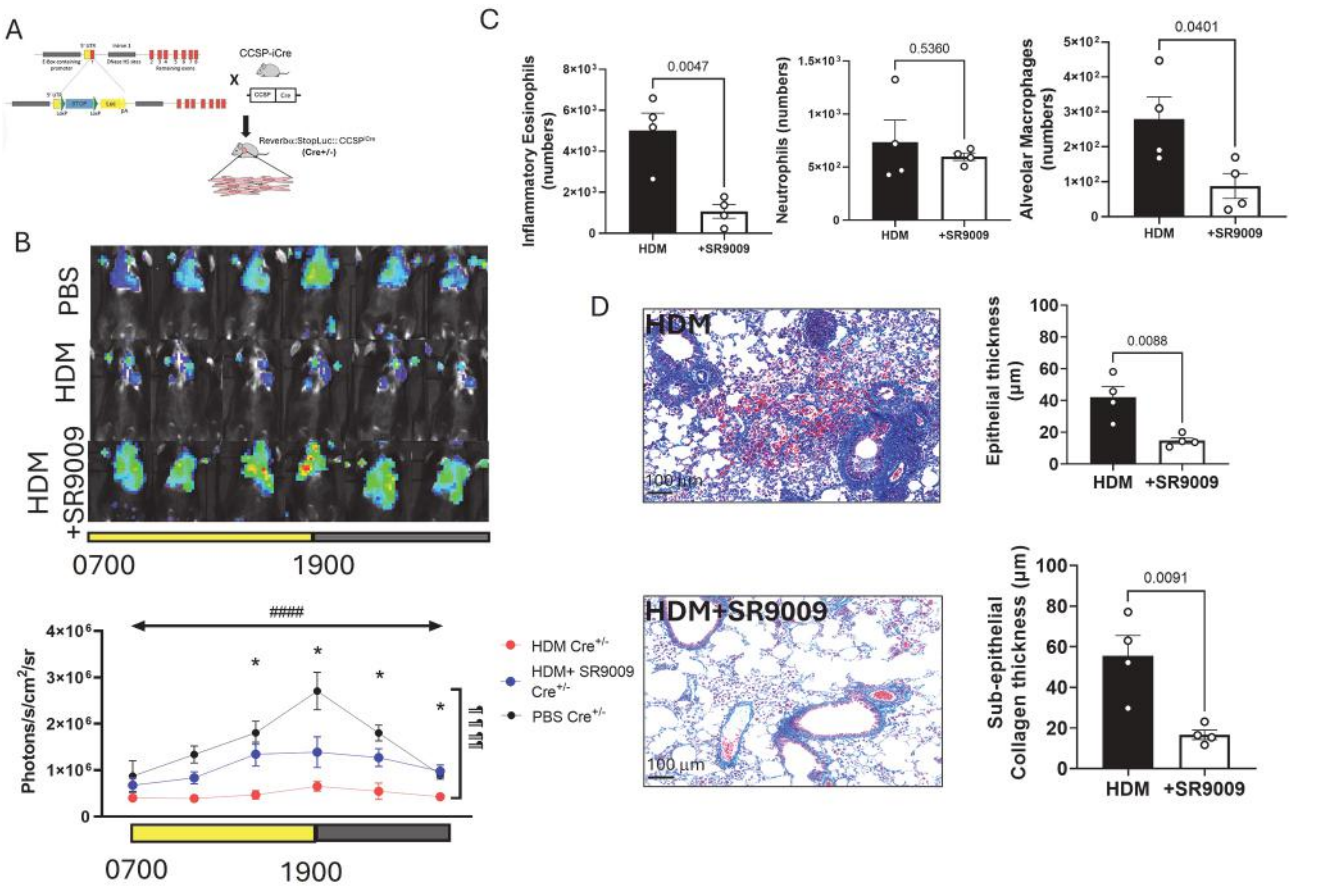
10.1136/thorax-2025-BTSabstracts.426

Asthma is a highly rhythmic disease with increased airway sputum eosinophilia and decline in lung function (FEV1) observed early morning.[1] We have previously shown that the time of day of allergen challenge in mice governs the amplitude of the resulting airway hyperresponsiveness (AHR). Furthermore, the clock gene, *NR1D1*, regulates time of day changes in AHR.[2] Since the molecular clock in the airway epithelium is also the pacemaker for the lung, we are interested to discover how allergen challenge regulates rhythmic downstream inflammatory processes in asthma (AHR). However, whether targeting this molecular clock with its agonist (SR9009) can attenuate House Dust Mite (HDM)-induced inflammation and pathophysiological changes remain unknow.

We employed a conditional *NR1D1* reporter BAC construct with luciferase under the *NR1D1* gene's regulatory control, crossed with CCSP-iCre mice to generate NR1D1-STOPluc$^{+/-}$ (Cre$^{+/-}$) animals (figure 1A). Mice were exposed to HDM allergen intranasally three times weekly for five weeks while a subset received the NR1D1/Rev-erba agonist SR9009 (i.p, at ZT6/1 PM) every day in the last 2 weeks of the HDM administration while another subset received vehicle alone. These mice were subjected to bioluminescence imaging performed every four hours post-luciferin injection, utilizing an IVIS imaging system. Precision-cut lung slices were also prepared, imaged continuously over 72 hours to assess bioluminescence in response to allergen exposure.

We propose that the Rev-erba agonist SR9009, will protect against HDM induced airway inflammation and associated pathology

The results indicate a diurnal pattern in *NR1D1* expression in NR1D1-STOPluc mice, peaking at ZT12 in non-allergic animals and showing significant reductions in HDM-treated mice ($p<0.001$ at ZT12, figure 1B). Administration of SR9009 to HDM challenged animals recovered NR1D1 expression and concomitantly reduced HDM-associated eosinophilia (figure 1C). SR9009 also reduced pathological changes induced by HDM such as epithelial thickness and subepithelial collagen (figure 1D,E). The result demonstrates




**Abstract P245 Figure 1** The protective effect of Rev-erba agonist SR9009 against allergic airways disease. **(A)** schematic diagram of the generation of NR1D1/Rev-erba StopLuc in CCSP- expressing airway epithelial cells hence, NR1D1-STOPluc:CCSP-iCre±(Cre±) reporter mice. **(B)** bioluminescence imaging of NR1D1- STOPluc:CCSP-iCre±mice demonstrating dampened expression of NR1D1 with HDM allergen challenge in comparison to vehicle PBS but prevented with SR9009. **(C)** SR9009 prevents eosinophilia and influx of inflammatory cells in the lungs. **(D)** SR9009 prevented airway remodelling associated with T2 asthma

the therapeutic potential of SR9009 and its protective role in T2 asthma.

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## P246 GLYCINE ATTENUATES AIRWAY HYPERRESPONSIVENESS ASSOCIATED WITH T2 ASTHMA

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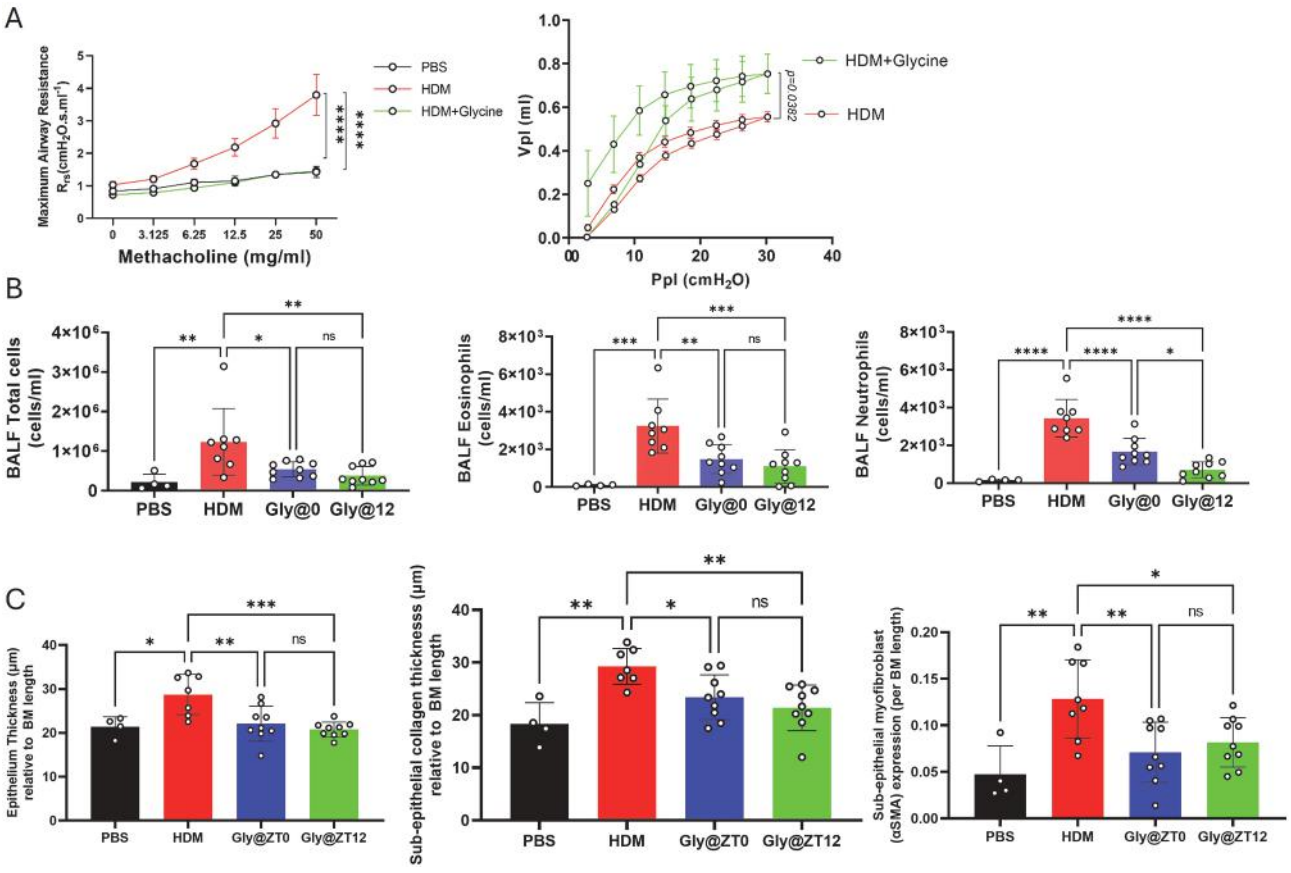
10.1136/thorax-2025-BTSabstracts.427

Asthma is a chronic respiratory disease exhibiting circadian rhythmicity and causing significant morbidity and mortality worldwide.[1] Current inhaled corticosteroids (ICS) maintenance therapies may not adequately control asthma or treat all subtypes. In addition, side-effects such as adrenal suppression and frequent patient non-compliance highlights the need for alternative treatments (NICE guidelines). Glycine, a non-essential amino acid approved for human use, plays a critical role in cellular metabolism and is significantly reduced in asthma patients.[2] We are interested to discover how glycine mediates its anti-inflammatory and immunomodulatory properties and thereby regulating airway hyperresponsiveness (AHR) associated with T2 asthma.

Female C57Bl/6 mice (8 weeks old) were housed under a controlled environment on a 12h light/12h dark cycle with free access to water and lab chow. These mice were subjected to a 5-week regiment of thrice-weekly intranasal house dust mite (HDM) challenges. On a subset of mice after three weeks, Glycine (100 mM in saline) was nebulized thrice weekly on alternate days to HDM administration for 2 weeks synergistically with HDM administration. Another subset of mice underwent saline (vehicle) nebulization on alternate days to HDM administration for a further 2 weeks. After 5 weeks, pulmonary function was measured terminally using Flexivent (SciReq). Lungs were lavaged post-mortem and comprehensive inflammatory cell analysis and lung histopathology were conducted. A lobe of the lung was used for performing single-nulcei RNA sequencing.

We propose that administration of inhaled glycine to the physiological allergen House Dust Mite (HDM) exposed mice for prolonged periods may result in alleviation of airway hyperresponsiveness

Preliminary data shows glycine significantly reduced airway hyperresponsiveness (AHR) as demonstrated by the reduction in airway resistance and improvement in the Pressure-Volume (PV) loops (figure 1A). Furthermore, glycine reduced airway eosinophilia and immune cellular influx (figure 1B).




**Abstract P246 Figure 1** Immunomodulatory role of glycine in T2 asthma. **(A)** Glycine reduced airway hyperresponsiveness and improved Pressure-Volume (PV) curves in comparison to the allergen challenged (HDM) animals at both 6AM (Gly@0) and 6PM (Gly@12) but did not show any time of administration effect. **(B)** Post-mortem lavaged lungs from mice were subjected to FACS analysis which demonstrates total immune cell, eosinophil and neutrophil influx at both 6AM (Gly@0) and 6PM (Gly@12) administration time. **(C)** Glycine administration reduced airway epithelial thickening, the presence of sub-epithelial collagen and expression/transition of fibroblasts to myofibroblasts (indicative of αSMA expression) at both 6AM (Gly@0) and 6PM (Gly@12) administration time

Histopathological analysis revealed glycine targeted airway remodelling by reducing airway epithelial thickness, sub-epithelial collagen and myofibroblast transition. Analysis of single nuclei RNA sequencing to identify key molecular pathway alterations is in progress.

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## P247 GSDMB MEDIATES EPITHELIAL TO MESENCHYMAL TRANSITION (EMT) IN HUMAN AIRWAY EPITHELIAL CELLS

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10.1136/thorax-2025-BTSabstracts.428

**Background** The polymorphisms of human *GSDMB* gene on chromosome 17 have been found to associate asthma or asthma traits worldwide. GSDMB is expressed exclusively in the epithelium in a highly tissue-specific manner and regulates inflammatory response through pryoptosis. We investigate whether GSDMB regulates epithelial to mesenchymal transition (EMT) in human airway epithelial cells.

**Methods** *GSDMB* knockout cells were generated by applying CRISPR/Cas9 techniques. GSDMB overexpression plasmids were used to generate overexpression model in HBEC3-KT cells. Cells were stimulated by HMGB-1 (100ng/ml) over 24-hours and α-SMA expression levels were examined by fluorescent staining and western blotting. Cells were also treated with TGF-β1 (5ng/ml) for 1, 3, and 5 days. Transcript levels of α-SMA, e-cadherin, tenascin C, fibronectin, collagen I and vimentin were quantified by qPCR. IL-6 and IL-8 levels in the supernatant were measured using ELISA.

**Results** Upon HMGB-1 stimulation, the expression of α-SMA in HBEC3-KT wild type cells increased, indicating a successful stimulation of HMGB-1 treatment in the cells. *GSDMB* knockout epithelial cells had minimal expression of α-SMA without treatment and remained unchanged post HMGB-1 treatment. Western blotting confirmed the different expression levels of α-SMA in the wildtype cells and *GSDMB* knockout cells. In contrast, α-SMA expression levels of *GSDMB* overexpression cells increased after HMGB-1 treatment. With TGF-β1 treatment, α-SMA levels were significantly decreased at 5 days in *GSDMB* knockout cells. Collagen I was significantly increased in *GSDMB* knockout cells. In *GSDMB* overexpressed cells, tenascin and fibronectin decreased after 5 days. IL-8 levels were significantly increased in GSDMB knockout cells after TGF-β1 stimulation.

**Conclusions** GSDMB regulates EMT in human epithelial cells by regulating collagen I, tenascin, and fibronectin levels.

Further research will focus on the mechanism of GSDMB in EMT and the results will bring new insight into the new targets for treatments of fibrotic lung diseases.

## P248 ORMDL3 INFLUENCES FERROPTOSIS THROUGH HMOX1 IN HUMAN BRONCHIAL EPITHELIAL CELLS

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10.1136/thorax-2025-BTSabstracts.429

**Background** The polymorphisms of *ORMDL3* on human chromosome strongly associate childhood asthma and asthma exacerbations. It has multiple functions in human epithelial cells, regulating de novo sphingolipid synthesis, protein folding, glycolysis, and the expression of the human rhinovirus (HRV) receptor intercellular adhesion molecule 1 (ICAM-1), which increases HRV infection. This is known to be a major exacerbator of asthma. We previously found ORMDL3 influencing the expression of the ferroptosis gene *HMOX1*. In this research, we investigate HMOX1 levels in *ORMDL3* knockout and overexpression cells.
**Methods** *ORMDL3* knockout cells were generated by applying CRISPR/Cas9 techniques in HBEC3-KT epithelial cells. *ORMDL3* knockdown cells were established by applying ORMDL3 siRNAs in A549 epithelial cells. *ORMDL3* overexpression plasmids were used to generate an overexpression model in HBEC3-KT cells. Cells were stimulated with LPS/IL-13 or erastin to induce ferroptosis in epithelial cells, ferroptosis-associated genes *GPX4*, *SLC7A11*, *HMOX1*, *DHODH*, *ACSL4*, and *ACSL3* were quantified by qPCR, and GPX4, SLC7A11, and HMOX1 were analysed by immunofluorescent staining and Western blotting. IL-6 and IL-8 levels from cell supernatants were examined with ELISA.
**Results** In *ORMDL3* knockdown A549 cells, the transcript levels of *HMOX1* were lower, whereas in *ORMDL3* overexpression HBEC3-KT cells, the transcript levels of *HMOX1* were higher. *ORMDL3* overexpression showed lower *GPX4* and *SCL7A11* levels in immunofluorescent staining after inducing by LPS/IL-13. The ongoing work included examining transcript levels of HMOX1 in *ORMDL3* knockout cells and GPX4, SCL7A11, HMOX1 levels in *ORMDL3* overexpression cells and ORMDL3 knockout cells treated with erastin.
**Conclusions** ORMDL3 regulates ferroptosis in human epithelial cells. It regulates ferroptosis gene HMOX1 levels. Our results provide new insights into ferroptosis in asthma and may identify potential targets for childhood asthma and asthma exacerbations.

## P249 KNOCKOUT OF ORMDL3 RENDERS AIRWAY EPITHELIAL CELL RESISTANCE TO HUMAN RHINOVIRUS INFECTION

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10.1136/thorax-2025-BTSabstracts.430

**Background** Orosomucoid-like protein 3 (ORMDL3) has important role to regulate human rhino virus infection in airway epithelial cells as one of mechanisms of asthma exacerbations. In this report we created *ORMDL3* knockout (KO) epithelial cell lines and tested the cells response to different stimulators and human rhinovirus 16 infections.
**Methods** CRISPR/Cas9 techniques were applied to create *ORMDL3* KOs in HEBC3-KT epithelial cells. Two targeted *ORMDL3* KO clones were established. *ORMDL3* KO cells were treated with different stimulators and human rhinovirus 16 (RV-A16). IL-6 and IL-8 levels, viral load, cytopathic effects were evaluated. *ORMDL3* overexpression cells transfected by plasmids were also created to test with the stimulators.
**Results** Two *ORMDL3* KO clones with 1 base-pair deletion and 5 base-pair deletion in exon 2 were selected. Both deletions caused the frame shifts of ORMDL3, and the translations end prematurely. The knockout cells released more IL-6 and IL-8 after IL-1β, TNFα, Poly I:C and LPS/CD14 stimulations. With RV-A16 infection, *ORMDL3* KO cells had less viral load, less cytopathic effects with higher level of IL-6 and IL-8 release. The effect on RV-16 infection could last 7 days in *ORMDL3* KO cells after viral infection. Intercellular adhesion molecule 1 (ICAM-1) levels in KO cells were significantly increased with or without RV-16 infections.
**Conclusions** We established *ORMDL3* KO epithelial cells, which have shown to response to different stimuli. *ORMDL3* KO cells significantly reduce RV-A16 infection. The *ORMDL3* KO cells could provide a useful platform for investigating the mechanisms of human rhino virus infection in the mechanisms of asthma exacerbations.

## P250 GSDMB REGULATES HUMAN RHINOVIRUS REPLICATION AND INFLAMMATION IN AIRWAY EPITHELIAL CELLS

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10.1136/thorax-2025-BTSabstracts.431

**Background** Polymorphisms on human chromosome 17q21 confer the major genetic susceptibility to childhood-onset asthma. The loci consist of *ORMDL3* and *GSDMB* genes. The risk alleles positively correlate with both genes' expression. ORMDL3 has been extensively studies and showed it regulates many pathways and human rhinovirus (HRV) infection in cell inflammation, but GSDMB's role in human rhinovirus infection and asthma exacerbation are incompletely understood.
**Methods** *GSDMB* knockouts in human airway epithelial HBEC3-KT cells were created by CRISPR/Cas9 techniques. The knockout cells were challenged by IL-1β, TNF-α, Poly IC, LPS and human rhinovirus HRV16. Cytokine levels of IL-6 and IL-8 from supernatants were examined by ELISA, viral loading was checked by resazurin assay and viral copies were measured by qPCR. *GSDMB* overexpression epithelial cells were established in HBEC3-KT cells. Cytokine levels in time course treatment were analysed by ANOVA. Transcript abundances were analysed by qPCR.
**Results** Human rhino virus infected to the *GSDMB* knockout cells to show reduced inflammatory response and viral loading. *GSDMB* overexpression cells augmented inflammatory response after HRV infection. Poly I:C, a synthetic double-stranded RNA (dsRNA) of virus, stimulated knockout cells showed dramatically reduction of levels of IL-6 and IL-8. Knockout cells showed differently to release IL-6 and IL-8 with different stimulators.

**Conclusions** Our results show GSDMB has important role in epithelial cells in viral infection. Consistent with its substantial genetic influence on childhood asthma. Actions on GSDMB provide a potential mechanism for the locus to confer susceptibility to HRV-induced asthma.

## P251 INVESTIGATION OF MYELOID-DERIVED SUPPRESSOR CELLS AS A BIOMARKER FOR THE DIFFERENTIAL DIAGNOSIS OF LUNG DISEASES

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10.1136/thorax-2025-BTSabstracts.432

**Introduction and Objectives** Myeloid-derived suppressor cells (MDSCs) are immature cells with immunosuppressive properties, divided into polymorphonuclear (PMN-) and monocytic (M-) subpopulations. Based on a limited number of disease-specific studies, they appear to be involved in a wide spectrum of lung diseases. We aimed to standardise a simple measuring protocol of MDSCs in the lungs and the systemic circulation and to comparatively investigate their differences among distinct lung diseases.

**Methods** In 16 patients with lung diseases, MDSCs were measured in peripheral blood (PB) and bronchoalveolar lavage (BAL) via flow cytometry and immunophenotypically defined as CD45+CD3−CD19−CD20−CD56−CD16−HLA-DR−CD33+CD11b+CD15+Lox-1+ PMN-MDSCs and CD45+CD3−CD19−CD20−CD56−CD16−HLA-DR−CD33+CD11b+CD14+ M-MDSCs, as shown in figure 1a. Total MDSCs were calculated as the sum of the two subpopulations. Their suppressive character was confirmed via T-cell suppression assay. BAL cell populations were also defined. Further clinical and laboratory data were collected, including diagnosis, stage of disease, findings from microbiological, cytological, and histological examinations, disease course, and end outcomes. The data were analysed with the non-parametric Wilcoxon signed ranks test for paired samples and the Kruskal-Wallis test for comparison between multiple groups.

**Results** Our study population included 4 patients with bronchiectasis, 6 patients with lung cancer, and 6 patients with interstitial lung diseases (ILD). MDSCs from all study subgroups showed normal capacity to suppress T cells. All MDSC subsets, including total MDSCs, M-MDSCs, and PMN-MDSCs, were statistically significantly increased in BAL compared to PB, as depicted in figure 1b, indicating the lung accumulation of these cells (p-value: <0.05 in all disease groups). Interestingly, total MDSCs in the BAL were statistically significantly differentiated between the three patient groups, as depicted in figure 1c, with the group of patients with ILD presenting the higher counts (p-value: 0.001).

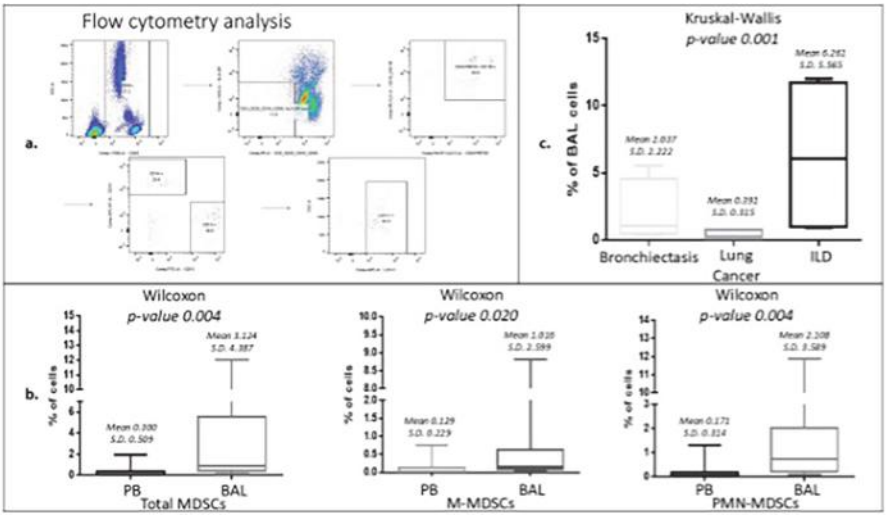
**Conclusions** Our preliminary data show for the first time that MDSCs could potentially be used as differential diagnostic biomarkers in lung diseases. Additional data are needed to develop disease- and sample-specific reference values of MDSCs and further investigate their quantitative alterations in different entities.

## P252 INVOLVEMENT OF MYELOID-DERIVED SUPPRESSOR CELLS IN ALLERGIC AIRWAY DISEASES: PRELIMINARY RESULTS OF A SYSTEMATIC REVIEW OF THE LITERATURE

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**Introduction and Objectives** Myeloid-derived suppressor cells (MDSCs) are immature cells of the myeloid lineage with immunosuppressive properties, divided into polymorphonuclear and monocytic subpopulations, and are commonly involved in chronic inflammatory diseases. We aimed to systematically review the literature to assess the quantitative and qualitative alterations of MDSCs and their involvement in allergic airway diseases, i.e. asthma and allergic rhinitis.




**Abstract P251 Figure 1**

**Methods** The systematic review protocol was registered in PROSPERO, and the PRISMA guidelines for systematic reviews were followed. The databases MEDLINE via PubMed, Embase, and Scopus were systematically searched for original research studies involving the measurement of human MDSCs in allergic airway diseases by the end of 2024. A narrative synthesis of the extracted data from the included studies was performed.

**Results** Among a total of 429 records screened, 8 studies were considered eligible to be included in the review. The studies were run from 2011 to 2021 and cumulatively included 212 patients with asthma and 90 patients with allergic rhinitis. Healthy controls (cumulatively 203) were included in 7 studies, while 4 studies also included patients with other respiratory diseases for comparison, i.e. chronic obstructive pulmonary disease (35 patients), pneumonia (65 patients), and respiratory viral infections (27 patients). Out of 6 studies about asthma, MDSC counts were found either increased or increasing after allergen challenge in bronchoalveolar lavage (2 studies) and in peripheral blood mononuclear cells (3 studies), while the monocytic MDSC subpopulation was found downregulated in isolated white blood cells in only 1 study. The 2 studies about allergic rhinitis used diverse experimental set-ups and were not comparable.

**Conclusions** Increased counts of MDSCs are mostly found in asthma, pointing towards the role of these immunomodulatory cells in its pathogenesis, despite the heterogeneity of the methodology among the different studies. Further research and harmonisation of the markers and techniques for the identification of MDSCs is needed, as they may be used as novel biomarkers and therapeutic targets in these disease entities.

# Updates in lung disease diagnostics (and PFTs)

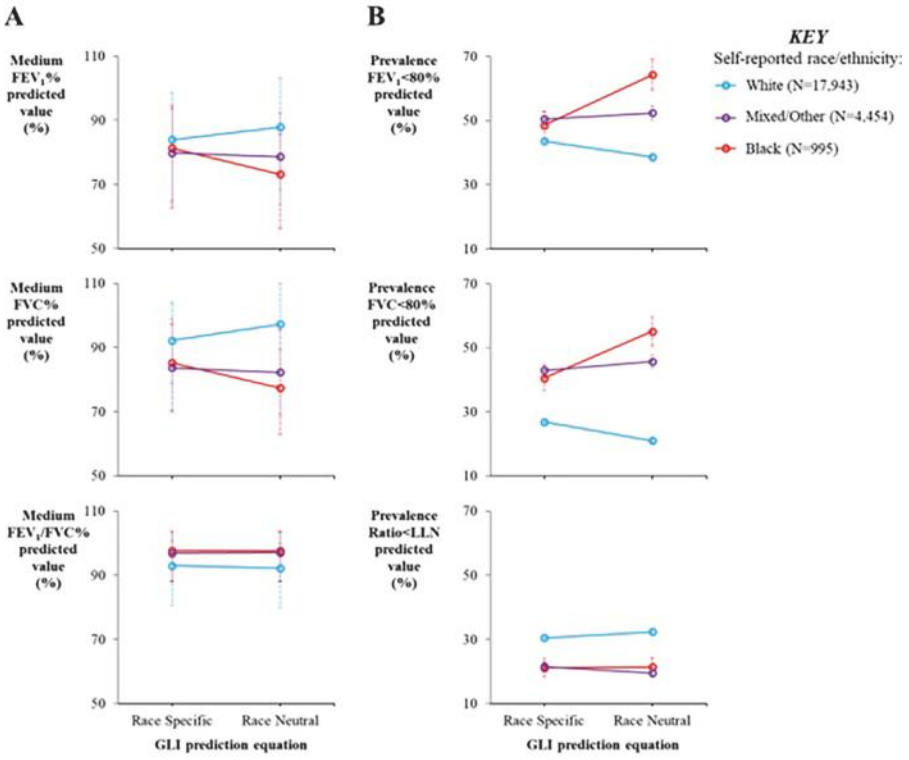
## P253 IMPACT OF APPLYING GLOBAL LUNG INITIATIVE 2022 RACE NEUTRAL EQUATIONS TO UK TERTIARY CARE LUNG FUNCTION DATA

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**Introduction** Respiratory care decision-making is influenced by spirometry value interpretation, guided by lung function equations. Global Lung Initiative (GLI)-2012 equations include race-specific adjustments, while the newer GLI-2022 adopt a race-neutral approach. Population based studies suggest this change impacts self-identified race/ethnic groups differently. To determine how this may impact clinical decision making, we applied both equations to clinical data routinely collected in a UK tertiary care setting.

**Method** We included spirometry laboratory data, performed clinically at two UK tertiary hospitals across 10 years among patients aged 18–95 years, excluding cases without self-defined




**Abstract P253 Figure 1** Impact of applying race-specific versus race-neutral lung function predictions upon: **(A)** Percent of predicted values (left panel shows median with interquartile range); **(B)** Classification of FEV, or FVC values as below 80% predicted, or the FEV,/FVC ratio as below the lower limit of normal (LLN) (right panel shows percentages with 95% confidence interval). Values are stratified according to self-reported race/ethnicity as White, Black, or Mixed/Other FEV,=Forced Expiratory volume in one second. FVC=Forced Vital Capacity. GLI=Global Lung Function Initiative

race/ethnicity or height. We included only the first time-point when complete data were provided. We compared $FEV_1$, FVC, and $FEV_1$/FVC percent of predicted values calculated using GLI-2012 versus GLI-2022 equations applied to the same dataset. We considered the impact upon prevalence of 'impaired' $FEV_1$ (<80% predicted), FVC (<80% predicted), and $FEV_1$/FVC.

**Results** Only 186 (<1%) from 23,578 patients self-identified as North/South-East Asian, so we excluded these from subsequent analyses. Among the remaining 23,392 (50% female; mean age 57 years), 17,943 (77%), 4,454 (19%), and 995 (4%) self-identified as white, mixed/other, and black respectively. Moving from race specific to neutral equations led $FEV_1$% and FVC% of predicted values to increase among the white-identifying sample and decrease among the black-identifying sample (figure 1). This led the prevalence of both $FEV_1$ and FVC impairment (<80% predicted) to decrease and increase among these respective groups. For example, median $FEV_1$% predicted increased from 84% to 88% among the white-identifying group and decreased from 81% to 73% among the black-identifying group. This led to the prevalence of impaired $FEV_1$ (<80% predicted) to decrease from 44% to 39% among the white-identifying group but increase from 49% to 64% among the black-identifying group. Median $FEV_1$/FVC% of predicted and prevalence of an impaired $FEV_1$/FVC.

**Conclusion** Applying GLI-2012 versus 2022 reference equations to lung function interpretation in UK tertiary care patients impacts self-identified race/ethnic groups differently. Impacts on disease classification, eligibility to clinical treatments, and access to clinical trials may differ accordingly.

## P254 HOME SPIROMETRY MONITORING AS AN ADJUNCT TO CLINICAL CARE IN PATIENTS WITH FIBROTIC INTERSTITIAL LUNG DISEASES: FINDINGS FROM THE REMOTE-ILD RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2025-BTSabstracts.435

**Introduction** Remote monitoring of lung function has been proposed as an adjunct to support clinical care in patients with interstitial lung diseases (ILD). Previous studies have shown that home spirometry correlates well with hospital measurements and that patients are willing to do these measurements at home.

**Objectives** To investigate the impact of introducing remote monitoring for the provision of contemporaneous lung function at clinical appointments for patients with ILD.

**Methods** REMOTE-ILD was a randomised controlled multicentre study in England (NCT05662124). Patients with fibrotic ILD were recruited and randomised (1:1) to remote monitoring (RM) or usual care (UC). Participants in the RM arm were asked to record home spirometry and oximetry three times weekly for fortnightly clinical review of their data. The primary endpoint was contemporaneous spirometry within two weeks at scheduled clinical appointments during a 12-month observation period. Chi-squared was used to test independence of endpoint.

**Results** 139 participants were recruited and randomised, 67 RM participants and 66 UC participants completed 12-month observation. A total of 113 patients had a scheduled clinical appointment, 63 participants in RM and 50 participants in UC arms. The primary endpoint was met in 58 RM participants (92%) and 14 (28%) UC participants ($p<0.001$).

In the RM arm, median adherence to weekly home spirometry was 80.8% (IQR 46.2–96.2). The mean percentage of spirometry readings per person which were acceptable according to ATS/ERS criteria was 74.46% (SD 21.35). There was high correlation between home and hospital FVC readings (rho 0.891, $p<0.001$), home spirometry FVC had a mean difference of 347 mL (SD 331) compared with hospital FVC.

Five RM participants were contacted by clinical teams for an additional outpatient appointment in response to abnormal home measurements, of whom two were subsequently started on new ILD medications. Three RM participants had domiciliary oxygen assessments arranged in response to home oximetry without need for additional clinical appointments.

**Discussion** Home spirometry led to a greater number of scheduled clinical appointments with contemporaneous lung function when compared with usual care. Availability supported clinical decision making, whilst remote monitoring supported responsive clinical management.

**Funding** This study is funded by the NIHR (NIHR203424).

## P255 OBSTRUCTIVE SPIROMETRY IN A SEVERE PECTUS EXCAVATUM COHORT: DATA FROM THE NATIONAL PECTUS MDT

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The National Pectus multi-disciplinary team (MDT) meeting was commissioned by NHS England in April 2023 to review all cases of pectus excavatum (PE) being considered for surgical management. In cases of severe PE, the chest wall compresses the heart and lungs, which may result in cardiac or respiratory function compromise on exercise, measured during cardio-pulmonary exercise testing (CPET). This MDT has led to a further ongoing study on the impact of the timing of surgery on patient-reported improvement in quality of life, and improvement in CPET parameters (the RESTORE trial).

All patients discussed in the MDT require prior CPET and pulmonary function testing (PFT) to assess for physiological limitation and investigate for other potential causes. The majority of PE patients have restrictive spirometry, however a

**Abstract P255 Table 1** Demographics (N=28)

| Mean age (±SD): | 22.3 (±9.5) years |
|---|---|
| Sex: | |
| Female | 4 (14.3%) |
| Male | 24 (85.7%) |
| FEV1/FVC: | |
| Z-score 0 to -1 with obstructive flow/volume loop | 5 (17.9%) |
| Z-score -1 to -1.64 | 12 (42.9%) |
| Z-score -1.64 to -1.96 | 2 (7.1%) |
| Z-score <-1.96 | 9 (32.1%) |

subset of 28 patients potentially had an obstructive spirometry pattern (table 1), with a mean FEV1/FVC 0.72. This population was predominantly male (85.7%) with an average age of 22.3 years.

Prior to surgical intervention, we propose these patients undergo further investigation to exclude undiagnosed obstructive airways disease, and in event of a diagnosis being made, receive a trial of treatment which may alleviate the need for an invasive surgical procedure.

## P256 OBSERVATIONAL STUDY OF PRESERVED RATIO IMPAIRED SPIROMETRY IN THE IRISH ADULT POPULATION

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10.1136/thorax-2025-BTSabstracts.437

**Background** Preserved Ratio Impaired Spirometry (PRISm) is defined by reduced forced expiratory volume in one second ($FEV_1$) with a preserved $FEV_1$/FVC ratio. It is associated with increased morbidity and may represent an intermediate stage in the development of chronic respiratory disease. Small airway dysfunction (SAD) contributes to conditions such as COPD, asthma, and idiopathic pulmonary fibrosis.

**Aim** To examine the association between PRISm and SAD in an Irish adult population.

**Methods** A cross-sectional study was conducted at the Respiratory Department, Connolly Hospital, Dublin. Spirometry results from patients were retrospectively reviewed. Data included demographics, clinical history, and post-bronchodilator spirometry. PRISm was defined as $FEV_1/FVC \geq 0.70$ with $FEV_1 < 80\%$ predicted. SAD was diagnosed when at least two of MMEF, $FEF_{50}$, and $FEF_{75}$ were <65% predicted.

**Results** Over five years, 962 (24.2%) met PRISm criteria. Of these, 51.4% were male ($p = 0.044$). PRISm patients were significantly older ($p = 0.001$). Although more common in Caucasians, this was not significant ($p = 0.125$). Most were overweight or obese ($p = 0.689$, not significant). Mean $FEV_1$ was significantly reduced in PRISm ($p < 0.001$). $MMEF_{75-25}$ was significantly lower ($p = 0.040$), with marginal reductions in $FEF_{50}$ and $FEF_{75}$ ($p = 0.093$, $p = 0.058$).

**Conclusion** PRISm is a prevalent spirometric pattern in Irish adults and shows a significant association with SAD, supporting its recognition as a distinct early airway disease phenotype.

## P257 EXPIRATORY CENTRAL AIRWAY COLLAPSE INCREASES LONGITUDINALLY IN YOUNG SMOKERS WITH PRESERVED LUNG FUNCTION

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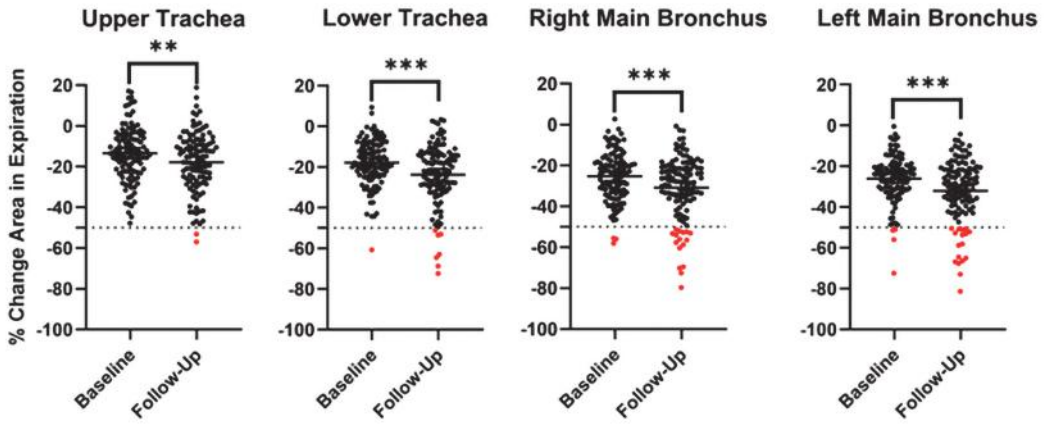
10.1136/thorax-2025-BTSabstracts.438

**Background** Expiratory central airway collapse (ECAC) comprises tracheobronchomalacia and excessive dynamic airway collapse. ECAC is associated with an increased risk of hospitalisation and greater symptom burden in COPD. We aimed to assess the prevalence and, for the first time, longitudinal stability of ECAC in young smokers with preserved lung function.

**Methods** We performed expiratory thoracic computed tomography (CT) scans at baseline and follow-up on current smokers aged 30–45 years old and with $FEV_1 \geq 80\%$ predicted, as part of the British Early COPD Network (BEACON) study. Morphological airway measurements were made from the upper trachea to the lobar bronchi. ECAC was defined as a reduction of the cross-sectional area of a central airway (trachea or main bronchus) by >50%.

**Results** 114 paired baseline and follow-up CT scans were evaluated (median age 40.2 years at baseline, median follow-up 5.4 years). The prevalence of ECAC was 5.3% (6/114) at baseline and 18.4% (21/114) at follow-up ($P = 0.001$). The degree of collapse of all the central airways increased on follow-up (figure 1). 3/6 of participants with ECAC at baseline did not have it at follow-up. All of the participants who developed ECAC at follow-up maintained $FEV_1 \geq 80\%$ predicted.

**Conclusions** The prevalence of ECAC increases longitudinally in young adult smokers despite preservation of their lung function. ECAC is dynamic in this patient group, meaning that serial assessments may be needed to diagnose it. Given the sequalae of ECAC in established COPD, further work is




**Abstract P257 Figure 1**

warranted to determine what drives increasing ECAC as young smokers age, so that appropriate preventive strategies can be identified.

## P258 EVALUATION OF PATIENTS WITH SUBGLOTIC-TRACHEAL STENOSIS AND UTILITY OF PULMONARY FUNCTION TESTS

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10.1136/thorax-2025-BTSabstracts.439

**Introduction** Subglottic-tracheal (STS) stenosis can be of malignant or benign aetiology. In its diagnosis, among other clinical, radiological and endoscopic data, the FEV1/FEP index has been described as having a high diagnostic specificity when it has a value > 10 L/min/ml.

**Aim** To analyse the general, endoscopic and in particular spirometric characteristics of our patients with STS.

**Methods** Retrospective descriptive study of patients with STS referred for interventional bronchoscopic treatment (from 2017 to 2024). General data, aetiology, lung function test with calculation of the FEV1/FEP index and therapeutic endoscopic manoeuvres were collected.

**Results** The sample consisted of 30 patients (79.3% female), mean age 62±13 years. The most frequent cause was idiopathic (41.4%) followed by stenosis secondary to tracheostomy (20.7%) and endothoracic goitre (10.3%). The remaining cases were: compression due to mediastinal masses, stenosis due to granulomatosis with polyangiitis or radiotherapy. Regarding endoscopic diagnosis, 85.7% of STS corresponded to grade 2 according to Cotton's classification, the rest being grade 1. Endoscopic treatment was performed in 25 patients, the most frequent (24.1%): cutting with an electric blade, balloon dilatation and instillation of intralesional corticosteroid. 44% of patients treated were referred to thoracic surgery. 55% of the patients had lung function test prior to endoscopic treatment. Of these, 25% had a normal volume flow curve, 12.5% had expiratory flattening and 62.5% had both inspiratory and expiratory flattening. FEV1/FVC index according to the morphology of the volume flow curve: 68.8% had an FEV1/EFP index >10L/min/ml.

**Conclusions** Spirometry and its volume flow curve are useful for the diagnosis and follow-up of tracheal pathology, with the FEV1/EFP index standing out.

**Abstract P258 Table 1** Spirometric values of those patients with lung function tests and mean FEV1/PEF index according to flow-volume curve morphology

| Spirometry | |
|---|---|
| **FVC (ml)** | 3042±928 |
| **FVC (%)** | 100.6±13.2 |
| **FEV1 (ml)** | 2221±659 |
| **FEV1 (%)** | 89.0±22.3 |
| **FEV1/FVC (%)** | 65.2±15.0 |
| **PFE (L/s)** | 3.28±1.59 |
| **PFE (%)** | 55.4±28.4 |
| **Flow-volume curve** | **VEF1/PFE (L/min/ml)** |
| **Normal** | 8.5±1.6 |
| **Expiratory flattening** | 11.4±2.1 |
| **Inspiratory and expiratory flattening** | 14.5±2.5 |

## P259 SHOULD PRIMARY CARE BE OFFERING FRACTIONAL EXHALED NITRIC OXIDE (FENO) TESTING? A REVIEW OF RAISED FENO LEVELS IN PATIENTS REFERRED TO SECONDARY CARE WITH A SUSPICION OF ASTHMA

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10.1136/thorax-2025-BTSabstracts.440

**Background** A direct access service for primary care patients to be referred into secondary care for pulmonary function tests has been in place for over 20 years. Due to the introduction of the BTS/NICE/SIGN guidance in November 2024, FeNO is now on the first stage of the flow chart as the initial test that should be performed in patients where a clinician has deemed the patient to have a history suggestive of asthma (https://www.nice.org.uk/guidance/NG245). If a patient was to have this test in Primary Care this would allow a quick, non-invasive point of care diagnostic test which could be performed at initial presentation with the GP.

**Aim** To identify the prevalence of raised FeNO levels in patients referred from Primary Care with a suspicion of asthma, whilst also investigating the ease of test procedure.

**Methods** A 5 year retrospective analysis of patients who were referred by Primary Care with a suspicion of asthma was performed.

Descriptive statistics were performed investigating patient demographics and FeNO results.

**Results** 7395 patient results were returned on first search. All patients from direct access primary care with ?asthma as part of their clinical details.

After data cleanse 4573 patients with only ?asthma as clinical details were included in the analysis.

See table 1.

96.4% (4410) achieved FeNO at their first visit, a failure rate of only 3.6%.

**Conclusion** 18.4% of patients attending directly from Primary Care would have a diagnosis of asthma using the BTS/SIGN/NICE 2024 guidance. A further 24.4% having an intermediate result which shows they may benefit from treatment with inhaled corticosteroids if they are symptomatic. Only 3.6% of patients were unable to perform the FeNO test.

This data suggests that 42.8% of patients could have potentially been tested and initiated on treatment with the correct diagnosis without the need for referral to Secondary Care which would reduce waiting times for diagnosis and treatment.

This study recommends the use of FeNO in Primary Care.

## P260 BRONCHIAL HYPERRESPONSIVENESS IN ASTHMA: THE USE OF DIRECT AND INDIRECT CHALLENGE TESTING AT DIAGNOSIS

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10.1136/thorax-2025-BTSabstracts.441

**Background** Bronchial challenge agents induce bronchoconstriction through distinct pathways, hence varying in sensitivity and specificity across patient cohorts. The BTS/NICE/SIGN 2024 asthma guidance recommends bronchial challenge testing (BCT) for diagnosis, but without specifying challenge agents. In contrast, ERS 2022 recommends a sequential approach: direct challenge followed by indirect if negative. We aim to establish the test concordance between methacholine and mannitol challenges ($BCT_{Mch}$ and $BCT_{Mann}$) and identify predictors of response to each in both adults and children. We will also assess the added value of sequential testing in adults.

**Methods** Symptomatic and untreated children and adults with GP-suspected asthma were recruited into the RADicA study. Clinical history, examination and objective tests including fractional exhaled nitric oxide (FeNO), blood eosinophil counts (BEC), spirometry, bronchodilator reversibility (BDR), skin prick testing (SPT) and both direct $BCT_{Mch}$ and $BCT_{Mann}$ were performed (on separate days prior to inhaled corticosteroid treatment).

**Results** Of 95 participants (median [IQR] age: 26.0 [13.2–39.1] years, 41.1% male) who completed both $BCT_{Mch}$ and $BCT_{Mann}$, 35.8% had a positive $BCT_{Mann}$ ($PD_{15}$<635 mg), and 33.7% a positive $BCT_{Mch}$ ($PD_{20}$<0.2 mg), with an overall agreement of 78.9% between the two tests: nine individuals had positive $BCT_{Mch}$ but negative $BCT_{Mann}$, compared to 11 $BCT_{Mann}$ positive and $BCT_{Mch}$ negative. Self-reported exercise-induced symptoms were not associated with either $BCT_{Mann}$ or $BCT_{Mch}$ outcomes. SPT, FeNO, BEC and BDR were associated with both positive $BCT_{Mann}$ and $BCT_{Mch}$ (table 1). In adults, following BTS/NICE/SIGN (2024) diagnostic pathway, 57–76% require BCT, of which 14–24% had positive $BCT_{Mann}$ and 11–30% for $BCT_{Mch}$. Following ERS (2022) diagnostic pathway, 43–71% patients require $BCT_{Mch}$, 79–89% had a negative result, requiring further $BCT_{Mann}$; of these only 12–16% subsequently tested positive.

**Conclusion** BCT is required in over half of cases using current UK and European diagnostic pathways; sequential testing

**Abstract P259 Table 1** FeNO results

| FeNO Level (ppm) | Patient numbers (n=?) | Percentage of Total patients (%) |
|---|---|---|
| <25 | 2451 | 53.6 |
| 25-49 | 1118 | 24.4 |
| ≥50 | 841 | 18.4 |
| Unable to perform test | 163 | 3.6 |

**Abstract P260 Table 1** Univariate logistic regression for predicting positive BCTMann and BCTMch in both adults and children

| | $BCT_{Mann}$ | | | $BCT_{Mch}$ | | |
|---|---|---|---|---|---|---|
| Predictors | OR | CI (95%) | P-value | OR | CI (95%) | P-value |
| SPT sensitisation ± | 5.800 | 1.98 – 17.00 | **0.001** | 5.06 | 1.73-14.85 | **0.003** |
| FeNO* | 7.27 | 2.76-19.19 | **<0.001** | 11.69 | 4.19-32.57 | **<0.001** |
| BEC# | 4.90 | 1.40-13.09 | **0.005** | 3.29 | 1.13-9.58 | **0.029** |
| BDR∞ | 5.96 | 1.99-17.80 | **0.001** | 9.55 | 3.01-30.33 | **<0.001** |

Note: *Positive fractional exhaled nitric oxide (FeNO)≥35ppb in children, ≥50ppb in adults. # Positive blood eosinophil counts (BEC) ≥0.4 x $10^9$ cells/L in adults, ≥0.5 x $10^9$ cells/L in children;± skin prick test (SPT) positive (≥3x3mm wheal size compared to negative control) in at least one common inhaled allergens including cat dander, dog, grass, birch, *Aspergillus Fumigatus*, house dust mite, *Alternaria* and *Cladosporium.* ∞Positive bronchodilator reversibility (BDR) was defined as >12% and 200ml improvement in $FEV_1$ following 400µg of salbutamol via a spacer.

advised by ERS, yields few additional diagnoses. We found no differential predictors of bronchial hyperresponsiveness between mannitol and methacholine. Given their moderate agreement, high cost and limited accessibility, identifying better predictors may help guide more efficient selection of challenge agents. This also highlight the urgent need for more accessible and efficient diagnostic tools.

## P261 QUANTIFYING OXYGEN REQUIREMENT IN EARLY WARNING SCORES IMPROVES RECOGNITION OF DETERIORATION COMPARED TO THE NATIONAL EARLY WARNING SCORE 2 (NEWS2) IN PATIENTS ON OXYGEN

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10.1136/thorax-2025-BTSabstracts.442

**Introduction** The National Early Warning Score 2 (NEWS2) is used across acute hospitals in England. It adopts a binary approach to oxygen therapy scoring; 0 points for room air, 2 points for supplemental oxygen. However, this simplistic approach may delay recognition of deteriorating patients, in those who have an escalating oxygen requirement despite otherwise stable vital signs. A recent meta-analysis demonstrated that early warning scores (EWS) with more granular oxygen quantification outperform their binary oxygen equivalents.[1]

**Aim** To compare the performance of seven vital sign only EWS containing graded oxygen weighting to NEWS2, to predict deterioration within 24 hours in hospitalised adults.

**Methods** A retrospective cohort study using data collected from a large tertiary NHS foundation trust, composed of four hospitals, including a university teaching hospital. All adults (age $\geq$ 16), admitted to hospital with recorded vital signs and an admission length > 24 hours were included. Admissions direct to intensive care (ICU) or discharged from the emergency department were excluded. We randomly sampled one observation set per admission. The outcome was a composite of unplanned ICU admission or death within 24 hours. We assessed the discrimination of seven EWS with oxygen graded in more detail than binary oxygen, compared with NEWS2.

**Results** There were 271,717 admissions with 8,441,252 observation sets. In 112,745 (41.5%) admissions, supplemental oxygen was required. Deterioration occurred in 14,979 (5.5%) admissions.

The best performing score was the oxygen graded Nottingham EWS (c-statistic 0.8398, 95% confidence interval 0.8321–0.8472), which outperformed NEWS2 (c-statistic 0.8102 (0.8022–0.8180)). Other fraction of inspired oxygen (FiO2)-based EWS, including NEWS-FiO2 (Malycha) and NEWS-FiO2 (Clarke), also showed better discrimination than NEWS2. See table 1.

**Abstract P261 Table 1** Comparison of the performance of 7 oxygen graded models versus NEWS2, at predicting deterioration within 24 hours in hospitalised adults on oxygen

| Model | C-statistic (95% confidence interval) |
|---|---|
| Nottingham EWS | 0.8398 (0.8321 – 0.8472) |
| NEWS-FiO2 (Clarke) | 0.8273 (0.8195 – 0.8348) |
| NEWS-FiO2 (Malycha) | 0.8175 (0.8096 – 0.8252) |
| NEWS2 | 0.8102 (0.8022 – 0.8180) |
| Leeds | 0.8060 (0.7977 – 0. 8139) |
| QADDS | 0.8046 (0.7960 – 0.8129) |
| Hamilton | 0.7900 (0.7811 – 0.7987) |
| MEWS-SF | 0.7827 (0.7736 – 0.7916) |

**Conclusions** Models with graded oxygen tended to show better predictive performance than NEWS2, however performance was influenced by how oxygen was incorporated. Addition of graded oxygen weighting in future iterations of NEWS may improve recognition of deterioration.

## REFERENCE

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## P262 EVEN NUMBER BIAS IN PULSE OXIMETRY MEASUREMENTS IN HOSPITALS IN DENMARK AND THE UK

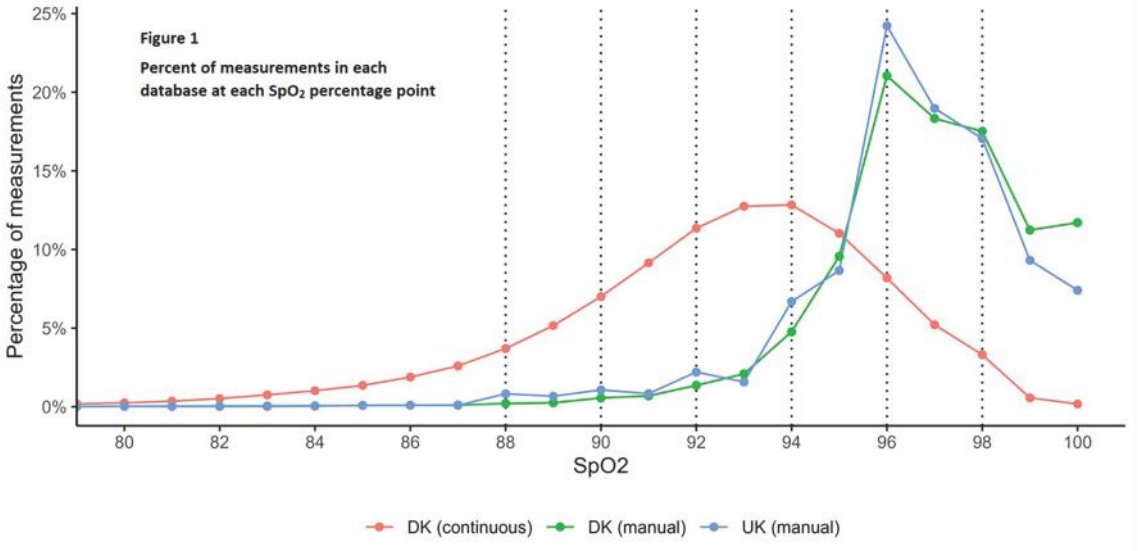
[1]BR O'Driscoll, [1]ND Bakerly, [2]MH Olsen, [2]J Molgaard, [2]EK Aasvang, [3]APH Karlsen, [3]CS Meyhoff. *[1]Northern Care Alliance NHS Foundation Trust, Salford, UK; [2]Copenhagen University Hospital – Rigshospitalet, Copenhagen, Denmark; [3]Copenhagen University Hospital – Bispebjerg and Frederiksberg, Copenhagen, Denmark*

10.1136/thorax-2025-BTSabstracts.443

**Background** Pulse oximetry measurements of oxygen saturation ($SpO_2$) may be subject to even number bias and boundary effects similar to other physiological measurements.[1] The extent and potential impact of this phenomenon are poorly documented. If present, such bias could have significant impact on the efferent limb of clinical escalation protocols and on future oxygen therapy guidelines. One would expect this phenomenon to be less prominent if the $SpO_2$ was collected automatically compared with manual monitoring.

**Methods** We have reviewed three large $SpO_2$ datasets from two countries: 11,553 automated $SpO_2$ entries in Denmark (DK-Auto), 7,602,352 manual $SpO_2$ entries in Denmark (DK-Manual) and 949,718 manual $SpO_2$ entries in the UK (UK-Manual).

**Results** 55.6% of DK-Auto entries, 57.5% of DK-Manual entries and 59.7% of UK-Manual entries were even numbers: (DK-Manual vs. DK-Auto, RR: 1.03,95%CI: 1.02–1.05, $p<0.0001$; UK-Manual vs. DK-Auto RR 1.07, 95%CI: 1.06–1.09, $p<0.0001$). The even-number bias was more pronounced in UK medical patients for whom 64.9% of SpO2 entries had even numbers (UK-Manual medical entries vs. DK-Auto medical entries RR 1.21, 95%CI: 1.17–1.25, $p<0.0001$). The proportion of even $SpO_2$ numbers was particularly high (67.0%) for UK medical patients who were using supplemental oxygen. The overall even-number bias is illustrated in figure 1. This bias was more pronounced for manual $SpO_2$ entries. The steps up from even to odd numbers are blunted in both manual datasets, whilst the steps down from even to odd numbers are exaggerated.




**Abstract P262 Figure 1**

**Conclusions** We found significant even number bias in routine pulse oximetry entries in the UK and Denmark. The bias was greater for manually collected data than for automated data. Even number bias in the automated data could arise from rounding bias in the oximeters that were used and/or the presence of $SpO_2$ peaks at 96% and 100%. Even number bias in the UK manual data was most marked for medical patients and for patients receiving supplemental oxygen where the $SpO_2$ might be rounded to an even number to meet a target saturation range or to avoid mandatory clinical escalation related to Early Warning Score boundaries. This bias could influence patient management and clinical outcomes.

## REFERENCE

1. Kleinig O, To MS, Ovenden CD, *et al*. Vital sign measurements demonstrate terminal digit bias and boundary effects. *Emergency Medicine Australasia* 2024;**36**: 543–546.

## P263 ASSESSMENT OF DIAPHRAGM MOTOR UNIT ACTIVITY USING HIGH-DENSITY SURFACE ELECTROMYOGRAPHY

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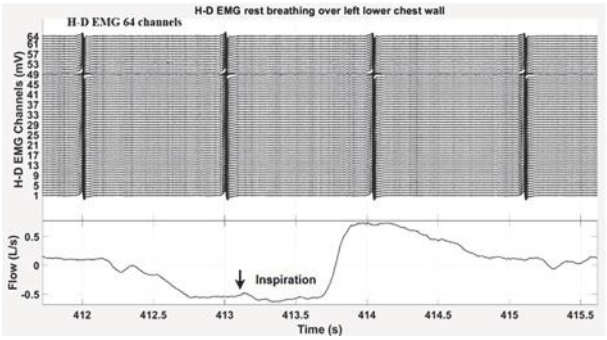
10.1136/thorax-2025-BTSabstracts.444

**Introduction** Respiratory muscle dysfunction leading to respiratory failure is a significant risk in both acute and chronic respiratory conditions. Developing non-invasive diagnostic indices of respiratory muscle weakness is crucial for improving access to such tools outside specialist clinical settings. While needle electromyography (EMG) enables the evaluation of individual motor unit activity, its invasive nature and limited clinical availability restrict its widespread application. High-density surface Electromyography (HD-sEMG) has shown considerable promise in assessing limb muscle activity, such as the tibialis anterior, where motor unit conduction velocity (MUCV) can be estimated and has been associated with recruitment thresholds and force production.[1] However, the potential of HD-sEMG in assessing respiratory muscles, particularly the diaphragm, remains largely unexplored. We aim to determine whether HD-sEMG can provide physiologically meaningful indices of diaphragm muscle activity, offering a novel and fully non-invasive approach to evaluating respiratory muscle function.

**Methods** Bilateral 64-channel HD-sEMG signals were recorded from the lower intercostal spaces, anterolaterally, during quiet, resting breathing. Signals were segmented using 500 ms sliding windows and processed via spike-triggered averaging across five columns of electrodes aligned with presumed diaphragm muscle fibre orientation. Motor unit conduction velocity (MUCV) was computed from these columns and averaged over the full breathing cycle.

**Results** In this ongoing study, signals recorded from five healthy adults (2 female, 3 male; median (IQR) age 24 (7) years, median (IQR) BMI 23.6 (0.3) $kg/m^2$) have been analysed to date. A representative HD-sEMG trace is presented in figure 1. Across the five participants, the median (IQR) MUCV was 2.5 (0.1) m/s.

**Conclusion** The observed MUCV values fall within the expected physiological range, supporting the feasibility of using HD-sEMG to assess diaphragm function. Future analyses will include motor unit recruitment thresholds and discharge rates at rest and during graded inspiratory loading, aiming to correlate these neural drive markers with inspiratory flow and mouth pressure. These metrics may help differentiate healthy individuals from patients with respiratory muscle weakness in clinical practice.




**Abstract P263 Figure 1**

## REFERENCE

1. Del Vecchio A, Negro F, Felici F, Farina D. Associations between motor unit action potential parameters and surface EMG features. *Journal of Applied Physiology* 2017;**123**(4),835–843.

## P264 THE RELATIONSHIP BETWEEN POSTURAL FVC CHANGE AND RESPIRATORY MUSCLE STRENGTH IN PATIENTS WITH MOTOR NEURONE DISEASE (MND)

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10.1136/thorax-2025-BTSabstracts.445

**Introduction** Reduced maximum inspiratory mouth pressure (PImax) indicates inspiratory respiratory muscle weakness. Drop in vital capacity (ΔFVC) greater than 20% from seated to supine posture is considered abnormal, whereas drop greater than 30% is considered pathognomonic for diaphragmatic paralysis.

**Aim** To assess whether drop of ΔFVC>20% can be used as surrogate of severe respiratory muscle weakness.

**Methods** A retrospective study which included data of MND patients from the medical registry of a tertiary lung function laboratory. Complete pulmonary function testing including FVC measurements in the seated and supine positions, respiratory muscle strength (RMS) assessment and pulse oximetry were performed in all patients.

**Results** Ninety-four MND patients (age: 60±11 yrs, height: 166±9 cm, BMI: 27.5±4.4 $kg{\cdot}m^{-2}$) were included. The vital capacity was measured in the seated (FVCseated: 95±23% pred) and supine position (FVCsupine: 86±25 %pred). PImax was 90±38%pred and PEmax was 90±35%pred. Forty-five patients had abnormal PImax%pred. while PEmax%pred. was abnormal in thirty-nine patients. Oxygen saturation in the seated position ($SpO_2$seated: 96±2%) and supine position

($SpO_2$supine: 94±2%) were also recorded. $SpO_2$ drop from the seated to the supine position was 2.4±2.2%. Seventeen patients had ΔFVC>20%. ΔFVC>20%was associated with $SpO_2$drop (r=-0.542, p=0.025). ΔFVC>20% was associated with PImax%pred (r=-0.343, p=0.002) and PEmax%pred (r=-0.333, p=0.003). Neither PImax (ΔFVC>20%, 76±37% vs ΔFVC<20%, 92±36%, p=0.115) nor PEmax (ΔFVC>20%, 81±35% vs ΔFVC<20%, 91±35%, p=0.268) were significantly different between the two groups.

**Conclusion** ΔFVC is related to respiratory muscle strength in patients with MND, and its use as an alternative when RMS assessment is not available might worth exploring.

## P265 FORCED OSCILLATION TECHNIQUE INDICES ARE RELATED TO UPPER AIRWAY DYSFUNCTION IN PATIENTS LIVING WITH MOTOR NEURONE DISEASE (PLWMND)

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10.1136/thorax-2025-BTSabstracts.446

**Introduction** Motor neurone disease (MND) is a progressive, degenerative, neuromuscular disorder. Patients with bulbar-onset present with swallowing and speaking abnormalities and have a worse prognosis.

**Aim** To assess the upper airway function of patients living with MND (plwMND) using the forced oscillation technique (FOT).

**Methods** Clinical assessment was performed in all patients. ALS Functional Rating Scale (ALSFRS-R) and the Sialorrhea scale were completed. Routine respiratory function testing, respiratory muscle assessment, and FOT were performed.

**Results** Forty-eight plwMND (age=66.5±9.6 yrs, height1.65 ±0.11 m, BMI=25.9±4.1 $kg{\cdot}m^{-2}$) were included in the study (17 patients with bulbar-onset). $FEV_1$, FVC, and $FEV_1$/FVC were 86±29%pred., 86±26%pred., and 78±12%, respectively. TLC was 86±16%pred. and DLco was 74±21%pred. X5tot, X19tot, R5tot and R19tot were -1.27±1.35 $cmH_2O$/(L/s), 0.53±0.59 $cmH_2O$/(L/s), 3.79±1.64 $cmH_2O$/(L/s), and 3.21 ±1.27 $cmH_2O$/(L/s), respectively. ALSFSR-R was 34.9±8.1au and the Sialorrhea scale was 2.1±1.4au. Patients with bulbar-onset had significantly greater R5exp (4.95±2.31 $cmH_2O$/(L/s)) and R19tot (3.80±1.69 $cmH_2O$/(L/s)) than patients with limb-onset. Additionally, patients with bulbar-onset had significantly greater Sialorrhea score (3.0±1.3au) compared to limb-onset patients (1.6±1.2au). Overall, Sialorrhea score showed a correlation with R19exp (r=0.336, p=0.010), X5exp (r=-0.297, p=0.021) and X19exp (r=-0.395, p=0.003).

**Conclusion** In plwMND, increased sialorrhea is associated with upper airway dysfunction which can be assessed by FOT.

## P266 TRANSCUTANEOUS ELECTRICAL STIMULATION OF THE UPPER AIRWAY DILATOR MUSCLES IN PATIENTS WITH MOTOR NEURONE DISEASE AND HEALTHY VOLUNTEERS

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10.1136/thorax-2025-BTSabstracts.447

**Introduction** Motor neurone disease (MND) is a progressive neurodegenerative disorder affecting motor neurones in the cortex, brainstem, and spinal cord. In advanced stages, it leads to sleep-disordered breathing, respiratory failure, and bulbar symptoms. We sought to visualise the contraction of the upper airway dilator muscles using ultrasound while applying transcutaneous electrical stimulation (TES) in the submental area.

**Methods** A case-control study was conducted involving patients with MND and healthy controls(NCT03160456). TES was delivered using a dual-channel stimulator (Premier Combo Plus EM-6300A, Everyway Medical Instruments, Taiwan) set at 30 Hz and 250 μs pulse width. Electrical current was individually titrated to a tolerable level of comfort. Stimulation was applied using 4×4 cm electrodes placed bilaterally in the submental region.

Ultrasound imaging was performed with a Venue system (GE Healthcare, USA) and a GE 12L-RS linear probe (3.4–12.6 MHz). The transducer was positioned 2 cm behind the chin, pointing cranially to capture sagittal and coronal planes of the tongue base muscles, including the geniohyoid and the genioglossus. Measurements included sagittal and coronal muscle diameters and cross-sectional area (CSA) with TES-off and TES-on.

**Results** 14 patients with MND (5 females, age 69±10 years; body mass index, BMI 26 ± 3 $kg/m^2$) and 11 healthy participants (4 females, age 34±8 years; BMI 23±3.6 $kg/m^2$) were recruited. In the sagittal plane, MND patients showed an increase of 5.0 (2.8)% in the midline diameter and 5.6 (3)% in the widest diameter with TES (p<0.001, respectively). Healthy participants demonstrated similar increases of 3.9 (2.8)% for midline and 3.9 (2.9)% for the widest diameters(p < 0.001, respectively). In the coronal plane, the midline diameter increased by 3.9 (1.5)% in MND patients and 2.9 (2.2)% in healthy controls, while the cross-sectional area (CSA) increased by 7.23 (4.2)% and 3.8 (3)% (p<0.001, respectively). All parameters responded similarly between groups, except for coronal CSA, which showed a significant difference (p = 0.033).

**Conclusion** TES can evoke contraction of the upper airway dilator muscles in MND patients, similar to what is observed in healthy volunteers. These findings may lead to early therapeutic interventional strategies aiming to preserve bulbar function in MND patients.

## P267 ASSESSMENT OF THE IMPACT OF NEBULISER TYPE ON FUGITIVE EMISSIONS IN A TREATMENT ROOM WHEN DELIVERING INHALED MEDICATIONS

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10.1136/thorax-2025-BTSabstracts.448

**Rationale** Nebulisers are widely used to deliver inhaled medications. Fugitive emissions are aerosols created by the nebuliser that are not inhaled by the patient. Fugitive emissions along with nebuliser exudate may potentially contain viral pathogens which can pose a risk to others in close proximity. A laboratory investigation was performed to assess the potential for exposure to fugitive emissions within a treatment room.

**Methods** Fugitive emissions produced by 3 different nebuliser types (AeroEclipse* II BAN* Nebuliser -breath-actuated, Neb-Tech† HDN† breath-enhanced, and Aerogen† Ultra-vibrating mesh) were assessed using salbutamol as a tracer and simulating an adult receiving a nebuliser treatment. Aerosol filters attached to breathing simulators were positioned 0.8 and 2 meters away from the nebuliser to represent caregivers within the room. Collection areas were also established within the test room to measure the amount of fugitive emissions/exudate that may have reached the caregiver but were not inhaled. The mass of salbutamol recovered from filters and collection surfaces was analysed by HPLC.

**Results** Total recovered emissions/exudate varied depending on the type of nebuliser used. The BAN* Nebuliser produced the lowest amount of fugitive emissions. The vibrating mesh nebuliser produced a significantly higher recovered amount directly below the nebuliser compared to the other two devices. The distance between the patient and the healthcare worker was not determined to be a significant factor in the amount of exposure to fugitive emissions.

**Conclusions** Healthcare workers should be aware of the potential risks associated with fugitive emissions from nebulisers and ways to minimise exposure. The choice of nebuliser can significantly affect the amount of fugitive emissions and exudate produced in a treatment environment.

## P268 EXTRA-FINE PARTICLE DEPOSITION OF TRIPLE INHALER THERAPIES USING THE NEXT GENERATOR IMPACTOR

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10.1136/thorax-2025-BTSabstracts.449

**Background** Small airways dysfunction is associated with worse disease control. Hence optimizing peripheral airways deposition using extra fine particles is considered desirable for achieving optimal disease control in asthma and COPD. This in vitro study compared the aerosol performance of BDP/FF/GB pMDI vs BUD/FF/GB pMDI and FLU/VIL/UMEC DPI, focusing on extra-fine particle delivery.

**Methods** The Next Generation Impactor was used to assess aerodynamic particle size distribution at 30 L/min for pMDI's and 60 L/min for DPI's. Extra-Fine Particle Fraction (eFPF, ≤1.36 μm) expressed as% of Delivered Dose (DD), and Mass Median Aerodynamic Diameter (MMAD) were compared across all active components.

**Results** BDP/FF/GB pMDI demonstrated substantially greater eFPF vs either BUD/FF/GB pMDI or FLU/VIL/UMEC DPI across all components (table 1). For the ICS moiety, the eFPF was 7.8-fold higher with BDP/FF/GB pMDI vs BUD/FF/GB pMDI and 14.8-fold vs FLU/VIL/UMEC DPI. For the LAMA moiety BDP/FF/GB pMDI was 6.6-fold higher vs BUD/FF/GB pMDI and 4.0-fold higher vs FLU/VIL/UMEC DPI. Concerning the LABA component, BDP/FF/GB pMDI was 4.2-fold higher vs BUD/FF/GB pMDI and 3.4-fold vs FLU/VIL/UMEC DPI.

Moreover, the MMAD was consistently smaller for BDP/FF/GB pMDI vs BUD/FF/GB pMDI or FLU/VIL/UMEC DPI across all moieties, in keeping with homogeneity of the extra fine solution formulation.

**Conclusions** The extra-fine formulation of BDP/FF/GB pMDI delivers a substantially higher proportion of extra-fine particles compared to both BUD/FF/GB pMDI and FLU/VIL/UMEC DPI. These findings suggest greater potential to target small airways, which in turn may be clinically relevant in optimising disease control in asthma and COPD.

**Abstract P268 Table 1** [1] within the BDP/FF/GB pMDI formulation; [2] within the BUD/FF/GB pMDI formulation; [3] within the FLU/VIL/UMEC DPI formulation

| | *ICS* | | | *LABA* | | | *LAMA* | | |
|---|---|---|---|---|---|---|---|---|---|
| | BDP[1] | BUD[2] | FLU[3] | FF[1] | FF[2] | VIL[3] | GB[1] | GB[2] | UMEC[3] |
| **eFPF (%)** | 31,0 | 4,0 | 2,1 | 31,5 | 7,5 | 9,3 | 30,2 | 4,6 | 7,5 |
| **MMAD (μm)** | 1,1 | 3,4 | 4,2 | 1,1 | 3,0 | 2,1 | 1,1 | 3,2 | 2,9 |

# Real world care of pleural fluid and infection

## P269 UTILITY OF THE RAPID SCORE AS A RISK PREDICTION TOOL IN THE MANAGEMENT OF PATIENTS WITH PLEURAL INFECTION: A SYSTEMATIC REVIEW AND META-ANALYSIS

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10.1136/thorax-2025-BTSabstracts.450

**Background** Pleural infection poses significant clinical challenges due to its high morbidity and mortality. The RAPID score, a validated risk stratification model incorporating Renal function, Age, Purulence of pleural fluid, Infection source, and Dietary factor, effectively predict patients' outcomes. However, the clinical utility of the RAPID score across different healthcare systems remain undefined.

**Objective** To systematically review and meta-analyse current literature on the prognostic value of the RAPID score in patients with pleural infection, focusing on its ability to predict 3-month mortality and its association with secondary clinical outcomes.

**Methods** A comprehensive literature search was conducted across PubMed, Ovid Embase, and Central Register of Controlled Trials, following PRISMA guidelines. Studies evaluating the RAPID score in adults with pleural infection and reporting on mortality or related outcomes were included. The primary outcome was 3-month mortality rate. Secondary outcomes were length of hospital stay, use of intrapleural enzyme therapy (IET), and need for surgical intervention. Two independent reviewers extracted the data and performed quality assessment. Random-effects meta-analyses were conducted to pool effect estimates.

**Results** A total of nine studies met the inclusion criteria, including 3,179 patients. The pooled analysis demonstrated that patients with high RAPID scores had significantly higher overall 3-month mortality (31.9%; 95% CI: 25.6%–38.9%) compared to low RAPID and medium score groups; 3.4% (1.9 – 5.9%), 11.6% (8.4 – 16.0%), longer hospital stays 18.8 days vs 15.8 days in low group, increasing likelihood of needing of IET 75.0 (71.3 – 78.4) vs (10.4%;2.1 – 38.7) and (72.0%;81.5 – 98.7%) in low and medium score groups respectively and higher surgical intervention 19.5% (9.9 – 35.0%) vs 10.3% (5.2 – 19.6%) in low score.

**Conclusion** This study supports the evidence that patients with pleural infection with a higher RAPID score have more complicated clinical course and higher mortality rate. Adoption of RAPID score-based clinical decision-making pathway at baseline may facilitate early risk stratification and personalised treatment approaches.

## P270 THE IMPACT OF THE PLEURAL EFFUSION MICROENVIRONMENT ON MACROPHAGE PHENOTYPE AND FUNCTION

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10.1136/thorax-2025-BTSabstracts.451

**Intro** Parapneumonic effusions complicate up to 40% of pneumonia hospitalisations and are associated with 3–6 fold increase in mortality. Progressive fibrinous septations increase the complexity of the parapneumonic effusion and render drainage more difficult. Macrophages are the dominant immune cells in the healthy pleural space, however, their role in parapneumonic effusions remains poorly understood.

**Methods** Patients in St James's Hospital undergoing drainage of parapneumonic effusion or drainage of transudative effusion (fluid accumulation secondary to hydrostatic pressure) were recruited, and clinical data recorded. Peripheral blood mononuclear cells (PBMCs) from healthy donors were differentiated into macrophages and incubated with the acellular component of pleural fluid for 24 hours to mimic the pleural effusion environment. Macrophage phenotype was interrogated by flow cytometry, and macrophage thrombin generation was evaluated using a fluorogenic assay.

**Results** 10 parapneumonic and 8 transudative effusions patients were recruited. Pleural fluid skewed macrophages toward an M2 phenotype, with increased expression of CD163 ($p=0.021$) and reduced expression of CD40 ($p=0.009$). Transudative fluid upregulated CD163 more than parapneumonic fluid ($p=0.045$). (figure 1a) Functionally, pleural fluid enhanced macrophage-dependent thrombin generation, reducing lagtime ($p<0.0001$), increasing peak thrombin ($p=0.0175$), and increasing endogenous thrombin potential (ETP) ($p<0.0001$), in a dose-dependent manner. Parapneumonic fluid promoted greater induction of macrophage-dependent thrombin generation than transudative fluid (figure 1b).

**Conclusion** These findings confirm that pleural fluid influences macrophage phenotype and increases their thrombogenic potential. This highlights the macrophage as a key player in the fibrin deposition characteristic of complicated parapneumonic effusions and suggests a potential immunothrombotic target for patients with pleural infection.

## P271 IMPACT OF EARLY VERSUS DELAYED INTRAPLEURAL FIBRINOLYTICS ON PLEURAL INFECTION OUTCOMES: AN OBSERVATIONAL STUDY

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10.1136/thorax-2025-BTSabstracts.452

**Background** The MIST2 trial demonstrated fibrinolytics reduce surgery rates and hospital stays in pleural infections, but optimal dosing & timing is unclear.This study compared early (within 24 hours) vs. delayed therapy.
**Methods** Between Nov. 2018 to Oct.2024, consecutive patients with pleural infections were included based on clinical signs (fever or raised inflammatory markers) and pleural fluid criteria (pH<7.2, gram stain positivity, or bacterial growth). All received drainage ± fibrinolytics (Alteplase 10 mg + DNase 5 mg daily for up to 3 days). Group 1 (pre-Nov. 2021) received fibrinolytics after drainage failure; Group 2 (post-Nov. 2021) received fibrinolytics early. Outcomes are listed in table 1.
**Results** Group 1(n=36) and Group 2(n=59) had similar mortality rates (5.56% vs. 5.08%). Group 2 had shorter hospital stays (7.73 vs. 10.92 days, p=0.115) and chest tube durations (4.40 vs.6.20 days,p <0.05).Predicted FVC at 4 months was higher in Group 2 (99.4% vs. 90.5%,p=0.031).
**Conclusions** Early fibrinolytic therapy appears to reduce hospital stay and chest tube duration without increasing mortality or requirement for surgery. Further research is needed.

## P272 IMAGING CHANGE AFTER INTRAPLEURAL FIBRINOLYTICS IN PATIENTS WITH INDWELLING PLEURAL CATHETERS AND SEPTATED EFFUSION: A RETROSPECTIVE REVIEW OF THE OXFORD PLEURAL UNIT DATA

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10.1136/thorax-2025-BTSabstracts.453

**Background** Intrapleural fibrinolytics are used in selected patients with indwelling pleural catheters (IPCs) and septated malignant pleural effusions (MPEs), but imaging data to support their effectiveness remain sparse.
**Aim** To assess whether fibrinolytics administered via IPC lead to a change in objectively scored ultrasound-visible septations.
**Methods** All patients from the Oxford Pleural Service who received intrapleural fibrinolytics via an indwelling pleural catheter (IPC) since routine ultrasound imaging has been recorded (2012–2025) were identified. Fourteen patients met inclusion criteria; 12 had stored thoracic ultrasound (TUS) images from before and after fibrinolysis. A standardised scoring system was developed to assess three domains: number of septations, septal thickness, and proportion of anechoic free fluid. All images were anonymised and presented in random order to an experienced pleural ultrasound practitioner, who was blinded to clinical information and whether the ultrasound was pre or post treatment.
**Results** Initial analysis of the data demonstrates that fibrinolytic therapy was associated with a reduction in the number

**Abstract P271 Table 1**

| | Group 1 | Group 2 | *p*-value(95% CI) |
|---|---|---|---|
| Mean Age(SD) | 62(15.24) | 59.66(17.44) | |
| Surgery in 3 months(%) | 3(8.33) | 1(1.694) | 0.118 |
| Mortality in 3 months(%) | 2(5.55) | 3(5.08) | 0.921 |
| Expected Mortality(RAPID score,%) | 10.33% | 12.5% | 0.749 |
| Hospital Stay(days,mean±SD) | 10.92(13.17) | 7.73(6.31) | 0.1157(-0.80-7.17) |
| Chest tube duration(days,mean ± SD) | 6.20(4.32) | 4.40(2.25) | 0.0153(0.35-3.24) |
| Mean Antibiotic duration,days(SD) | 19.78(14.49) | 18.31(8.64) | 0.535(-3.23-6.18) |
| Percent predicted FVC at 4 months (range) | 90.5(49-126) | 99.4(58-148) | 0.031 |

of pleural septations on ultrasound in most patients, and fluid drainage improved. Full assessment of the analysis will be presented at the BTS winter conference.

**Conclusion** This study suggests that intrapleural fibrinolytics visibly reduce pleural septations on ultrasound in patients with IPCs and septated MPEs. These imaging findings offer support for a biological effect of fibrinolytics on pleural architecture and provide a rationale for further prospective studies. The ultrasound scoring approach used here could support objective imaging endpoints in future trials.

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## P273 A NOVEL AMBULATORY PLEURAL EFFUSION PATHWAY IN A TERTIARY REFERRAL HOSPITAL IN IRELAND REDUCED PLEURAL EFFUSION ADMISSIONS BY 31% OVER 9 MONTHS

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10.1136/thorax-2025-BTSabstracts.454

**Background** Traditionally, pleural effusion procedures in Ireland are performed in the inpatient setting. Recent evidence from other jurisdictions shows that ambulatory pathways for pleural effusion procedures avoids admission, lowers healthcare costs and improves patient experiences. Previous audit data from our centre showed that 96% of pleural effusion procedures were performed in the inpatient setting. We established a pilot Ambulatory Pleural Effusion Pathway, in partnership with community nursing colleagues, for assessment and management of pleural effusions in the outpatient setting, offering diagnostic and therapeutic assessment of pleural patients. Here, we sought to assess the impact of our pilot pathway on inpatient admissions.

**Methods** Our pathway was launched in September 2024. We performed a retrospective review of all patients cared for through the Ambulatory Pleural Effusion Pathway over the first nine months of this pilot. Data was collected regarding patient demographics, referral source, days from referral to assessment, underlying diagnosis, and procedures performed. The numbers of procedures performed at the Ambulatory Pleural Effusion Procedure list was compared to previous audit-based estimates of the number of pleural effusion procedures performed in our centre in order estimate the number of avoided admissions.

**Results** Over 9 months, the pathway provided care for 43 individual patients, including 102 in-person reviews at the ambulatory pleural list and 235 telephone contacts with our Pleural CNS. 43 patients underwent 55 procedures, including 30 thoracocenteses, 3 pleural biopsies, 5 IPC insertions, 27 IPC reviews, 2 IPC insertions, and 1 chest drain insertion. 57% of patients had malignant pleural disease, and 47% were older than 75 years. 55% were referred by the Oncology Service. Previous audit data indicated 236 inpatient pleural effusion procedures performed over one year (=177 over nine months), and that those potentially suitable for ambulatory management (i.e. with no other medical issues requiring admission) had average LOS of 14.9 days. Based on this, our pilot pathway reduced admissions involving pleural effusion procedures by 31% (i.e. 55/177), saving 894 bed days over 9 months.

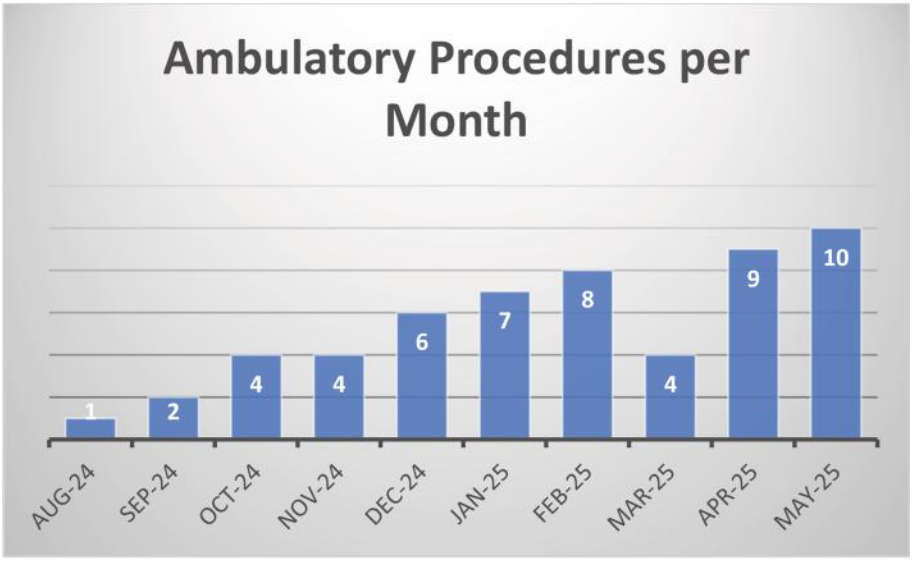
**Conclusion** A pilot Ambulatory Pleural Effusion Pathway can substantially reduce admissions to an Irish hospital for pleural effusion procedures and save bed days.

## P274 DIAGNOSTIC UTILITY AND COST EFFECTIVENESS OF PLEURAL FLUID ANALYSIS DURING LOCAL ANAESTHETIC THORACOSCOPY

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10.1136/thorax-2025-BTSabstracts.455

**Background** Pleural fluid is routinely sent for cytological and microbiological analysis during local anaesthetic thoracoscopy (LAT) in patients with unexplained unilateral pleural effusion.




**Abstract P273 Figure 1**

**Abstract P274 Table 1**

| | Pleural Fluid | Corresponding Pleural Biopsies |
|---|---|---|
| **Cytology (n=93)** | 58 – No malignant cells | 22 - pleural malignancy<br>32 - fibrous pleuritis<br>4 - unremarkable |
| | 8 – Atypical cells | 7 - pleural malignancy<br>1 - benign |
| | 12 – Malignant cells but insufficient for molecular markers | 12 - pleural malignancy |
| | 15 – Malignant cells and sufficient for molecular markers | 15 - pleural malignancy |
| **Microbiology (n=109)** | 106 - No bacterial growth | No relevant finding |
| | 3 – Contaminant or no clinically relevant growth | No relevant finding |
| | 109 - Mycobacterial culture negative | 1 – Acid fast bacilli growth |

However, there is currently no established guidance on the utility of additional fluid analysis alongside pleural biopsy.

**Aim** To evaluate the diagnostic utility and cost effectiveness of concurrent pleural fluid cytological and microbiological analysis during LAT, in addition to pleural biopsy for histology and microbiology.

**Method** Retrospective analysis of 123 consecutive LAT procedures performed between January 2022 and December 2024. Cytological analysis was performed on 93 pleural fluid samples and results were categorised as: no malignant cells, atypical cells, malignant cells with insufficient sample for molecular markers, or malignant cells with sufficient sample. Microbiological analysis, including bacterial and mycobacterial cultures, was performed on 109 samples and categorised as: clinically relevant growth or no growth.

**Results** Of the 123 patients, mean age was 74.2 years and male 78% (96).

Of the 93 fluid cytology samples: 58 (62%) showed no malignant cells, of these 22 had malignancy confirmed on pleural biopsy, 32 showed fibrous pleuritis, and 4 were unremarkable. 8 (7%) showed atypical cells, of which 7 had confirmed malignancy on pleural biopsy and 1 was benign. 12 (13%) cytology samples showed malignant cells insufficient for molecular markers, and all had confirmed pleural malignancy on biopsies. 15 (16%) demonstrated malignant cells sufficient for molecular markers and all had confirmed pleural malignancy on biopsies.

Of 109 pleural fluid samples sent for bacterial and mycobacterial cultures: 2 grew organisms of no clinical significance, and 1 showed contaminant growth. One patient had positive pleural biopsy culture for acid fast bacilli, with negative pleural fluid culture.

Pleural fluid cytological, bacterial and mycobacterial analyses incurred an additional cost of £37, £27 pounds and £50 per patient, respectively.

**Conclusion** Cytological analysis of pleural fluid during LAT did not provide additional diagnostic value or alter patient management. Similarly, microbiological cultures showed no growth, in the absence of clinical suspicion of infection. Routine use of these tests add healthcare costs without any benefit. Microbiological analysis, however, could be considered in clinically relevant cases where infection is suspected.

## P275 DO SELF-DRAINERS HAVE HIGHER INDWELLING PLEURAL CATHETER INFECTION RATES THAN NON-SELF-DRAINERS?

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10.1136/thorax-2025-BTSabstracts.456

**Introduction** Indwelling pleural catheters (IPCs) are widely used for symptomatic relief in malignant pleural effusion (MPE). While IPC drainage is typically managed by District Nurses (DNs), an increasing number of patients and carers now undertake self-drainage (SD). Self-management offers patients greater autonomy, flexibility, and reduced reliance on healthcare resources. Reported IPC-related infection rates typically range from 5% to 10%. This analysis aimed to compare infection outcomes between self-drainers (SD) and non-self-drainers (NSD) within a tertiary pleural service.

**Methods** A retrospective analysis was conducted on all IPC insertions performed between January 2024 and February 2025.

**Results** A total of 111 patients were included. 56 patients were male (50.5%). 14/111 (12.6%) were SDs and 97/111 (87.4%) were NSDs. Overall infection rate was 20/111 (18%). The median age of SD to NSD was 52 vs 71 years,

**Abstract P275 Table 1** Comparison of infection rates and time between SD and NSD – 3 patients in SD group and 0 in NSD group had both pleural and wound infection

| | SD | NSD | P value |
|---|---|---|---|
| **Overall infection rate** | 5/14 (35.7%) | 15/97 (15.5%) | 0.128 |
| **Pleural infection** | 4/14 (28.6%) | 10/97 (10.3%) | 0.076 |
| **Median time to pleural infection (days)** | 57 | 58.5 | 1 |
| **Wound infection** | 4/14 (28.6%) | 5/97 (5.2%) | 0.014 |
| **Median time to wound infection (days)** | 37 | 51 | 0.29 |

respectively. Pleural and wound infection rates were higher in SD group with similar median time to pleural infection in both groups, however shorter median time in SD group with respect to wound infections (see table 1).

Among 61 patients receiving SACT, 12 (19.6%) developed infections, compared to 8/50 (16%) in the non-SACT group (p=0.80). By diagnosis, infection rates were highest in breast cancer (28.6%), mesothelioma (26.7%), and lymphoma (25%), though these differences were not statistically significant. No significant difference in infection rates was observed between malignant (19/104) and benign effusions (1/7). The time IPCs remained in-situ (range 5–430 days, median 74) was not associated with increased infection risk.

**Discussion** Self-drainage was associated with significantly higher wound infection rates and a trend toward increased pleural and overall infections. The small sample of SD patients and higher prevalence of infection-prone diagnoses such as breast cancer within this group may confound interpretation. Nonetheless, these findings highlight a need for more education, not only for SD but all IPC patients. Based on this, our service is introducing a structured checklist and competency-based 'license' for all patients and carers undertaking self-management. These tools are designed to serve both as a practical aide-mémoire and as a framework for assessing aseptic technique in self-management.

## P276 TALC PLEURODESIS-ANYTHING BUT A DRY SUBJECT: UNDERSTANDING THE CHALLENGES OF DELIVERING TALC PLEURODESIS AS A FIRST-LINE TREATMENT TO PATIENTS WITH MALIGNANT PLEURAL EFFUSION IN A LARGE UK PLEURAL CENTRE

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10.1136/thorax-2025-BTSabstracts.457

**Introduction** Malignant pleural effusion (MPE) affects 50,000 patients per year in the UK and is characterized by debilitating symptoms of breathlessness. Talc pleurodesis (TP) and indwelling pleural catheters (IPC) are offered as definitive treatment options to manage MPE with no superiority of either treatment in symptom control.[1] However, IPCs are being increasingly offered as the preferred treatment due to perceived logistical challenges in delivering TP. The aim of this study is to understand the challenges of using TP as a treatment for MPE.

**Method** We conducted a retrospective analysis of the pleural procedure dataset to identify all patients who were treated with talc pleurodesis (talc poudrage or slurry) for MPE between Jan'22 and Dec'24. Patient demographics, procedure wait times, admission duration, pleurodesis success and follow-up intervals were extracted from electronic medical records and analyzed.

**Results** Of the 194 procedures performed to manage MPE, 40 patients were treated with TP. Median age was 77.5 ( IQR 18) ;22/40 (55%) were male. 35/40 (87.5%) were elective admissions with an average waiting time of 7.8 working days from time of decision to admission. Mean hospital stay was 8.8 days, reduced to 7.5 days for those who underwent thoracoscopy and talc poudrage. Daily drain output and chest X-ray were 1.4 times more likely to be used than the SIMPLE protocol to guide chest drain removal. Talc pleurodesis was successful in 61.3% of patients; 51.6% of patients had their first follow-up appointment at the recommended 2 weeks. 8/40 (20%) did not receive talc pleurodesis as planned due to reasons including dislodged chest drain, poorly expansile lung or pain intolerance.

**Conclusion** Although TP is recommended as a first-line treatment for the management of MPE, timely delivery remains challenging with long waiting times. Local pleural services should work to provide TP as a treatment option to patients with an acceptable success rate in a timely fashion.

### REFERENCE

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## P277 LENT SCORE UTILISATION IN INDWELLING PLEURAL CATHETER (IPC) PATHWAYS: AN AUDIT-BASED RECOMMENDATION

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10.1136/thorax-2025-BTSabstracts.458

**Background** Indwelling pleural catheters (IPCs) are established for recurrent malignant pleural effusions (MPEs), offering symptom control and outpatient management. Current British Thoracic Society (BTS) guidance emphasises patient-centred decision-making and advises that the LENT prognostic scoring system could be used for planning MPE management. This would include deciding if IPC insertion is appropriate.

**Methods** We conducted a retrospective analysis of 25 IPC insertions from 2023–24. In addition to the data required for LENT calculation, mortality, complications and IPC removal were reviewed. The LENT score was calculable in 17 patients and grouped as Low (0–1), Moderate (2–4), or High (5–7). We assessed survival duration and 1-month mortality by risk group.

**Results**

- LENT Score Distribution (n=17;):
- Low Risk (n=3): 33% deceased (died at 1 month)
- Moderate Risk (n=12): 66%deceased; Median survival 1.5 months (range 1–9); 50% 1-month mortality
- High Risk (n=2): 100% deceased; 100% 1-month mortality
- Complications:
- Immediate: 6 Patients (2 admitted; no IPC removals) complications were Pain, Pneumothorax, Bleeding
- Delayed: 5 Patients (4 admitted) complications were empyema, pain, accidentally cut in A&E.
- IPC Removals: 36% (9/25)
- N=3 removed due to delayed complications (pain, accidental cut, empyema)
- N= 6 removed due to spontaneous non-drainage
- Prior to IPC, 68% (17/25) had ≥1 pleural procedure—most commonly a single therapeutic drain.

**Conclusion/Recommendations** LENT score stratification closely correlates with post-IPC survival. Low-risk patients (18%) showed excellent outcomes, supporting IPC use in this group. Moderate-risk patients (70%) had highly variable trajectories. Despite a median survival of 1.5 months, some died within days, highlighting the importance of combining LENT scoring with clinical judgement. In high-risk patients (12%), rapid mortality questions IPC utility, suggesting one-off large-volume aspiration and symptom palliation (e.g., morphine) may be more appropriate.

We acknowledge the small sample size. Importantly LENT scoring requires pleural aspiration prior to IPC consideration. If incorporated into decision making this would preclude up front IPC insertion even if malignancy is highly suspected. This increases the number of required patient attendances. Nonetheless, integrating the LENT score supports BTS aim of outcome-driven, individualised care.

## P278 UNEXPECTED ACTIVITY OF THE PLEURAL NURSE SPECIALIST FOR IPC PATIENTS IN A TERTIARY CENTRE

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10.1136/thorax-2025-BTSabstracts.459

**Introduction** The Pleural Nurse Specialist (PNS) plays a pivotal role in the management of patients with indwelling pleural catheters (IPCs), particularly within a cohort often characterised by complex comorbidities and palliative care needs. Beyond performing pleural procedures, the PNS is instrumental in ongoing fluid surveillance, symptom control, patient support and care coordination. As the primary point of contact for IPC patients, the PNS is well positioned to respond to patient concerns. This analysis aimed to quantify the volume and nature of unplanned patient-initiated contacts with the PNS following IPC insertion, and to evaluate the associated clinical outcomes and service implications.

**Method** A retrospective analysis was conducted, reviewing patient contacts following IPC insertion between January and December 2024. Data were collected on unplanned telephone contacts initiated by patients or carers, subsequent interventions, and the number of planned and unplanned face-to-face (F2F) reviews.

**Results** Ninety-nine patients were included (median age 71 years; 52.5% male), of whom 64.6% were receiving systemic anti-cancer treatment. A total of 138 unplanned telephone contacts were recorded. Forty patients (40.4%) contacted the PNS two or more times. The average time to first call was five weeks post-IPC insertion. The most common reasons for contact were symptom reporting (e.g., breathlessness, pain) and requests for information or reassurance.

Subgroup analysis by performance status (PS) suggested a trend toward higher call rates among those with PS 0–1, although this did not reach statistical significance (see table 1). Planned F2F reviews occurred in 82 patients (82.1%), with 41 patients (41.4%) attending at least two reviews. Following 23 patient-initiated calls (16.7%), unplanned F2F reviews were arranged, potentially averting escalation to primary or emergency care. Notably, 59.4% of emergency admissions (13/22) occurred outside of PNS service hours.

**Abstract P278 Table 1** Summary of phone calls by patient to PNS team

| Number of calls | 1 or more | 2 or more | 3 or more | 4 or more | 5 or more | F2F appt made due to call | Emergency admission |
|---|---|---|---|---|---|---|---|
| Total (n=99) | 60 (60.6%) | 40 (40.4%) | 22 (22.2%) | 10 (10.1%) | 6 (6.1%) | 23 (23.2%) | 22 (22.2%) |
| PS 0 (n=11) | 7 (63.6%) | 5 (45.5%) | 2 (18.2%) | 1 (9.1%) | 0 (0%) | 3 (27.3%) | 4 (36.4%) |
| PS 1 (n=25) | 18 (72%) | 12 (48%) | 9 (36%) | 5 (20%) | 4 (16%) | 5 (20%) | 4 (16%) |
| PS 2 (n=20) | 10 (50%) | 7 (35%) | 4 (20%) | 1 (5%) | 0 (0%) | 4 (20%) | 3 (15%) |
| PS 3 (n=15) | 6 (40%) | 3 (20%) | 1 (6.7%) | 0 (0%) | 0 (0%) | 0 (0%) | 4 (26.7%) |
| SACT (n=64) | 40 (62.5%) | 28 (43.8%) | 16 (25%) | 8 (12.5%) | 5 (7.8%) | 16 (25%) | 11 (17.2%) |
| No SACT (n=35) | 20 (57.1%) | 12 (34.2%) | 6 (17.1%) | 2 (5.7%) | 1 (2.9%) | 7 (20%) | 11 (31.54%) |

**Discussion** These findings underline the value of the PNS, reducing emergency health service utilisation and maintaining continuity in pleural disease management. The capacity of the PNS to triage concerns, arrange timely intervention, and support patients holistically suggests a model of care that is both patient-centred and resource-efficient. Further work is needed to evaluate the impact of non-IPC-related PNS contacts on broader service delivery.

## Advances in pulmonary vascular medicine

### P279 EVALUATING AGE-ADJUSTED D-DIMER CUT-OFFS AND THE ROLE OF HIGHER MULTIPLIERS IN PULMONARY EMBOLISM DIAGNOSIS

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10.1136/thorax-2025-BTSabstracts.460

**Background** The age-adjusted D-dimer (AADD) cut-off (age ×10 ng/mL) improves specificity in pulmonary embolism (PE) diagnosis but requires validation in heterogeneous populations. This study evaluates AADD's performance in a diverse cohort and explores increasing the age-adjustment multiplier to enhance specificity.

**Methods** A retrospective analysis included 277 patients (mean age 63.9 years; 18.1% PE prevalence) undergoing CT pulmonary angiography and D-dimer testing at Oxford University Hospitals (2018). Exclusions included trauma, perioperative states, and chronic PE. Conventional D-dimer (500 ng/mL) was compared to AADD (multipliers ×10 and ×11). Sensitivity, specificity, negative/positive predictive values (NPV/PPV), and accuracy were calculated.

**Results** Both conventional D-dimer and AADD demonstrated 100% sensitivity and NPV. AADD (×10) increased specificity from 11.45% (95% CI: 7.6–16.3%) to 20.70% (15.6–26.6%), correctly reclassifying 9% of PE-negative patients. Raising the multiplier to ×11 further improved specificity to 26.43% (20.8–32.7%) without compromising sensitivity. Accuracy increased from 27.44% (conventional) to 39.71% (×11 multiplier). Multipliers >12 reduced sensitivity, but ×11 maintained 100% sensitivity.

**Discussion/Conclusion** In a heterogeneous population, AADD safely ruled out PE while enhancing specificity, reducing unnecessary imaging. Increasing the multiplier to ×11 provided additional gains in specificity (14.98% vs. conventional; 5.73% vs. ×10) without missed diagnoses. These findings support refining AADD thresholds to optimize diagnostic efficiency. While single-centre data and sample size limit generalizability, results align with prior studies, advocating for multiplier adjustments to improve clinical utility. Adoption of AADD with a ×11 multiplier could enhance cost-effectiveness and patient safety, particularly in older populations vulnerable to contrast-related complications. Further multicentre studies are warranted to validate these findings.

### P280 INCIDENTAL PULMONARY EMBOLISM (IPE) AND THE VALUE OF CT PULMONARY ANGIOGRAPHY FOLLOW-UP VERSUS EXPERT REVIEW

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10.1136/thorax-2025-BTSabstracts.461

**Introduction and Objectives** PE is the third most common cardiovascular disease worldwide and is potentially life-threatening if not identified and treated early. Incidental PE (iPE) identified on non-dedicated thoracic imaging is increasing in incidence and clinical practice in verifying iPE is heterogeneous.

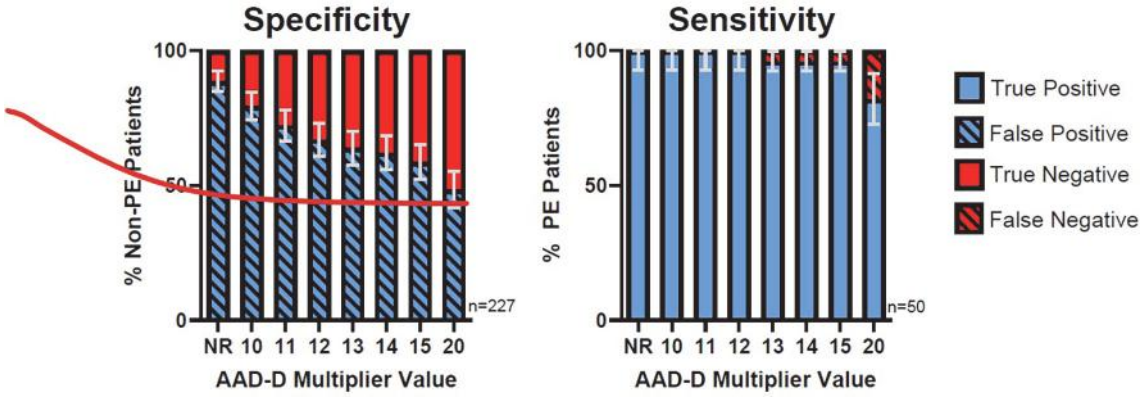
**Objectives** Assess the prevalence of iPE and frequency of CT pulmonary angiography (CTPA) validation.

Identify the diagnostic accuracy of iPE without CTPA validation and factors predicting false-positive diagnosis.

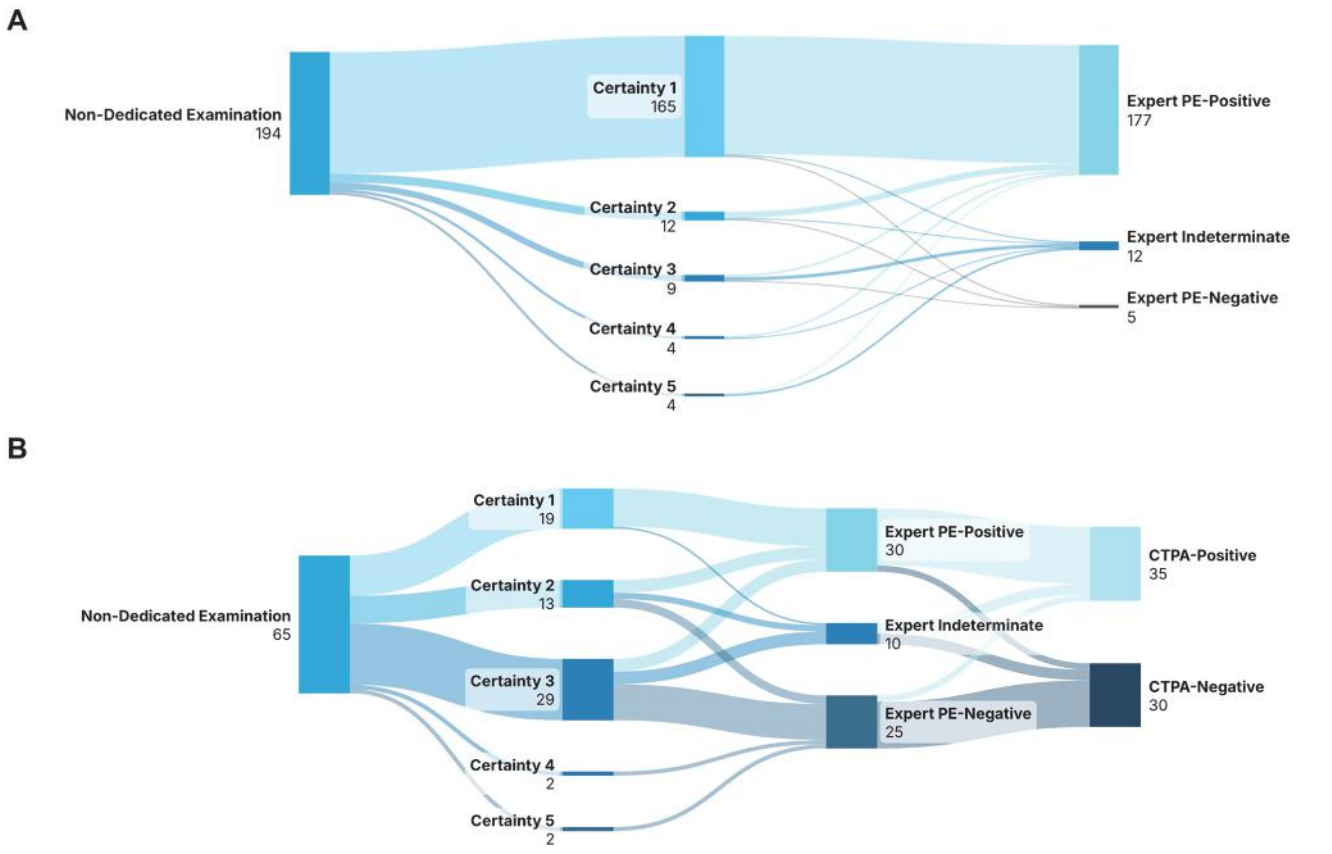
**Hypothesis** Expert review could reduce the requirement for CTPA in confirming iPE.

**Methods** We conducted single-centre retrospective analysis of 25,214 consecutive non-CTPA contrast-enhanced CT chest studies from over an 18-month period (01/12/2022–31/05/2024). Text string search of the initial radiology reports was used to identify iPE. Reports were classified by diagnostic certainty (levels 1 to 5) for iPE according to reported radiologist opinion and pertinent clinical data collected.

An expert review of non-dedicated scans was performed by fellowship-trained thoracic subspecialists (HS, JCLR) blinded to confirmatory imaging. Subgroup analysis was performed for those receiving layered CTPA testing to validate iPE diagnosis.




**Abstract P279 Figure 1**




**Abstract P280 Figure 1** **(A)** Sankey diagram showing original diagnostic certainty of non-dedicated CT identifying iPE not receiving CTPA follow-up and the subsequent expert of diagnoses. **(B)** Sankey diagram showing original diagnostic certainty of non-dedicated CT identifying iPE receiving CTPA follow-up and subsequent expert and CTPA classification of these diagnoses. Initial radiology reports identifying iPE were classified into 5 certainty levels on the basis of report language compared with published literature on reporting diagnostic certainty

**Results** iPE was reported in 1%(n=260/25,214) of non-dedicated studies (51% female, 73±12 years). 25%(n=65/260) received confirmatory CTPA. 54%(n=35/65) of iPE were confirmed on CTPA. This indicates that 12%(n=30/259) were treated for false-positive iPE. Review reclassified 8%(n=22/259) of studies as indeterminate and requiring CTPA. Agreement between the expert review of the iPE scan and the ground-truth confirmatory CTPA diagnosis had a κ statistic of 0.78.

False-positive studies reported lower diagnostic certainty than true-positives ($p<0.001$). Expert review of the subgroup receiving CTPA reclassified 37%, predicted CTPA ground-truth in 72% and confirmed artefact for 32%. Where the reporter was confident of iPE diagnosis, 93%(n=194/209) were true-positives according to expert review, where unconfident this was 26%(n=13/50).

Mortality was high in this iPE group, primarily reflecting the underlying indication (predominantly metastatic malignancy) for the non-CTPA CT chest.

**Conclusions** iPE was reported in 1% of scans, with expert review confirming this for 80%. Diagnostic certainty in initial reports can stratify the likelihood of true-positive iPE, guiding targeted expert review to minimise unnecessary CTPA and treatment of false-positive iPE.

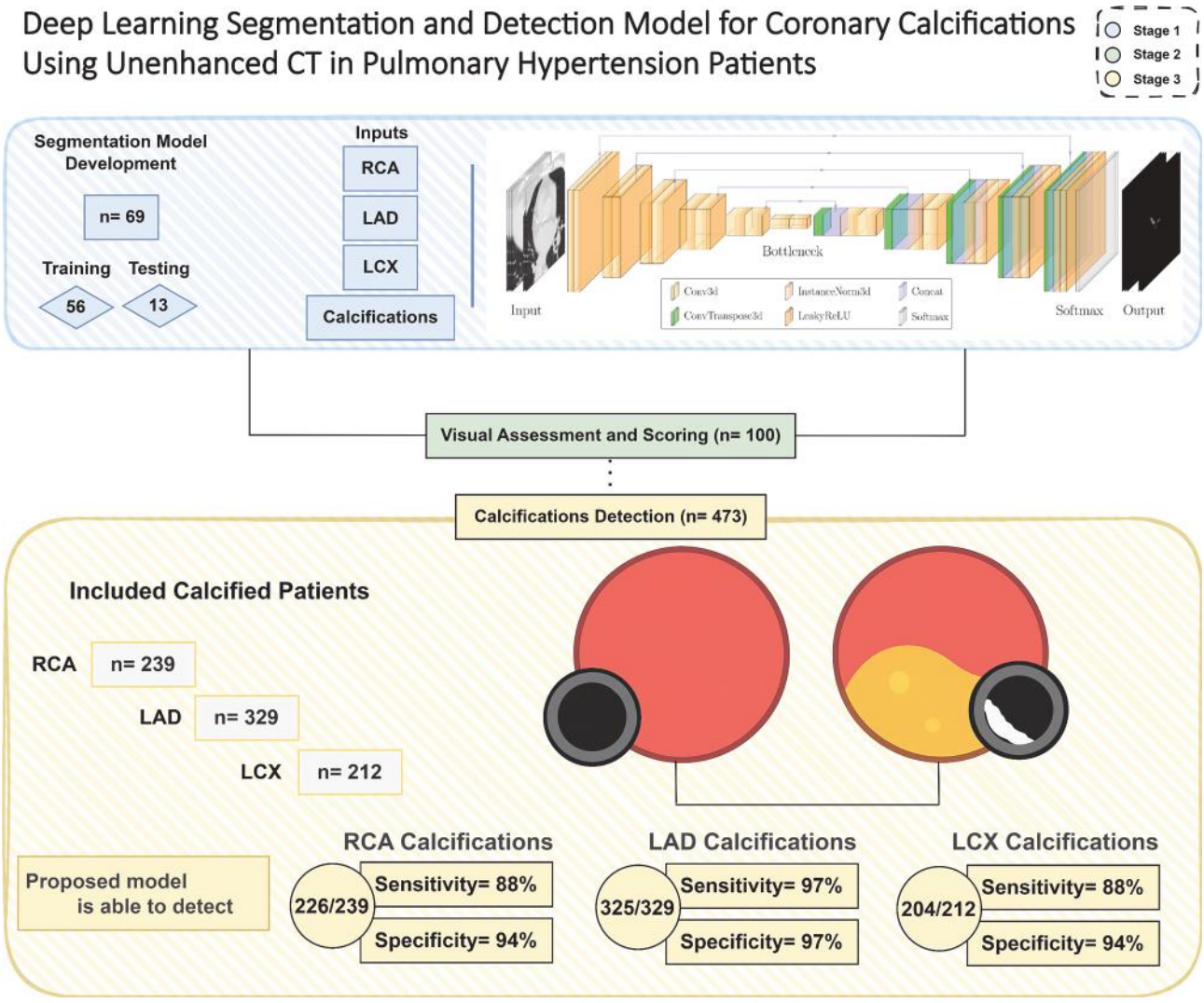
## P281 DIAGNOSTIC ACCURACY OF A DEEP LEARNING MODEL FOR CORONARY CALCIFICATION DETECTION ON UNENHANCED CT IN PULMONARY HYPERTENSION PATIENTS

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10.1136/thorax-2025-BTSabstracts.462

**Aim** To assess the diagnostic accuracy of a fully automated deep learning (DL) model in segmenting the coronary arteries and detecting the calcifications using unenhanced and ungated CT thorax scans in patients with pulmonary hypertension.

**Methods** A two-stage 3D segmentation pipeline was developed using 69 CT scans to identify coronary artery calcifications in the right coronary artery (RCA), left anterior descending artery (LAD), and left circumflex artery (LCX). The model




**Abstract P281 Figure 1** Graphical abstract including three stages: segmentation model development stage, visual assessment stage, and calcification detection and diagnostic accuracy stage

was trained with anatomically refined labels and region-based optimisation to improve structural coherence. Model outputs were visually assessed in a separate cohort of 100 scans by two independent, experienced observers. Segmentation and detection performance was evaluated against manually annotated ground truth using a binary analysis in 473 scans. Volumetric measurements of calcifications were compared with baseline severity gradings derived from an experienced radiologists' reports. Interobserver variability between DL and manual ground truth was calculated using Cohen's kappa and p-value.

**Results** The model was able to segment and detect the coronary arteries and calcifications with high performance. The majority of the scans were rated as excellent in the visual assessment with kappa values ranging from 0.68 to 0.81 for coronary artery segmentation and from 0.79 to 0.85 for calcification segmentation. The AI model accurately detected the presence of calcifications in the RCA ($\kappa = 0.82$, $p < 0.001$), LAD ($\kappa = 0.93$, $p < 0.001$), and LCX ($\kappa = 0.82$, $p < 0.001$). The model detected coronary calcifications with a sensitivity of 95%, specificity of 98%, positive predictive value of 99%, and negative predictive value of 88%. Calcification volume increased progressively with disease severity.

**Conclusion** The DL model trained on unenhanced and ungated CT scans accurately segmented and detected the presence of calcifications as well as the severity with high accuracy, highlighting its potential for future clinical practice in the detection of incidental coronary artery disease.

## P282 AUTOMATED THREE-DIMENSIONAL MEASUREMENTS OF MAXIMUM PULMONARY ARTERY DIAMETER AND RATIO WITH AORTA ON CT PULMONARY ANGIOGRAM ARE PREDICTIVE OF PULMONARY HYPERTENSION IN PATIENTS WITH LOW PROBABILITY ECHOCARDIOGRAPHY

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**Introduction and Objectives** Main pulmonary artery diameter (MPA) and MPA to aorta diameter ratio (MPA:Ao) on CTPA are recognised imaging biomarkers of pulmonary hypertension (PH). Manual two-dimensional (2D) measurements may not capture true MPA in three-dimensional (3D) space; they are also prone to inter-observer error and are not reliably reported in clinical practice.[1]

We developed and evaluated an automated CTPA analysis pipeline for 3D MPA and MPA:Ao extraction to predict PH at right heart catheterisation (RHC) versus manual 2D measurements. Hypothetical clinical impact was assessed in a cohort with chronic thrombo-embolic pulmonary disease (CTEPD) and low echocardiographic probability of PH.[2]

**Methods** Retrospective study in 284 consecutive CTPAs within 6 months of diagnostic RHC (age 63±14 years, 39% male (111/284)).

The pipeline was trained (n=95, 37372 slices) and validated (n=24, 9696 slices) using manually labelled segmentations. Optimal thresholds for PH at gold-standard RHC for 2D and 3D measurements were derived (n=70, 27613 slices) and tested (n=95, 35232 slices).

Theoretical clinical impact was assessed in a consecutive CTEPD cohort (n=50, age 58±17, 56% male (28/50)). We evaluated reclassification of PH risk pre-RHC using 3D measurements.

**Results** Automated 3D measurements were feasible in 98% (278/284) and correlated with manual 2D measurements ($R^2$=0.82).

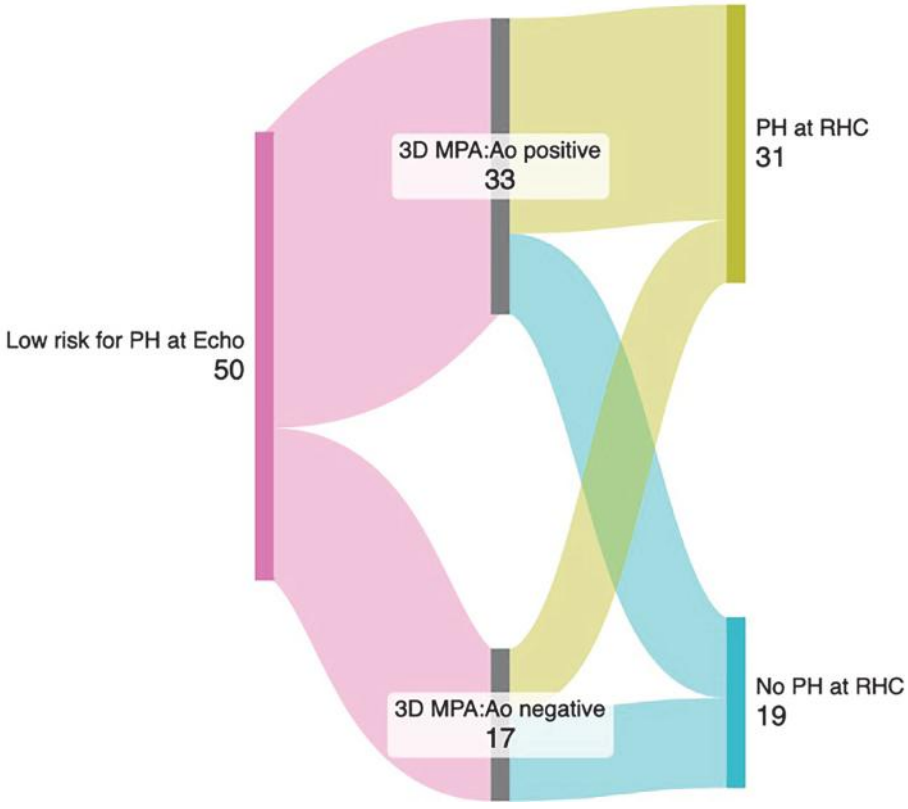
Automated 3D MPA:Ao correctly identified significantly more patients with PH in the test cohort (90% (55/61) vs. 71% (43/61) $X^2$=16.5, p<0.05).

In the CTEPD cohort, MPA:Ao was recorded in only 12% (6/50) of contemporaneous routine clinical reports. 62% (31/50) had PH at RHC. 3D MPA:Ao correctly predicted PH in 77% (24/31) with a specificity of 53% (see figure 1).

**Conclusions** Automated 3D measurements from CTPA are feasible, more reproducible than routine clinical practice and 3D MPA:Ao has the best diagnostic performance. In a low probability echocardiography cohort, automated 3D measurements identified additional cases of PH in theoretical pre-RHC risk stratification.

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**Abstract P282 Figure 1** Sankey Diagram demonstrating re-classification of pulmonary hypertension risk pre-right heart catheterisation using automated 3D measurements on CTPA in a cohort of patients with chronic thrombo-embolic pulmonary disease and low probability of pulmonary hypertension on echocardiography by ESC/ERS guidelines

## P283 PULMONARY HYPERTENSION ASSOCIATED WITH COVID-19-INTERSTITIAL LUNG DISEASE

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**Introduction** Post COVID interstitial lung disease (ILD) can occur following COVID-19 (Myall et al, 2021). The prevalence of pulmonary hypertension (PH) due to post-COVID ILD in a US cohort referred for lung transplantation was 27% (Thomas et al, 2023); this has not been reported in the UK. We describe consecutive patients with right-heart catheterisation (RHC)-confirmed PH due to post-COVID ILD, referred from a large area to the Royal Brompton PH Service from 2020–2025.

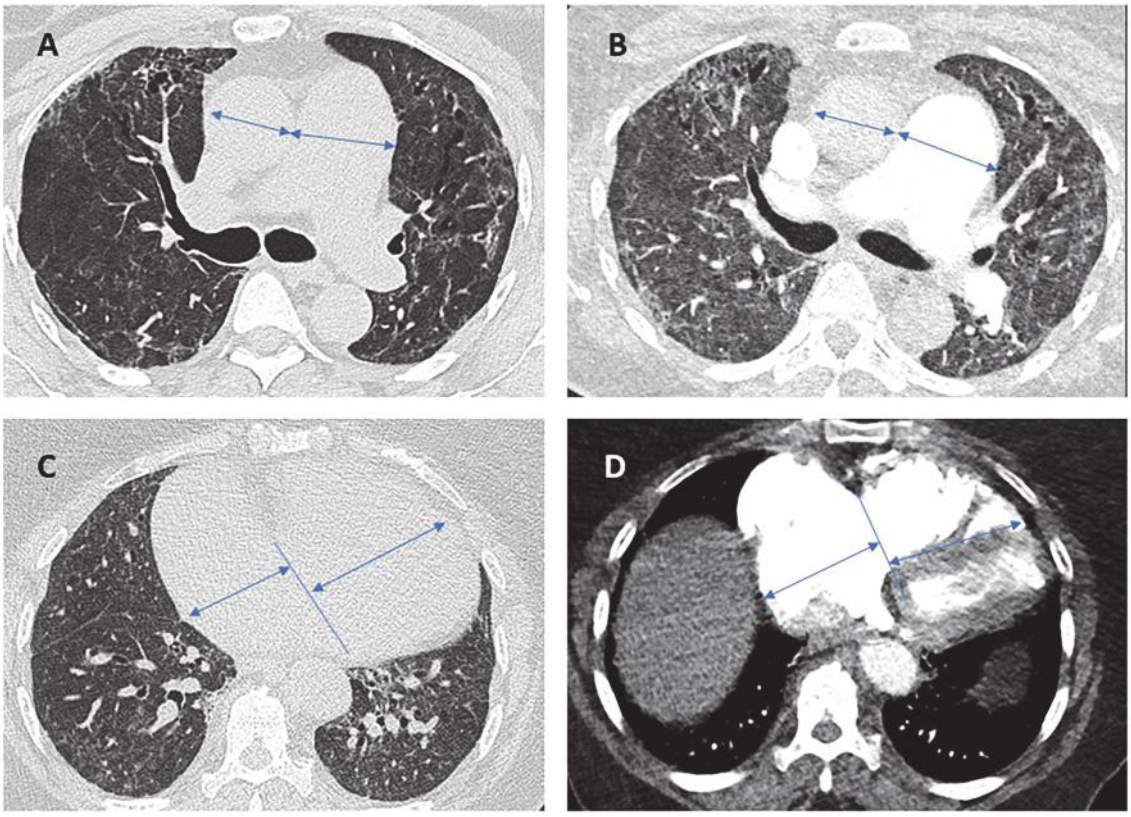
**Methods** PH was defined at RHC as mPAP>20 mmHg and a PVR>2 Wood units (WU). Clinical details were recorded at PH diagnosis and first follow-up (3–6 months). Other causes of ILD including CTD-ILD were excluded. Data are mean+-SD and median (IQ range) unless specified. Registry ethics was approved (RSID 195474).

**Results** Patients (n=10) were male (n=6), age 62 +-10 y at PH diagnosis, ex-smokers (n=5), mostly WHO III (n=8), 6MW 137 (60–202) m. Patients were diagnosed with ILD-PH at median 2.4 (1.1–4.4) years after their initial COVID illness. All had undergone invasive ventilation on ICU for 8 (6–24) weeks, one (who later died) needed ECMO. None had pre-existing lung disease.

BNP was median (range) 99 (65–539) ng/L. RAP was 4 (3–11) mmHg, mPAP 34+-11 mmHg, PCWP 9.4 +- 6 mmHg, CO 3.8 (2.7–4.5) l/min and PVR 7.0 (4.7–10.7) WU. FVC was 78 (54–87)%, TLCO 33 (23–46)%, KCO 55 (45–57)% predicted. PVR was over 5 WU in 7 (78%). CT patterns included organising pneumonia, with elevated PA:aortic ratio at 1.07 +- 0.19 and RV:LV ratio in all subjects at 1.2+-0.08 (figure 1A-D).

Following ILD-PH diagnosis, 4 patients were treated with sildenafil. At median follow up 1.5 (0.4–2.5) years from PH diagnosis, there have been 2/10 deaths and no lung transplantations so far.

**Conclusion** Post Covid-ILD-PH was only seen in patients after prolonged invasive mechanical ventilation and is likely to be a rare complication of COVID-19. All patients had elevated BNP, and RV:LV and/or PA:aortic ratio on unenhanced CT scans: these non-invasive screening markers should prompt referral for echocardiography and onwards to PH centres.




**Abstract P283 Figure 1**

## P284 INTERIM RESULTS FROM PHINDER: PULMONARY HYPERTENSION SCREENING IN PATIENTS WITH INTERSTITIAL LUNG DISEASE

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**Introduction** Pulmonary hypertension (PH) in patients with interstitial lung disease (ILD) is associated with increased morbidity and mortality. Current guidelines lack an evidence-based approach for detection of PH in the setting of ILD. The PHINDER study (NCT05776225) aims to identify clinical features associated with PH in patients with ILD to develop a diagnostic risk tool.

**Methods** PHINDER is a multicenter, prospective study enrolling 300 ILD patients with features suggestive of PH. Participants underwent routine assessments (physical exam, pulmonary function tests, high-resolution computed tomography, echocardiogram) followed by right heart catheterization (RHC). An interim analysis conducted in January 2025 used descriptive statistics to summarize baseline features. Univariate logistics regression analysis was performed to assess predictors for PH, defined as mPAP >20 mmHg, PVR >2 Wood Units (WU), and PAWP ≤ 15 mmHg.

**Results** Of the 143 patients enrolled, 128 underwent RHC. Participants were primarily white (82%), male (56%) with mean age 70±13 years and mean time from ILD diagnosis 4.1±4.2 years. Of these, 66 (52%) had PH-ILD with mPAP 28.3±5.3 mmHg and PVR 4.3±1.9 WU. Findings on lung function (DLCO, DLCO%,% predicted FVC/% predicted DLCO), serum creatinine, echocardiography (TAPSE/PASP), and CT (pulmonary artery enlargement, pulmonary artery/aorta diameter, and RV/LV diameter >1) were associated with a diagnosis of PH.

**Discussion/Conclusion** We identified several features from commonly used clinical assessments that were predictive of PH in ILD. Results from the fully enrolled study will be used to develop a multicomponent screening tool to aid in detection of PH in ILD.

## P285 CHANGE IN RIGHT VENTRICULAR EJECTION FRACTION IN GROUP 3 PULMONARY HYPERTENSION HAS PROGNOSTIC VALUE: INSIGHTS FROM THE ASPIRE REGISTRY

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10.1136/thorax-2025-BTSabstracts.466

**Background** Right ventricular function as assessed by cardiac magnetic resonance (CMR) has utility in the monitoring of patients with pulmonary arterial hypertension.[1] Whether changes in right ventricular function have prognostic value in group 3 pulmonary hypertension is not known.

**Methods** Consecutive patients from the ASPIRE registry with group 3 PH diagnosed between 2009 and 2023 were included. Patients underwent a comprehensive assessment at baseline including pulmonary function and shuttle walk testing, emPHasis10, CMR and right heart catheterisation. Follow up assessment was carried out with repeat CMR after initiation of treatment. Prognostic value was assessed using univariable then stepwise multivariable Cox regression analyses.

**Results** Follow-up CMR after initiation of pulmonary vasodilator treatment was available in 112 patients, 59% female with a mean age 64 (SD 11) years; 45% had COPD/emphysema, 42% ILD, 12% CPFE, and 13% non-parenchymal restriction. There were 35 deaths with a median survival of 32 months. The baseline RVEF was 36.04% (SD 11.1) and mean DRVEF was 2.14%. Predictive variables from univariate analysis were incorporated into a multivariable model and refined. Age (HR 1.02, 95% CI 1.00–1.04 p=0.034), mean pulmonary artery wedge pressure (HR 0.94 95% CI 0.89–0.99 p=0.014), DRVEF (HR 0.98, 95% CI 0.96–0.99, p = 0.009), and TLCO (HR 0.77, 95%CI 0.64–0.93 p=0.008) were independently associated with survival.

**Conclusions** Change in RVEF is a strong, independent predictor of mortality in group 3 PH. Further exploration of CMR parameters as a clinical outcome measure in group 3 PH is warranted.

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## P286 IS THERE DIURNAL OR SEASONAL VARIATION IN THE 6-MINUTE WALK TEST DISTANCE IN PATIENTS WITH PULMONARY VASCULAR DISEASE?

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10.1136/thorax-2025-BTSabstracts.467

**Introduction** The 2002 American Thoracic Society (ATS) 6-Minute Walk Test (6MWT) guidelines advise '*repeat testing should be performed about the same time of day to minimize intraday variability*'. It is currently unknown if there is diurnal and/or seasonal variation in the 6MWT in pulmonary vascular disease (PVD).

**Aims** To investigate the association between 6-Minute Walk Distance (6MWD), time of day and season of testing.

**Method** Retrospective 6MWT data was collected from 2019–2025. The 6MWT was conducted in accordance with ATS 6MWT guidelines (2002). Independent sample t-test was performed to determine morning vs afternoon differences in 6MWD. One way ANOVA was performed to determine seasonal differences. Time of day and seasonal effects were further investigated using multivariate linear regression analyses, adjusted for age, sex, BMI and WHO functional class. Time was modelled as a continuous variable (per 1 hour increment) and as a binary predictor of morning (08:00–12:00 am) vs afternoon (12–18:00 pm).

**Results** The cohort consisted of 1618 patients, WHO PH Groups 1 (n= 322), 2 (n=72), 3 (n= 51), 4 (n = 946), and

chronic thromboembolic pulmonary disease (n= 227). 6MWD was significantly higher in the morning (363 ± 134 meters; n=684) than in the afternoon (333 ± 144 meters; n=865) (p<0.001). 6MWD was not significantly impacted by season (p=0.75). Multivariable linear regression analyses demonstrated that the 6MWD decreased by 4.1 meters (95% CI 1.57–6.64) (p=0.002) for every hour increment in the time of the day, and by 16.34 meters (95% CI 5.72–29.95) (p=0.0003) in the afternoon compared to the morning.

**Conclusion** The time of day was found to impact 6MWD, with patients with PVD achieving higher 6MWD in the morning. 6MWTs should be conducted at the same time to eliminate this possible fatiguing effect and optimise interpretation of intertest differences in clinical practice and trials. Prospective studies are needed with repeated measurements in the same individuals at different times of the day to validate these findings.

## P287 INTRAPULMONARY SHUNT DETECTION IN HHT: STANDARDIZED TECHNIQUE FOR AGITATED SALINE (BUBBLE ECHO) STUDIES

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**Introduction and Objectives** Intrapulmonary right-to-left shunts (RLS) are an important clinical concern in individuals with Hereditary Hemorrhagic Telangiectasia (HHT). Along with respiratory symptoms like dyspnea, serious neurological complications can arise including ischemic strokes and brain abscess. HHT is an autosomal dominant genetic disorder characterized by arteriovenous malformations (AVMs), with pulmonary AVMs (PAVMs) being a common manifestation that

**Abstract P287 Table 1** Comparison of shunt assessment protocols across accredited echo labs

| Lab | Injection Volumes | Injection Method | Grade Scale |
|---|---|---|---|
| Facility 1 | 9.9 mL saline; 0.1 mL air Back extrapolation of blood if possible | 3-way stopcock; in 4 chamber view | Semi-quantitative **0-4** scale; **ventricular** opacification assessed |
| Facility 2 | 9 mL saline; 0.5-1 mL air or 8 mL saline; 1 mL air; 1 mL blood | 4-way stopcock; in 4 chamber view | **0-3 scale:** Small (1-5 bubbles), moderate (6-29 bubbles), or large (≥ 30 bubbles) |
| Facility 3 | 9 mL saline; 0.5-1 mL air or 8 mL saline; 1 mL air; 1 mL blood | 4-way stopcock; in 4 chamber view | **0-3 scale:** Small (1-5 bubbles), moderate (6-29 bubbles), or large (≥ 30 bubbles) |
| Facility 4 | 9 mL saline; 0.5-1 mL air or 8 mL saline; 1 mL air; 1 mL blood | 4-way stopcock; in 4 chamber view | **0-3 scale:** Small (1-5 bubbles), moderate (6-29 bubbles), or large (≥ 30 bubbles) |
| Facility 5 | 9 mL saline; 0.5-1 mL air or 8 mL saline; 1 mL air; 1 mL blood | 4-way stopcock; in 4 chamber view | **0-3 scale:** Small (1-5 bubbles), moderate (6-29 bubbles), or large (≥ 30 bubbles) |
| Facility 6 | 10 mL saline | 3-way stopcock; in 4 chamber view | Semi-quantitative **0-4** scale; **atrial** opacification assessed |
| Facility 7 | 8-9 mL saline; 0.5-1 mL air; 0.5-1 mL blood | 3-way stopcock; in 4 chamber view | **Positive** if bubbles are present & **negative** if no bubbles are detected |
| Facility 8 | 9.9 mL saline; 0.1 mL air Back extrapolation of blood if possible | 3-way stopcock; in 4 chamber view | Semi-quantitative **0-4** scale; **ventricular** opacification assessed |
| Facility 9 | These labs are not participating in this study | | |
| Facility 10 | | | |
| Facility 11 | | | |
| Facility 12 | Currently no specific policy in place for bubble studies | | |

contributes to intrapulmonary shunting. Screening for PAVMs is important in HHT to prevent risks of paradoxical embolization, appropriate management and rarely death from pulmonary hemorrhage. Agitated saline contrast echocardiography ('bubble echo studies') are done through HHT Centers to identify RLS and grading the same when present, and are recommended through the International HHT Guidelines. Western Canada has a large rural/remote population, and therefore satellite HHT clinics by health jurisdiction and associated echo labs performing these studies have been recommended to improve access to care and hopefully maintain quality of RLS assessments. This policy study aimed to assess the written policies governing bubble echo techniques and protocols in use within accredited echo labs across the province of Alberta, Canada.

**Methods** We conducted an observational cross-sectional study at a single time point (as of July 15, 2024) to evaluate the practices and protocols used in echocardiographic assessments for RLS to screen individuals with HHT - working with the provincial accreditation body, the College of Physicians and Surgeons of Alberta (CPSA). All CPSA-accredited adult echo lab facilities were contacted (telephone/email/in-person). Policy data was collected on the proportion of CPSA-accredited labs specifically performing shunt assessments, including review of available written policies for the shunt assessment protocols.

**Results** A total of 62 CPSA-accredited adult echocardiography facilities were reviewed, of which only 12 reported performing bubble echo studies for RLS assessment. The policy review of the participating echo labs revealed notable variations in the saline injection techniques and the grading scales/criteria used for shunt evaluation in bubble echo studies.

**Conclusion** Current accreditation criteria in Alberta and other health jurisdictions do not mandate a standardized protocol for bubble echo shunt assessments. Considering a standardized protocol could reduce variability between labs and allow for more reliable comparisons over time.

### P288 RISK FACTORS FOR RECANALISATION FOLLOWING PULMONARY ARTERIOVENOUS MALFORMATION EMBOLISATION: RESULTS FROM A TERTIARY REFERRAL CENTRE

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10.1136/thorax-2025-BTSabstracts.469

**Introduction and Objectives** Embolisation represents the primary therapeutic intervention for pulmonary arteriovenous malformations (PAVMs); however, post-procedural recanalisation remains a recognised complication. This study aimed to identify potential risk factors associated with recanalisation following PAVM embolisation at a tertiary referral centre.

**Methods** A retrospective, single-centre, observational analysis was conducted on 40 patients with PAVMs embolised. Demographic variables, angiographic measurements (supplying artery, vein, sac diameters), embolisation modalities including coils, Amplatzer vascular plugs (AVPs), microvascular plugs (MVPs), or combinations, and the presence of recanalisation (defined as <70% reduction in size on follow-up CT angiography) were collected. Univariate logistic regression analysis was performed.

**Results** A total of 109 pulmonary arteriovenous malformations (PAVMs) were embolised across 40 patients, with follow-up imaging available for 86 PAVMs. Recanalisation was significantly associated with male gender (odds ratio [OR] 3.52, p = 0.018), larger feeding artery diameter (OR 1.410, p = 0.008), greater sac diameter (OR 1.052, p = 0.022), and complex PAVM morphology (OR 5.464, p = 0.002). Although not reaching statistical significance, there was a trend toward higher recanalisation rates in lesions treated with coil embolisation (OR 1.926, p = 0.166). Embolisation with vascular plugs was associated with less risk of recanalisation but was insignificant (AVP [OR 0.782, p = 0.654], MVP [OR 0.342, p = 0.078]).

**Conclusions** Male gender, larger feeding artery diameter, sac dimensions, and complex PAVM morphology were associated with an increased risk of recanalisation. Coil-only embolisation demonstrated a non-significant trend toward higher recanalisation rates. Larger, prospective studies are needed to validate these findings and optimise embolisation strategies.

## More than just inhalers in asthma

### P289 PATIENT AND CLINICIAN EXPERIENCES OF A FENO-GUIDED ASTHMA MANAGEMENT INTERVENTION IN PRIMARY CARE

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10.1136/thorax-2025-BTSabstracts.470

**Introduction** Asthma affects over four million people in the UK and patient outcomes remain suboptimal. An objective measure of airway inflammation may provide opportunities for better patient care. An intervention to support the use of fractional exhaled nitric oxide (FeNO) to optimise management in primary care asthma reviews was developed using the Person-Based Approach. The intervention includes an online training package, a web-based FeNO-guided algorithm, which generates management recommendations for clinicians, and a patient information booklet. A randomised-controlled trial of the intervention is expected to finish in late 2025. This study forms part of a nested process evaluation.

**Aim** Explore patient and clinician experiences of the intervention.

**Methods** Patients in the intervention arm and clinicians with a trial role were purposively sampled for in-depth interviews. Separate interview topic guides for clinicians and patients were informed by existing literature and refined during interviews. Audio-recordings were transcribed verbatim and analysed using inductive thematic analysis.

**Results** 27 clinicians participated. Clinicians accepted the FeNO test and intervention components as these could be incorporated into existing reviews. They found patients mostly accepted the test and management changes and clinicians reported seeing patient benefits. Clinicians felt the training and the personalised recommendation from the FeNO-guided management algorithm supported shared decision-making but their judgement remained important in considering patient history, thus being able to override recommendations was important.

27 patients participated and were largely accepting of FeNO-testing and management changes, when explained. When reasons for FeNO testing, results or management were not clearly communicated, patients described frustration and uncertainty. Patients highlighted the benefits of FeNO-guided management when their FeNO result was surprisingly high and did not align with their perception of asthma control and when significant changes in management, such as switching from fixed dose regimens to maintenance and reliever therapy, were suggested. Patients liked use of FeNO to monitor changes in inflammation and assess the impact of management changes.

**Conclusions** Patients and clinicians report high levels of intervention acceptability, because the intervention could be incorporated into asthma reviews and most patients could perform the test. When explained clearly by clinicians, FeNO results supported shared management decisions.

## P290 FEASIBILITY OF A DIGITAL-FIRST APPROACH TO INHALER OPTIMISATION IN ASTHMA CARE: SUPPORTING NET ZERO NHS GOALS

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10.1136/thorax-2025-BTSabstracts.471

**Background** The carbon footprint of asthma care in the UK is largely driven by metered-dose inhalers (MDIs). National policy encourages switching to dry powder inhalers (DPIs), which offer similar outcomes with significantly lower environmental impact. However, limited general practice capacity constrains opportunities for structured face-to-face medicines optimisation. Eclipse, a population health platform, offers a scalable solution to identify eligible patients and deliver targeted, digital-first interventions.

**Aims** To evaluate the feasibility and patient engagement of a digital-first, population health-led approach to inhaler optimisation, supporting low-risk asthma patients to switch from Fostair 100/6 MDI to Fobumix Easyhaler DPI.

**Methods** Eligible patients were identified using the Eclipse population health platform across 15 consenting practices in LLR ICB. Inclusion criteria were patients aged $\geq$18, coded with asthma, and prescribed Fostair 100/6 MDI. Exclusions included respiratory specialist care, a respiratory admission in the past 12 months, care home residency, or palliative/dementia codes.

Patients were contacted centrally via SMS linking to a tailored information page and questionnaire assessing willingness to switch and baseline Asthma Control Test (ACT) score. Non-responders were followed up by telephone.

Based on responses and ACT scores, patients were triaged within Eclipse for remote or in-practice follow-up. Those opting to switch were referred to their community pharmacy for New Medicine Service follow-up.

**Results** Of 709 eligible patients, 689 (97.2%) successfully received an SMS. Among these, 412 accessed the digital information page, and 123 completed the questionnaire online. A further 441 completed it via telephone, resulting in 564 total responses. Of these, 281 (49.8%) opted to switch, 50 (8.9%) requested more information, and 206 (36.5%) declined. Demographic breakdown is presented in table 1.

**Conclusions** This pilot demonstrates that a digital-first approach to inhaler optimisation is feasible and well accepted, with telephone hub support playing a key role in maximising patient engagement. The high response rate, particularly through follow-up calls, underscores the value of combining digital outreach with personalised support. Strong uptake and willingness to switch highlight the model's potential for scalable implementation across primary care. It supports environmentally sustainable prescribing in line with NHS Net Zero priorities, while preserving clinical safety and patient choice.

**Abstract P290 Table 1** Demographic Breakdown of Patients Engaged in the Digital-First Inhaler Optimisation Pilot

| Demographic | Total eligible patients | Respondents who opted to switch | Respondents who declined the switch |
|---|---|---|---|
| Total Patients | 709 | 281 (40%) | 206 (29%) |
| Age 18-40 | 147 | 44 (30%) | 50 (34%) |
| Age 40-60 | 267 | 111 (42%) | 69 (26%) |
| Age Over 60 | 295 | 126 (43%) | 87 (29%) |
| Male | 302 | 126 (42%) | 94 (31%) |
| Female | 407 | 155 (38%) | 112 (28%) |
| Ethnicity Asian | 47 | 14 (30%) | 17 (36%) |
| Ethnicity Black | 6 | 2 (33%) | 2 (33%) |
| Ethnicity White | 441 | 182 (41%) | 131 (30%) |
| Other Ethnicity Groups | 215 | 83 (39%) | 56 (26%) |

## P291 CLINICAL EFFECTIVENESS OF ANTI-INFLAMMATORY RELIEVER THERAPIES (AIR) FOR EXACERBATION PREVENTION: A NON-INFERIORITY STUDY

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Current guidelines recommend anti-inflammatory reliever (AIR) therapies that combine inhaled corticosteroids (ICS) and formoterol for newly diagnosed asthma and patients treated with SABA only. Budesonide-formoterol (BUD-F) is the only combination licensed for as-needed AIR in the UK; however, beclometasone-formoterol (BDP-F) may be used as an alternative in the real-world setting. We conducted a non-inferiority study (pre-specified 20% non-inferiority margin; IRR 1.20) for exacerbation prevention of BDP-F (Fostair) AIR compared with BUD-F (Symbicort) AIR. We compared exacerbations pre- and post-AIR initiation, oral corticosteroid (OCS) use, and risk of acute cardiovascular (CVD) events.

A historical cohort study using the Optimum Patient Care Research Database, UK. Adults with asthma, no other chronic respiratory condition, and ≥1 year GP registration who initiated BDP-F AIR or BUD-F AIR ('likely intent' based on prescription instructions and ≤1 SABA inhaler at follow-up) for the first time (index date) after July 2012 were followed up for ≤1 year. Inverse probability treatment weighting (IPTW), adjustment and prior event rate ratio (PERR) methods controlled for baseline differences between groups. Non-inferiority of exacerbations used Poisson regression (incident rate ratios [IRR] with bootstrapped confidence intervals [95% CI]). Changes in OCS use and incidence of acute CVD events post-initiation of AIR were also compared.

Among 8,750 adult patients, 1,630 (18.6%) were initiated onto BDP-F AIR and 7,120 (81.4%) BUD-F AIR between Jul 2012 and Apr 2025. BDP-F AIR was found to be non-inferior to BUD-F AIR for exacerbation prevention: IRR=0.85 (0.61,1.16). The percentage of people with ≥1 exacerbation at baseline reduced by 7.0% after initiation of BDP-F AIR (relative reduction: 67.5% [57.1,79.8]; p<0.001) and 3.3% after initiation of BUD-F AIR (relative reduction: 76.9% [66.2,88.9]; p<0.001). OCS use reduced with both AIR regimens, but the reduction was significantly greater in the BDP-F AIR group (0.24 mg vs 0.05 mg; p<0.001). Incidence of CVD events post-initiation of AIR was similar for both groups: IRR=1.04 for BDP-F vs BUD-F [0.46,2.34]).

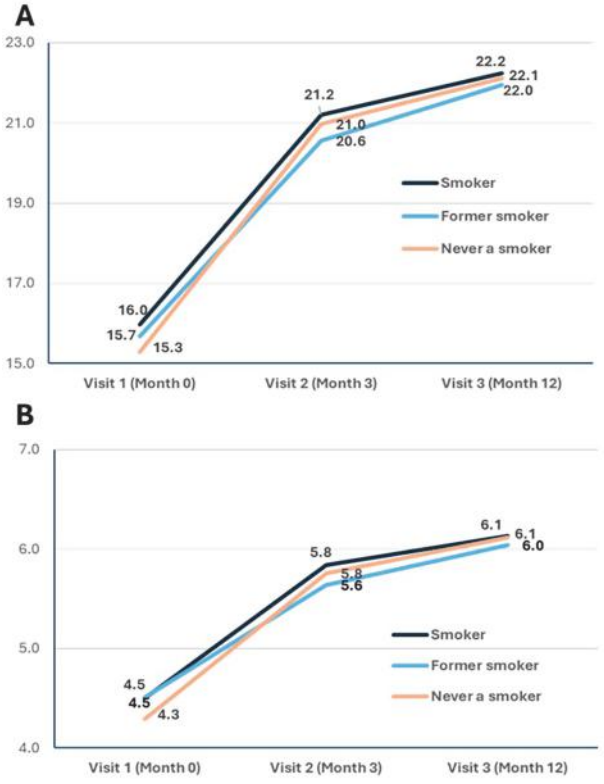
BDP-F AIR is non-inferior to BUD-F AIR for exacerbation prevention, results in significantly lower OCS use, and has comparable outcomes for acute cardiovascular events.

## P292 EFFECTIVENESS OF EXTRAFINE SINGLE-INHALER TRIPLE THERAPY ON ASTHMA CONTROL AND QUALITY OF LIFE ONE YEAR POST TREATMENT ACCORDING TO PATIENTS' SMOKING STATUS: THE TRIANGLE STUDY

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**Introduction** The efficacy of extrafine single-inhaler triple therapy (efSITT), with beclomethasone dipropionate/formoterol fumarate/glycopyrronium (BDP/FF/G) in asthma has been assessed in randomized controlled trials, excluding though both current and ex-smokers with a smoking history of ≥ 10




**Abstract P292 Figure 1** Mean of the total ACT score across the three study visits, by smoking status. The ACT score ranges from 5 (worst status) to 25 (complete control). Base: Number of patients at each visit for whom ACT was recorded (N=331, 331 and 301 respectively for each visit), p<0.05 for all comparisons. **Figure1B**: Mean value of the total AQLQ (S) score at the three study visits, by smoking status. The AQLQ (S) score ranges from 1 (maximal impairment) to 7 (no impairment). Base: Number of patients at each visit, with recorded AQLQ (S) (N=333, 331 and 301 respectively for each visit), p<0.05 for all comparisons

pack years. The Triangle study provides real-world evidence of efSITT's effectiveness in asthma patients in Greece.

**Aims and objectives** This observational study aims to assess therapy pathways and health-related outcomes of BDP/FF/G in moderate-to-severe asthma patients, previously treated with medium dose ICS+LABA or open triple therapy, including patients across all smoking statuses.

**Methods** This interim analysis includes 333 patients who received treatment with BDP/FF/G (87/5/9 μg) for 48 weeks. The co-primary objectives are assessment of asthma control (ACT) and description of treatment pathways. Secondary objectives include assessment of health-related quality of life (HRQoL), evaluated by AQLQ (S). Results were stratified by patients' smoking status.

**Results** The mean age of participants was 56.9 years, with a mean BMI of 28.6 kg/m$^2$ and a mean asthma duration of 11.7 years since diagnosis. Prior to switching to efSITT, 81.6% of patients were on ICS+LABA and 18.4% on ICS +LABA+LAMA. The main reasons for switching included worsening of symptoms or need for treatment escalation (72.3%), exacerbations (34.3%), deterioration of lung function (31.3%), regimen simplification or poor compliance (17.6%), and adverse effects of previous treatment (2.7%).

Overall, 39.3% of patients were never smokers, 31.5% were current smokers and 29.1% ex-smokers with a history of 36.3 and 18.9 mean pack years, respectively. After 1 year of treatment, ACT score improved by more than 6 points in all groups (all $p<0.05$) (figure 1A). Additionally, ≥88% of patients in each group achieved the minimum clinically important difference (MCID) in ACT score (≥3-point increase).

At baseline, the mean (±SD) AQLQ (S) score in the overall population was 4.4±1.0. At the 12-month follow-up, the mean change from baseline exceeded 1.5 points in all groups ($p<0.05$), exceeding the MCID threshold of 0.5 points.

**Conclusions** Our real-world data suggest a significant improvement in asthma control and HRQoL after switching to BDP/FF/G regardless of smoking status, enhancing our understanding on efSITT's effectiveness in these understudied populations.

## P293 EXACERBATION AND RESCUE MEDICATION REDUCTION WITH EXTRAFINE FORMULATION SINGLE-INHALER TRIPLE THERAPY IN ADULT ASTHMA PATIENTS: OUTCOMES FROM THE TRIMAXIMIZE UK STUDY

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**Background** Randomised controlled trials have shown clinical benefits of extrafine formulation single-inhaler triple therapy (SITT) consisting of beclometasone/formoterol/glycopyrronium (BDP/FF/G) in asthma patients uncontrolled on a combination of an inhaled corticosteroid and long-acting beta-agonist with at least one exacerbation in the prior year,[1] yet there is limited evidence in a real-world setting.

**Methods** TriMaximize is a prospective, non-interventional, multicentre study describing patient-characteristics and therapy pathways in adults with moderate-to-severe asthma, treated with BDP/FF/G.

This data set describes the treatment impact with BDP/FF/G on moderate and severe exacerbation rates and rescue medication use from the UK cohort (NCT04902573).

**Results** Of 166 patients enrolled across 20 UK centres, 147 are included (19 excluded as inclusion criteria not fulfilled or missing core baseline data) in the full analysis set. Patients are adults diagnosed with asthma, able to consent, with/without concomitant COPD. Before enrolment, 106 (72.1%) patients were treated with ICS/LABA and 41 (27.9%) with ICS/LABA/LAMA (both fixed or free combination).

Patients at baseline had one or more exacerbations in the prior year (defined as moderate; 34.1%, severe; 32.6%, moderate and severe 33.3%) with a mean baseline moderate/severe exacerbation rate of 3.0/year. Mean rescue medication use at baseline was 11.2 puffs/week with 72.2% patients reporting an intake of rescue medication in the previous week. After twelve-months of treatment the mean change in moderate/severe exacerbation rate was -2.0/year ($p<0.0001$). Rescue medication use also significantly decreased (-4.6 puffs/week) compared to baseline ($p=0.0037$) along with a reduction in the proportion of patients reporting rescue medication use within the previous week (reduced to 63.6%).

**Conclusion** Significant decreases in both moderate/severe exacerbation rate and rescue medication use were observed twelve-months after switching from an ICS/LABA or ICS/LABA/LAMA (both fixed or free combination) to treatment with SITT BDP/FF/G in adult patients with moderate-to-severe asthma.

### REFERENCE

1. Virchow JC, *et al. Lancet*. 2019;**394**(10210):1737–49.

## P294 THE BETRI STUDY: PRELIMINARY INFORMATION ON CHARACTERISTICS OF PATIENTS WITH ASTHMA OR ASTHMA-COPD OVERLAP TREATED WITH MEDIUM-STRENGTH INHALED CORTICOSTEROID/LONG-ACTING BETA-AGONISTS (ICS/LABA) AND SWITCHING TO HIGH-STRENGTH ICS/LABA OR MEDIUM-STRENGTH BECLOMETHASONE/FORMOTEROL/GLYCOPYRRONIUM (BDP/FF/G)

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**Introduction/Aim** GINA guidelines 2024 currently recommend step-up to either ICS/LABA/LAMA or high dose ICS/LABA for patients whose asthma remains uncontrolled on medium dose ICS/LABA. However, there is a lack of real-world evidence on patient characteristics prior to the choice of treatment.

This study will generate further understanding on the patient characteristics of both populations in a RW setting.

**Method** Utilising UK patients' electronic medical records extracted from the Optimum Patient Care Research Database (2.9 million patients with asthma; mean follow-up of 11.4

years), this cohort study included adults diagnosed with asthma initiating msBDP/FF/G or hsICS/LABA on/after 1st January 2017 following ≥1 year of msICS/LABA use. Patients' baseline characteristics were described and stratified by msBDP/FF/G or hsICS/LABA usage.

**Results** 2,566 patients prescribed msBDP/FF/G and 15,113 patients prescribed hsICS/LABA were included, with similar sex distribution (59,0% vs 63,5% male), ethnicity distribution (white 88.9% vs 84.5, black 2.8% vs 4.9%, asian 0.2% vs 1.4%) and prevalence of allergy diagnosis (52.5% vs 53.6%). Patients prescribed msBDP/FF/G were older than those prescribed hsICS/LABA (mean±standard deviation: 65.7±14.0 vs 53.2±17.3 years), were more active smokers (30.6% vs 17.2%), and had higher prevalence of concomitant COPD diagnosis (44.0% vs 5.4%), and cardiovascular risk factors or diseases - hypertension (48.6% vs 31.6%), diabetes (24.5% vs 13.8%), ischaemic heart disease (24.9% vs 11.3%), heart failure (6.9% vs 2.7%), and chronic kidney disease (14.2% vs 7.3%) -, but less allergic rhinitis (21.7% vs 28.7%). In the year prior, patients prescribed msBDP/FF/G had lower percent predicted PEFR (median 63.1% vs 74.3%) and percent predicted FEV1 (median 65.0% vs 78.7%), two or more exacerbations (32.6% vs 19.0%), three or more SABA prescription (69.7% vs 58.4%) and also had higher cumulative oral corticosteroid doses (684.4±926.8 mg vs 533.4±865.8 mg).

Conclusion msBDP/FF/G was preferentially prescribed over hsICS/LABA to patients with asthma-COPD overlap, severe disease, and who were older and active smokers. Exacerbations, SABA, steroid burden and cardiovascular co-morbidities were also important drivers. Confounder-matched/adjusted comparisons of efficacy between msBDP/FF/G and hsICS/LABA are warranted.

## P295 TREATMENT WITH EXTRAFINE FORMULATION SINGLE-INHALER TRIPLE THERAPY IMPROVES HEALTH-RELATED QUALITY OF LIFE AND ASTHMA CONTROL – RESULTS FROM THE TRIMAXIMIZE UK STUDY

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10.1136/thorax-2025-BTSabstracts.476

**Background** The efficacy of an extrafine formulation single-inhaler triple therapy (SITT) consisting of beclometasone/formoterol/glycopyrronium (BDP/FF/G) in patients with asthma has been demonstrated in randomised controlled trials (RCTs),[1] yet there is limited evidence in real-world settings. TriMaximize is a real-world study of patients with moderate-to-severe asthma being treated with BDP/FF/G. Here we present the results from the UK cohort (NCT04902573).

**Methods** TriMaximize is a prospective, non-interventional and multicentre study describing patient-characteristics and therapy pathways in adult patients with moderate-to-severe asthma, treated with BDP/FF/G.

This twelve-month data set describes the treatment impact with BDP/FF/G on health-related Quality of Life (HRQoL) and asthma control, assessed by the Mini Asthma Quality of Life Questionnaire (Mini-AQLQ) and the Asthma Control Test (ACT), respectively.

**Results** Of 166 patients enrolled across 20 UK centres, 147 are included (19 excluded as inclusion criteria not fulfilled or missing core baseline data) in the full analysis set. Patients are adults with a diagnosis of asthma, able to consent, with/without concomitant COPD. Before enrolment, 106 (72.1%) patients were treated with ICS/LABA and 41 (27.9%) with ICS/LABA/LAMA (fixed or free combinations).

Treatment with BDP/FF/G for twelve months significantly improved HRQoL ($p < 0.001$) and asthma control ($p < 0.001$) (table 1), exceeding the minimal clinically important difference of 3 points for ACT and 0.5 points for Mini-AQLQ.

**Conclusion** Significant improvements in HRQoL and asthma control were observed over a twelve-month period with SITT BDP/FF/G, proving to be a valuable treatment option for adult patients with moderate-to-severe asthma.

### REFERENCE

1. Virchow JC, *et al. Lancet*. 2019;**394**(10210):1737–49.

**Abstract P295 Table 1** ACT and Mini-AQLQ scores at baseline and after twelve-months of treatment with SITT BDP/FF/G, in patients previously treated with ICS/LABA or ICS/LABA/LAMA (fixed or free combination). Changes from baseline were calculated using paired t-tests

| | ACT total score | | Mini-AQLQ total score | |
|---|---|---|---|---|
| | n | Mean (SD) | n | Mean (SD) |
| **Baseline** | 124 | 13.5 (4.9) | 103 | 3.8 (1.2) |
| **12 months** | 68 | 17.0 (5.4) | 53 | 4.8 (1.4) |
| **Change from baseline** | 62 | 4.6 (5.5) p<0.0001 | 46 | 1.1 (1.3) p<0.0001 |

## P296 PRESSURE METERED DOSE INHALERS (PMDI) VS. DRY POWER INHALERS (DPI) FOR BRONCHODILATION AND INHIBITING EXERCISE-INDUCED BRONCHOCONSTRICTION IN ATHLETES: A SYSTEMATIC REVIEW

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10.1136/thorax-2025-BTSabstracts.477

**Introduction** Exercise-induced bronchoconstriction (EIB) is commonly reported among athletic populations and can impair both respiratory health and athletic performance. Inhaled beta-2 agonists and glucocorticoids, delivered by pressurised Metered-Dose Inhaler (pMDI) or Dry Powder Inhaler (DPI), are widely used by athletes to manage symptoms. However, inhaler technique alongside inspiratory flow requirements may influence drug deposition and clinical effectiveness.

**Aim** The review aims to investigate pMDI compared to DPI in eliciting bronchodilation and inhibiting bronchoconstriction in athletes.

**Method** Studies were identified through a systematic search of MEDLINE, SPORTDiscus, and CINAHL databases via EBSCOhost. Inclusion criteria were peer-reviewed studies with placebo-controlled, randomised or crossover design. Eligible participants were classified as athletes or physically active individuals with a diagnosis with asthma, EIB, or related symptoms. Studies were required to investigate inhaled medication delivered via pMDIs, DPIs, or both. Risk of bias was assessed using Cochrane Risk of Bias tools. The review followed PRISMA reporting guidelines and registered on the PROSPERO database (registration number: CRD42024569378).

**Results** Total of 2,767 records identified, 209 full-text articles were assessed for eligibility. Fifteen studies were eligible, but only 13 unique datasets were included in the analysis. Average sample size was 12 participants (range 6–35).

Placebo groups displayed no significant bronchodilation across the included studies. Five studies investigating bronchodilator treatment administered via pMDI device, reported mean increase in $FEV_1$ from baseline ranging from 6% to 10%. For DPI-administered bronchodilators, four studies reported a mean increase ranging from 5% to 16%.

Eleven studies investigated the mean fall in $FEV_1$ following a bronchial challenge test. Among the six studies using pMDI administration, placebo groups showed a mean fall in $FEV_1$ ranged from -14% to -37%, whereas pMDI administered interventions reduced the mean fall in $FEV_1$ ranging from 0% to -17%. Similarly, five DPI studies found placebo groups experienced a mean fall in $FEV_1$ ranging from -10% to -29%, compared to DPI administered interventions attenuated the mean fall in $FEV_1$ ranging from -3% to -7%.

**Conclusion** Both pMDI and DPI devices demonstrated comparable bronchodilation and bronchoprotection in athletes. Results indicate that both devices are effective options for respiratory care in athletes.

## P297 BUDESONIDE/GLYCOPYRRONIUM/FORMOTEROL FUMARATE DIHYDRATE TRIPLE THERAPY FOR INADEQUATELY-CONTROLLED ASTHMA: KALOS AND LOGOS STUDY RESULTS

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A long-acting muscarinic antagonist (LAMA) may be added to medium-/high-dose inhaled corticosteroid (ICS)/long-acting $\beta_2$-agonist (LABA) therapy in patients with uncontrolled asthma. KALOS and LOGOS, two Phase 3, double-blind, double-dummy randomised studies, compared fixed-dose triple combination budesonide/glycopyrronium/formoterol fumarate dihydrate (BGF) with budesonide/formoterol fumarate dihydrate via metered-dose inhaler (MDI; BFF) and via pressurised MDI ([Symbicort®] BUD/FORM).

Participants (≥12 y) with inadequately-controlled asthma despite ICS/LABA use were randomised (pooled N=4311) to BGF 320/28.8/10 µg, BGF 320/14.4/10 µg, BFF 320/10 µg or BUD/FORM 320/9 µg (BID) for 24 to 52 weeks. Key lung function endpoints included change from baseline in morning pre-dose trough $FEV_1$ and $FEV_1$ $AUC_{0–3}$ over 24 weeks; Day 1 onset of action was also assessed. Pooled analyses across studies assessed severe exacerbation rate. Safety measures included AE monitoring.

BGF 320/28.8/10 µg improved lung function endpoints in each study, and in the pooled analysis BGF 320/28.8/10 µg reduced severe exacerbation rate vs combined ICS/LABA comparator arms (BFF+BUD/FORM; see table 1 for European regulatory approach). AE frequency was comparable across treatments in each study.

BGF 320/28.8/10 µg is superior to dual ICS/LABA in adults and adolescents with asthma inadequately-controlled by ICS/LABA.

**Abstract P297 Table 1** Summary of primary and secondary efficacy endpoint results (European regulatory approach)

| | KALOS[a] | LOGOS[b] |
|---|---|---|
| | BGF 320/28.8/10 µg vs BFF 320/10 µg + BUD/FORM 320/9 µg combined (BID) | |
| *Primary*[c]: Change from baseline in morning pre-dose trough $FEV_1$ (mL) over 24 weeks | | |
| Difference in LS means (95% CI); p-value | 56 (31, 82); <0.001 | 97 (70, 123); <0.001 |
| *Key secondary*[c]: Change from baseline in $FEV_1$ $AUC_{0-3}$ (mL) over 24 weeks | | |
| Difference in LS means (95% CI); p-value | 69 (44, 94); <0.001 | 112 (87, 138); <0.001 |
| | Compared within group at baseline and 5-minutes post dose | |
| *Secondary:* Change in $FEV_1$ (mL) at 5 minutes post-dose on Day 1 (onset of action) | | |
| LS means change from baseline (95% CI); p-value | 161 (145, 177); <0.001 | 180 (162, 197); <0.001 |
| | Pooled KALOS and LOGOS[d] | |
| | BGF 320/28.8/10 µg vs BFF 320/10 µg + BUD/FORM 320/9 µg combined (BID) | |
| *Primary pooled*[e]: Severe exacerbation rate | | |
| Rate ratio (95% CI); p-value | 0.86 (0.76, 0.97); 0.012 | |

[a]KALOS: Treated (BGF 320/28.8/10 µg, n=594; BFF, n=606; BUD/FORM, n=602); completed study (BGF 320/28.8/10 µg, n=541; BFF, n=560; BUD/FORM, n=551).
[b]LOGOS: Treated (BGF 320/28.8/10 µg, n=585; BFF, n=604; BUD/FORM, n=594); completed study (BGF 320/28.8/10 µg, n=535; BFF, n=540; BUD/FORM, n=544).
[c]Model included treatment, visit, prior ICS dose (medium vs high) and treatment-by-visit interaction as categorical covariates and baseline trough $FEV_1$ and percent reversibility as continuous covariates.
[d]Pooled analysis: Treated (BGF 320/28.8/10 µg, n=1179; BFF, n=1210; BUD/FORM, n=1196); completed study (BGF 320/28.8/10 µg, n=1076; BFF, n=1100; BUD/FORM, n=1095).
[e]Model adjusted for baseline trough $FEV_1$, percent reversibility, baseline severe asthma exacerbation history (0, 1, ≥2), prior ICS dose (medium vs high), region and study.

N=726 participants randomised and treated with BGF 320/14.4/10 µg; recruiting for this treatment arm stopped during the SARS-CoV-2 pandemic and it was deprioritised in the testing approach.

$AUC_{0-3}$, area under the curve from 0–3 hours; BFF, budesonide/formoterol fumarate dihydrate via metered-dose inhaler; BGF, budesonide/glycopyrronium/formoterol fumarate dihydrate via metered-dose inhaler; BID, twice daily; BUD/FORM, budesonide/formoterol fumarate dihydrate via pressurised metered-dose inhaler; CI, confidence interval; $FEV_1$, forced expiratory volume in 1 second; ICS, inhaled corticosteroid; LS, least squares.

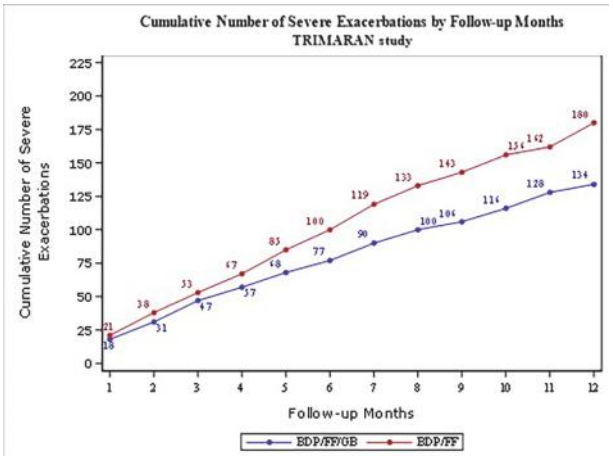
## P298 TIME COURSE OF EXACERBATION REDUCTION WITH EXTRAFINE BECLOMETHASONE DIPROPIONATE, FORMOTEROL FUMARATE, AND GLYCOPYRRONIUM BROMIDE (BDP/FF/GB) PMDI IN ADULTS WITH MODERATE TO SEVERE ASTHMA: A POST-HOC ANALYSIS OF THE TRIMARAN AND TRIGGER STUDIES

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**Rationale** Patients with asthma uncontrolled by medium to high dose ICS/LABA (GINA Steps 4 and 5) may benefit from the addition of a LAMA. The time to achieve a reduction in severe exacerbations is not well understood. TRIMARAN (Triple versus medium strength of ICS/LABA) and TRIGGER (Triple versus high strength ICS/LABA) studies tested the efficacy of extrafine BDP/FF/GB combination vs BDP/FF combination containing medium to high BDP dose.

**Methods** We conducted a *post-hoc* analysis of the TRIMARAN and TRIGGER studies [Virchow JC et al, Lancet 2019] in patients with asthma to assess the frequency of severe exacerbations every 4 months to 12-months. A generalised linear mixed model for repeated measures considering a Poisson distribution was used.




**Abstract P298 Figure 1**

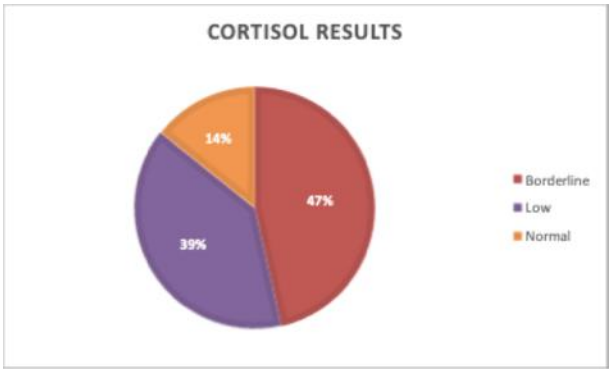
**Results** A total of 1149 and 1142 subjects in TRIMARAN and TRIGGER, respectively received BDP/FF/GB or BDP/FF. A reduction in severe exacerbations of 16.3% and 10.7% with BDP/FF/GB vs BDP/FF was observed in the first 4 months in TRIMARAN and TRIGGER, respectively (adjusted rate ratio [RR] of 0.837, 95% CI 0.572–1.231, p=0.3682 and RR of 0.893 95% CI 0.634–1.257, p=0.5148). The reduction in the subsequent 8 months (period 4–12 months) was of 30.5% and 24.1%, in TRIMARAN and TRIGGER, respectively (RR of 0.695, 95% CI 0.501–0.965, p=0.0296 and of 0.759, 95% CI 0.565–1.019, p=0.0663).
**Conclusions** A clinically relevant reduction in the number of severe asthma exacerbations can be seen as early as 4 months following step-up therapy to BDP/FF/GB. This reduction was progressive and sustained over the remainder of the 12-month treatment.

## P299 ADRENAL SUPPRESSION IN ASTHMA

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**Introduction** Inhaled and systemic corticosteroids are fundamental in asthma management, particularly in severe cases. Prolonged use can lead to adrenal suppression (AS), a potentially life-threatening condition that often remains undetected. Existing guidelines primarily focus on high-dose steroid exposure, potentially underestimating risk in patients on medium-dose inhaled corticosteroids (ICS).
**Objectives** This evaluation aimed to assess the prevalence of adrenal suppression in patients referred to a severe asthma clinic and identify associated risk factors.
**Methods** A retrospective review was performed using electronic health records from consecutive patients referred to the Severe Asthma Clinic from the Asthma and Allergy Clinic in the last 2 years at Wythenshawe Hospital. Data collected included ICS and oral corticosteroid (OCS) doses, additional steroid routes, morning serum cortisol levels, synacthen test outcomes, and relevant risk modifiers. ICS doses were standardised to beclomethasone dipropionate (BDP) equivalents, and adrenal function was assessed using NICE and local thresholds.
**Results** Of the 38 patients reviewed, 28 patients met inclusion criteria. 78.6% of the cohort was female (n=22) with a mean age of 47.3 years (range 27–69). 86% had abnormal morning cortisol results (see figure 1), yet only 7 underwent synacthen testing, of which 2 failed (29%). Most patients were on high-dose ICS (68%), and 57% had taken at least one OCS course within the last year. Notably, even among those on medium-dose ICS with no OCS use, abnormal cortisol results were common. Additional steroid exposure and enzyme inhibitors were common, indicating additive risk.
**Conclusion** Cortisol suppression is prevalent in this cohort and could be under-recognised in the severe asthma population, including those on medium-dose ICS. The findings suggest that current screening thresholds may be insufficient and highlight the importance of assessing cumulative steroid burden, including non-oral routes and interacting medications. Greater clinician awareness, routine biochemical screening in pre-selected high-risk individuals, and provision of steroid emergency cards are essential to reduce preventable morbidity. Future prospective studies are needed to assess the risk of adrenal suppression in patients on long-term medium and high dose ICS and inform national guidelines.




**Abstract P299 Figure 1** Distribution of morning cortisol results among the study cohort

## P300 EFFECT OF BASELINE ALVEOLAR GAS TRANSFER MEASUREMENT (KCO) ON RESPONSE TO BIOLOGICS IN SEVERE ASTHMA IN THE UK SEVERE ASTHMA REGISTRY

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**Introduction** Alveolar gas transfer measurements are known to be raised in many patients with asthma, potentially due to ventilation-perfusion mismatch. In contrast, in the other major airways disease, COPD, alveolar gas transfer is often reduced due to emphysema. Mucus plugging in airways disease can also negatively affect gas transfer. Differences in response to biologics have been noted between patients with asthma and COPD, although they are often clinically difficult to differentiate.
**Objective** To assess whether alveolar gas transfer, KCO, as measured at baseline, affects response to biologics in patients in the UK Severe Asthma Registry.
**Methods** Patients within the UK Severe Asthma Registry with recorded KCO at baseline and data on biologic response were identified. Mean KCO was compared between those patients achieving and those not achieving a positive composite response to biologics, a $\geq$ 50% reduction in oral steroid requiring exacerbations and/or in maintenance oral steroids. Characteristics and outcomes were also compared between patient subgroups stratified by KCO (<70%, 70–120%, >120%).
**Results** Baseline KCO was reported in 3586 patients in UKSAR. 216 patients had a KCO <70%, 2919 a KCO 70–120%, and 451 a KCO >120%. Comparing subgroups, with

increasing KCO there were significantly increased proportions of patients who had never smoked (KCO <70%, 31.0% never smoked; KCO 70–120%, 60.0%; KCO >120%, 67.4%; p<0.001) and who were male (KCO <70%, 34.3% male; KCO 70–120%, 36.4%; KCO >120%, 52.8%; p<0.001). There were no significant differences in eosinophil counts, exacerbations in preceding year or maintenance oral steroid use.

Data on biologic response was available in 620 patients (of whom 76% were composite responders). There was no significant difference in mean KCO between those that did or did not meet the composite response threshold (95.3% vs. 97.4%, p=0.21).

Similarly, the proportion of patients achieving a composite response was not significantly different in the subgroups stratified by baseline KCO (KCO <70%, 72.7% composite responders; KCO 70–120%, 75.9% responders; KCO >120%, 78.9% responders; p=0.81).

**Conclusions** Although there was a trend towards an increased composite response rate in patients with increased alveolar gas transfer measurements, alveolar gas transfer does not significantly affect biologic response.

## P301 EOSINOPHILIC INFLAMMATION IN ASTHMA AND COPD: A UK BIOBANK STUDY

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**Introduction and Objectives** Eosinophilic inflammation is recognised to play an important role in asthma. This is reflected in the recent British Thoracic Society (BTS) guidelines which focus on assessing fortype 2 inflammation, particularly elevated eosinophil count,when making a diagnosis of asthma. We investigated the pattern of eosinophilic inflammation in patients with asthma and chronic obstructive pulmonary disease (COPD) in the UK biobank.

**Methods** UK Biobank participants were screened for a coded diagnosis of asthma, COPD or asthma/COPD overlap at baseline or in linked clinical records. We recorded the highest ever eosinophil count taken since the year preceding diagnosis. Participants were separated into three groups: highest recorded eosinophil count $\geq$ 0.3 x $10^9$/L, >0.15 and <0.3 x $10^9$/L, and ≤0.15 x $10^9$/L. This was compared to control data from

**Abstract P301 Table 1** Demographics and highest recorded eosinophil count of study participants

| | Asthma (n=33191) | COPD (n=8130) | Asthma and COPD (n=6915) | Controls (n=411382) |
|---|---|---|---|---|
| **Sex** | | | | |
| Male | 13478 | 4458 | 3356 | 188790 |
| Female | 19713 | 3672 | 3559 | 222592 |
| **Smoking status** | | | | |
| Never smoked | 17583 | 1310 | 1766 | 231820 |
| Previous smoker | 11941 | 3980 | 3248 | 139315 |
| Current smoker | 3433 | 2742 | 1812 | 38199 |
| Not recorded | 234 | 98 | 89 | 2048 |
| **Ethnicity** | | | | |
| White | 31164 | 7847 | 6597 | 387494 |
| Mixed | 234 | 42 | 938 | 2350 |
| Asian/Asian British | 830 | 96 | 119 | 7934 |
| Black/Black British | 459 | 39 | 58 | 6515 |
| Chinese | 58 | 5 | 6 | 1374 |
| Other | 258 | 45 | 49 | 3733 |
| Not recorded | 188 | 56 | 48 | 1982 |
| **Eosinophil count (x $10^9$/L)** | | | | |
| ≤0.15 | 12443 (37.5%) | 2760 (33.9%) | 2216 (32%) | 185818 (45.2%) |
| >0.15 and <0.3 | 10987 (33.15%) | 2750 (33.8%) | 2295 (33.2%) | 131783 (32%) |
| ≥0.3 | 9761 (29.4%) | 2620 (32.2%) | 2404 (34.8%) | 93781 (22.8%) |

participants without diagnosed asthma/COPD. Demographics and summary results are described in table 1.

**Results** In total there were 33191 patients with asthma, 8130 patients with COPD, and 6915 with both diagnoses. When taking the highest recorded value, 9761 (29.4%) patients with asthma, 2620 (32.2%) patients with COPD, and 2404 (34.8%) patients with both diagnoses had an eosinophil count $\geq$ 0.3 x $10^9$/L. This was compared to 411382 controls, of whom 93781 (22.8%) had an eosinophil count $\geq$ 0.3 x $10^9$/L.

**Conclusions** Rates of eosinophilic inflammation were similar in patients with asthma, COPD and asthma plus COPD. This suggests that there may be shared treatable traits across disease boundaries. The high rate of eosinophilic inflammation in COPD is particularly relevant given recent interest in monoclonal antibody therapy for COPD patients and suggests that a significant proportion of patients may be eligible for such treatment.

It is evident from this data that elevated eosinophil count is a poor discriminator between asthma and COPD. Clinicians should be mindful of this, especially in the context of recent BTS asthma guidelines which may prompt a diagnosis of asthma in the setting of elevated eosinophil count. 70.6% of asthma patients have never had high eosinophils, emphasising the role of eosinophil count as a rule-in rather than rule-out test for asthma, with a low diagnostic sensitivity.

## P302 THE VIRTUAL PATIENT STRATIFIES PATIENT-SPECIFIC RESPONSE TO BRONCHIAL THERMOPLASTY WITH HIGH ACCURACY

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**Introduction** Bronchial Thermoplasty (BT) is an available therapy for severe asthma. However, we are unable to stratify *a priori* clinical responders limiting its widespread adoption.

**Aim** To establish BT's clinical benefit and identify responder attributes to enable clinical decision-making.

**Methods** We pooled pre/post-BT patient biopsies from 8 sites (119 patients) that were analysed for airway smooth muscle (ASM) mass, reticular basement membrane, and epithelial integrity to assess the impact on airway remodelling: biological response. Clinical response was assessed via exacerbation and hospitalisation frequency, asthma control questionnaire scores (ACT, ACQ) and asthma quality of life questionnaire score (AQLQ). To stratify patient response, we used the virtual patient[1] to create patient-specific digital avatars, simulated BT, and compared predictions vs clinical data.

**Results** Patients showed significant reductions in ASM mass (mean±SD) 15.1±6.8% to 6.7±4.1%, basement membrane 6.4±2.5µm to 5.5±2.1µm, and improvement in epithelial integrity 30.7±19.1% to 39.9±16.9% ($p<0.05$ all). Annual asthma exacerbation rate decreased: 5.8±5.3 to 1.2±1.7 ($p<0.001$). Asthma-related hospital admissions decreased: 1.5 ±2.5 to 0.3±0.7 ($p<0.001$). ACT improved from 7.7±2.9 to 15.2±6.2, ACQ from 3.0±1.3 to 2.4±1.3, and AQLQ from 3.2±1.6 to 4.4±1.7 ($p<0.05$ all). The virtual patient predicted impact on airway remodelling with 90% and clinical response with 70% accuracy.

**Conclusions** Bronchial Thermoplasty showed excellent biological and clinical response. Our computer model supports the view that BT benefits are largely an indirect vs direct result of heat-driven cell death. The model helped identify the patient profile most likely to benefit from Bronchial Thermoplasty.

### REFERENCE

1. Saunders, *et al. Science Transl Med*. 2019;**11**:eaao6451.

## P303 REAL WORLD ASSESSMENT OF IMPACT OF DIGITAL HOME MONITORING IN DIFFICULT-TO-TREAT ASTHMA

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**Background** Patients on an optimised asthma therapy regime who frequently exacerbate should be considered for biologic therapy. However, self-reporting of symptoms can be unreliable, and adherence to maintenance therapy can be difficult to confirm.

**Hypothesis** Digital lung function and adherence monitoring can predict need for biologic therapy better than self-reporting of symptoms.

**Aim** To review a national programme of digital adherence and peak flow monitoring in patients with difficult-to-treat asthma being considered for biologic therapy.

**Methods** The first 150 patients with difficult-to-treat asthma referred to the national HSE Digital Living Lab programme were analysed. Patients were given digital adherence and peak flow meters. Adherence, peak expiratory flow (PEF), diurnal variability (DV) and exacerbation frequency were recorded over a 3-month period. A device-detected exacerbation was defined by a 20% reduction in PEF from baseline. Self-reported exacerbations were also recorded.

**Results** A total of 117 participants (78%) engaged sufficiently with the digital monitoring programme. Outcome data were available for 72 participants (62%). Among these, 67 patients (74%) were considered for biologic therapy. Of those considered, 53 (79%) subsequently initiated therapy following the digital monitoring period. As summarised in table 1, baseline PEF, DV, exacerbation rate and blood eosinophil count were predictive of progression to biologic therapy.

**Abstract P303 Table 1** Population demographics and factors predicting progression to biologic therapy. (All results presented as median [IQR]).

| **All Participants (n=150)** | |
|---|---|
| Gender | 82 Female; 68 Male |
| Age | 52 [39-63] |
| PEF (L/min) | 346 [264-444] |
| Diurnal Variation (%) | 12.3 [7-23] |
| Adherence (number of days ICS taken as prescribed/number of day monitored, %) | 59 [17-81] |
| Device-Detected Exacerbations | 1 [0-2] |
| Self-Reported Exacerbations | 0 [0-1] |

**Clinical Characteristics Predicting Consideration of Biologic Therapy (Outcome Available, n=72)**

| | **Considered for Biologic (n=53)** | **Not Considered for Biologic (n=19)** | **p-value** |
|---|---|---|---|
| PEF (L/min) | 289 [230-376] | 455 [347-512] | **<0.0001** |
| Diurnal Variation (%) | 18 [9-26] | 11 [5-13] | **0.006** |
| Adherence (Number of days ICS taken as prescribed/number of days monitored, %) | 76 [48-91] | 63 [21-80] | 0.26 |
| Exacerbations during Monitoring (Device Detected) | 1 [0-3] | 0 [0-2] | **0.037** |
| Exacerbations during Monitoring (Self-Reported) | 1 [0-1] | 0 [0-1] | 0.15 |
| Recent or historical blood eosinophil count | 0.47 [0.3-0.8] | 0.2 [0.1-0.3] | **0.003** |
| Recent or historical FeNO | 45 [18-86] | 35 [16-83] | 0.677 |

**Conclusion** Factors identified by home digital monitoring were associated with a subsequent requirement for biologic therapy. Importantly, baseline lung function, diurnal variability and device-detected exacerbations may not be captured through traditional monitoring methods. Patients do not always perceive exacerbations, placing them at risk of under-treatment in the absence of digital monitoring. This programme underscores the value of digital health tools in enhancing referral pathways and informing decisions regarding biologic therapy in patients with difficult-to-treat asthma.

# Impact of treatment on COPD outcomes: from smoking to biologics

## M1 HOW VAPING CHEMICALS COMPROMISE LUNG SURFACTANT

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**Background** The massive increase in vaping worldwide is primarily due to the perceived safety of vapes as a nicotine alternative to cigarettes. However, the long-term health effects of vaping remain unknown. The E-cigarette or Vaping Use-Associated Lung Injury (EVALI) outbreak of 2019 showed the dangers of using vaping products without proper research into the chemicals contained in them. Aldehydes give vapes their unique flavours, and they all react with propylene glycol during vaporization to form acetals. Acetals are more hydrophobic than aldehydes, so will hypothetically compromise the first line of defense against inhaled hazardous substances (i.e. lung surfactant), via interaction with the lipid monolayer.
**Aim** Characterize the effects of Ethyl Vanillin (EV) and Ethyl Vanillin Propylene Glycol Acetal (EVPGA) on the surface tension, compressibility and hysteresis of lung surfactant.
**Methods** A constrained sessile drop surfactometer and drop-shape analysis were used to measure the surface tension of water drops that had been surface-lined with Dipalmitoylphosphatidylcholine (DPPC), a synthetic lipid-mixture of lung surfactant (SLS), or clinically used surfactants (Alveofact), during multiple compression-expansion cycles at physiological breathing rates. EV and EVPGA were then spread at the surfactant-lined drop's air-liquid interface. The compression-expansion cycle was repeated, and the minimum surface tension was compared before and after the vaping chemicals were applied. Compressibility and hysteresis before and after adding the vaping chemicals were calculated and compared, using data collected from the compression-expansion cycles.
**Results** EVPGA reduced the compressibility of DPPC by 54.12% ($P=0.0354$) and Alveofact by 37.71% ($P=0.0116$) in the first 5 cycles. EV raised the minimum surface tension of Alveofact by 55.67% during the first 5 cycles ($P=0.0130$) and 86.87% ($P=0.0036$) in the last five and decreased the compressibility by 45.84% ($P<0.0001$) in the last 5 cycles. EV and EVPGA had no significant changes on SLS or hysteresis.
**Conclusion** In our study, EV and EVPGA significantly reduced the effectiveness of the lung surfactant by compromising its biophysical properties of surface tension and compressibility. This necessitates further research into many other different acetals and their flavouring parent aldehydes to gain a holistic understanding of the dangers of chronic vape use to prevent future deaths.

## M2 IS THE MESSAGING ABOUT STAFF VAPING CONSISTENT IN NHS ORGANISATIONS ACROSS THE UK?

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Smoking is a worldwide global public health concern, with significant health effects. Vaping, or the use of electronic cigarettes (e-cigarettes), has emerged as an alternative to traditional nicotine replacement therapies (NRTs) for smoking cessation. Guidance from Public Health England (PHE) in 2016 recommends that hospital trusts introduce vaping-friendly approaches to support smoke free sites, considering vaping distinct from smoking and allow vaping on hospital sites.[1] Most public places and workplaces are now smoke free, vape free.

The current study aims to map out current vaping policies in NHS organisations. UK NHS organisations were contacted to provide current smoking policies, from each policy, information about vaping restrictions for staff was explored. 165 NHS organisations (England-137, Scotland-9, Wales-14, Northern Ireland-5) were contacted to obtain relevant smoke free policies, through freedom of information requests between June 2024–2025.

151 policies were obtained, in 29.2% (44/151) vaping restrictions were stated to be the same as smoking restrictions. In 27.2% (41/151), vaping was prohibited on site in contrast, 33.1% (50/151) allowed vaping in outside areas, with a small number allowing vaping in designated areas ($n=7$) and some stipulating not within 10–15 metres of buildings ($n=5$). Inconclusive details about vaping were noted in policies from 11 NHS organisations (7.3%). There were conflicting examples where staff were or were not allowed to vape in uniforms openly with visible ID and in one policy vapes were available for purchase on hospital premises. The fire hazard potential of vapes was also highlighted in a small number of policies.

Concerns have been highlighted about potential health risks and the influence of vaping on workplace environments, vaping prevalences continue to rise and a consistent approach is needed.

### REFERENCE

1. Public Health England. Use of e-cigarettes in public places and workplaces: Advice to inform evidenced-based policy making. Public Health England, 2016.

## M3 SUPPORTING 'SWAP TO STOP' SMOKING CESSATION: A PILOT TRIAL OF VAPE INFORMATION PROVISION WITHIN LUNG CANCER SCREENING

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**Introduction** Vaping is an effective quit tool, yet people mistakenly believe it is more harmful than smoking. This mixed-methods study (REC ref:24/NW/0351) assessed the impact of low-cost, easy-read information materials on acceptance of a free vape offered through the 'Swap-to-Stop' scheme within lung cancer screening (LCS).
**Methods** This single-centre two-arm crossover study entailed six one-week blocks (A-B-B-A-B-A) between Feb-May 2025. Control weeks (A) consisted of usual care: very brief advice from an NCSTC-certified nurse, followed by a vape offer with usage guidance. Intervention weeks (B) consisted of usual care plus an educational video, posters and leaflets in the waiting area. Outcomes included the number of vapes offered, acceptance rates and number of smokers leaving with a vape. Vape usage and quit data were collected via phone at 30-days (data collection ongoing, to be presented at the conference). A

subset of participants took part in qualitative interviews to explore perceptions of the materials.

**Results** 517 smokers attended LCS during the study, of whom 302 (58.4%) were offered a vape and 172 (57.0%) accepted. Overall 33.3% (172/517) smokers left with a vape (table 1).

More vapes were offered during intervention weeks (207/298:69.5% *vs* 95/219:43.4%, p<0.001) but acceptance rates were similar (114/207:55.1% *vs* 58/95:61.1%, p=0.330). The higher offer rate meant more smokers left with a vape in intervention weeks (114/298:38.3% *vs* 58/219:26.5%, p=0.005). Acceptance did not differ with sex, ethnicity, education or pack years, but vapes were more likely to be accepted by socioeconomically deprived participants (unadjOR=1.16, 95%CI 1.06–1.26, p=0.001), people who had previously struggled to quit (unadjOR=2.41, 95%CI 1.57–4.02, p<0.001) and younger participants (unadjOR=0.95, 95%CI 0.91–0.99, p=0.02).

Preliminary qualitative findings suggest mixed impact of the materials. While some found them helpful, others reported limited impact due to pre-existing, strong, negative beliefs. An important influence on vape acceptance was the attitude of the nurse offering it.

**Conclusions** Informational materials increased the likelihood of smokers leaving LCS with a vape, due to more offers from clinical staff rather than higher acceptance among participants. Nurses' attitudes influenced acceptance. To maximise impact, staff should receive training to deliver consistent vape offers combined with supportive, accurate messaging that addresses misconceptions and builds trust.

**Abstract M3 Table 1** Characteristics of screening participants who accepted or declined a vape offer

| | Declined vape | Accepted vape | p |
|---|---|---|---|
| **Total, n** | 130 | 172 | |
| **Male, n (%)** | 71 (54.6) | 85 (49.4) | 0.371 |
| **Age, yrs, median (IQR)** | 64 (59–68) | 62.5 (58–66) | **0.02** |
| **Ethnicity, n (%)** | | | |
| White | 120 (92.3) | 160 (93.0) | |
| Black (non-hispanic) | 3 (2.3) | 4 (2.3) | |
| Asian | 2 (1.5) | 2 (1.2) | 0.93 |
| Other | 3 (2.3) | 5 (2.9) | |
| Prefer not to say | 2 (1.5) | 1 (0.6) | |
| **Education level, n (%)** | | | |
| Postgraduate/Professional (Masters/PhD/equiv) | 4 (3.1) | 6 (3.5) | |
| College graduate (Bachelors degree/equiv) | 10 (7.7) | 14 (8.1) | |
| Some college (further education but no degree) | 10 (7.7) | 12 (7.0) | 0.36 |
| Post high school training (A-levels/equiv) | 21 (16.2) | 16 (9.3) | |
| High school graduate (GCSEs) | 61 (46.9) | 77 (44.8) | |
| Less than high school graduate (left school ≤15) | 24 (18.5) | 47 (27.2) | |
| **Socioeconomic position, IMD decile, n (%)** | | | |
| 1 (Most deprived) | 2 (1.5) | 17 (9.9) | |
| 2 | 4 (3.1) | 9 (5.2) | |
| 3 | 8 (6.2) | 19 (11.1) | |
| 4 | 17 (13.1) | 20 (11.6) | |
| 5 | 17 (13.1) | 17 (9.9) | **0.016** |
| 6 | 5 (3.9) | 14 (8.1) | |
| 7 | 10 (7.7.) | 17 (9.9) | |
| 8 | 19 (14.6) | 16 (9.3) | |
| 9 | 24 (18.5) | 25 (14.5) | |
| 10 (Least deprived) | 24 (18.5) | 18 (10.5) | |
| **Smoking status** | | | |
| Current | | | |
| Former | | | |
| **Smoking history, pack years, median (IQR)** | 39.8 (25.0–51.3) | 40 (26.6–51.7) | 0.59 |
| **Have you struggled to quit in the past? n (%)** | | | |
| No | 73 (56.2) | 58 (33.7) | **<0.001** |
| Yes | 57 (43.9) | 114 (66.3) | |

## M4 MULTIMODAL EVALUATION OF AI-ASSISTED APPROACHES TO SMOKING CESSATION

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As the capabilities of large language models (LLMs) continue to improve, AI chatbots may develop strong persuasive capabilities. As such, there is growing interest in the use of interactive, LLM-powered tools for smoking cessation; examples include the World Health Organization's chatbot Sarah and BeFreeGPT and BasicGPT, which were developed recently by a group at George Washington University. As with other applications of generative AI to clinical and public health communication tasks, there is a need to evaluate both the accuracy of information produced and whether it is tailored appropriately to the intended audience.

Previous work has established that AI chatbots can produce accurate information about smoking cessation (Abrams et al. 2025). In this paper, we apply a suite of computational methods, as well as user testing with online participants, to investigate accessibility and tailoring of information. For 12 common internet queries about 'how to quit smoking,' we generated three responses per query using Sarah, BeFreeGPT, and BasicGPT. We then evaluated these responses using a multimodal approach encompassing i) natural language processing metrics for syntactic complexity, jargon, and lexical cohesion, ii) structured semi-quantitative assessments such as the Patient Education Materials Assessment Tool and the Clear Communication Index, and iii) direct user testing. For the user studies, we recruited participants who reported smoking through Prolific, with quota sampling to achieve samples representative of the U.S. population for age, race, ethnicity, and education. Participants, who were randomly assigned either a human-written control passage or a passage generated by BeFreeGPT, answered reading comprehension questions about the material provided and rated its persuasiveness using a Likert scale.

For all three sets of passages, we observed substantial heterogeneity across our different modes of evaluation. Of particular interest, we found that our additional computational metrics often yielded divergent predictions from length-based readability formulas, which remain standard in the field. AI-generated passages, however, were rated as significantly more persuasive in the user study, suggesting that AI-assistant approaches to smoking cessation warrant further investigation.

## M5 SMOKING CESSATION PROVISION AND OUTCOMES FOLLOWING INITIATION OF A NEW SMOKING CESSATION SERVICE ALONGSIDE AN URGENT SUSPECT LUNG CANCER CLINIC

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**Introduction** Stopping smoking at the time of lung cancer diagnosis is associated with improved overall survival, with one study demonstrating 22-month longer median survival in people with stage IA-IIIA who quit compared to those who continued to smoke.[1] However, smoking cessation (SC) interventions are known to be inconsistently delivered in routine care. Here we present outcomes before and after the introduction of a dedicated SC service alongside an urgent suspected lung cancer clinic (USC).

**Methods** A new standard operating procedure for outpatient provision of SC interventions was developed, including brief guides to prescribing Varenicline and Cytisine as well as reminders regarding web referral to the local authority provided stop smoking service (LSSS). In addition, two LSSS smoking cessation practitioners (SCPs) attended two of the four lung cancer clinics per week (one USC clinic, one follow-up clinic). Clinic letters were audited for documentation of SC referrals and pharmacotherapy/cessation aids; SC outcomes were derived from LSSS records.

**Results** A total of 117 people currently smoking at the time of their USC attendance were studied, of which 52 were first seen between November 2024 and January 2025 (prior to initiation of the service) and 65 were first seen between February and April 2025 (following initiation). Overall mean age was 66 yrs, 40% were female, 44% were from the lowest IMD decile, 84% were of white ethnicity, and 59% had an eventual diagnosis of lung cancer. SC interventions and outcomes from the two time periods are shown in table 1. Following initiation of the service, the proportion of patients referred to LSSS increased from 21% to 57% ($p=0.0001$), the proportion prescribed any pharmacotherapy/cessation aids in clinic increased from 6% to 40% ($p<0.0001$), and the number with any SCP-recorded self-reported quit increased from 10% to 20% ($p=0.192$). The proportion of patients referred to LSSS and issued with pharmacotherapy in clinic varied widely between clinicians.

**Discussion** Increases in SC referrals, provision of pharmacotherapy, and self-reported quits were seen within the first three months of the service. Ongoing audit cycles aim to improve consistency of provision of these interventions.

### REFERENCE

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## M6 MAKE EVERY CONTACT COUNT- OPPORTUNISTIC LUNG CANCER SCREENING AND SMOKING CESSATION AT A COMMUNITY DIAGNOSTIC CENTRE

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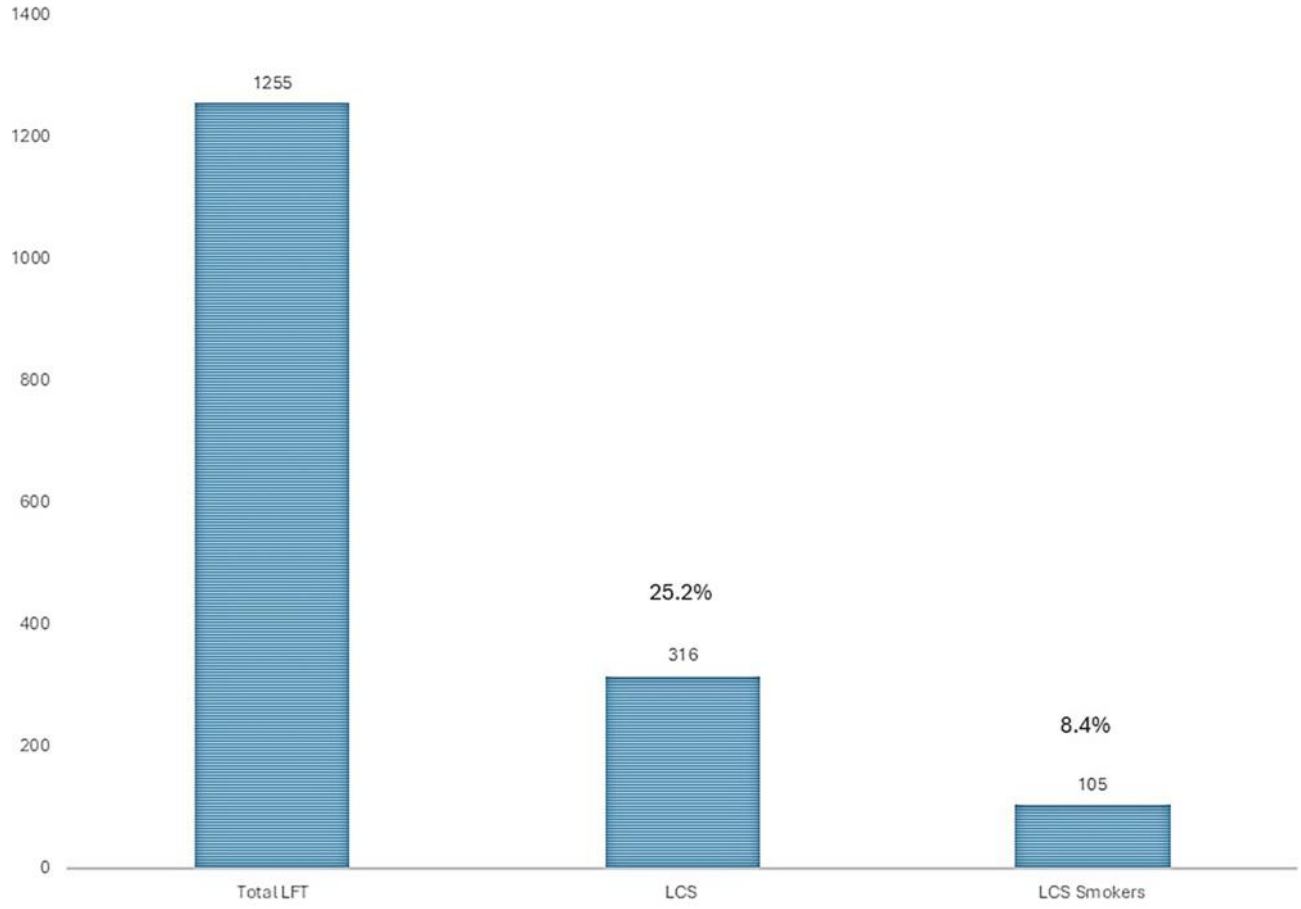
10.1136/thorax-2025-BTSabstracts.490

**Introduction** Lung cancer screening (LCS) reduces lung cancer mortality and health inequality. Implementation is challenging at a time when screening invites are limited. We sought to establish the numbers of patients attending our community

**Abstract M5 Table 1** Smoking cessation referrals, provision of cessation aids, and outcomes during the study period

| | November 2024 - January 2025 | February 2025 – April 2025 |
|---|---|---|
| Total number of people currently smoking seen (n) | 52 | 65 |
| | | |
| Referred to LSSS (n,%) | 11 (21%) | 37 (57%) |
| Declined referral to LSSS (n,%) | 18 (35%) | 15 (23%) |
| No record of discussion of referral in clinic letter (n,%) | 23 (44%) | 13 (20%) |
| | | |
| Prescribed Varenicline from clinic (n,%) | 2* (4%) | 19 (29%) |
| Prescribed Cytisine from clinic (n,%) | 0 (0%) | 2 (3%) |
| Prescribed NRT from clinic (n,%) | 1 (2%) | 5 (8%) |
| Prescribed any pharmacotherapy/cessation aids (n,%) | 3 (6%) | 26 (40%) |
| Declined cessation aid (n,%) | 10 (19%) | 14 (22%) |
| No record of discussion about cessation aid (n,%) | 39 (75%) | 23 (35%) |
| | | |
| Seen by LSSS SCP but no documented quit (n,%) | 6 (12%) | 12 (18%) |
| Seen by LSSS SCP and any recorded quit (n,%) | 5 (10%) | 13 (20%) |

* request for GP to prescribe in clinic letter




**Abstract M6 Figure 1** Total Number of patients referred for LFT, those meeting age criteria for LCS and the number of current smokers within that group. Percentage of total LFT patients shown. Abbreviations: LFT- lung function tests, LCS- lung cancer screening

diagnostic centre (CDC) for lung function testing (LFT) that would qualify for opportunistic LCS and smoking cessation services (SCC).

**Methods** Patients attending the CDC for LFT from November 2024 to January 2025 were assessed by age and smoking status to determine whether they would qualify for LCS (*55*–74 years and current or ex-smokers) and/or referral to tobacco dependency services (TDS).

**Results** In the 3 months 1255 LFT attendances were recorded of which 316 patients within the LCS age range (25.2%, figure 1). Of those 149 (47%) were females and 167 (54%) males, with a median age of 65 yrs. 105 patients were current smokers warranting referral to tobacco dependency services, representing 8.4% of total LFT and 33.2% of within LCS age range.

**Conclusion** One quarter of LFT referrals are within the age range for LCS, representing a group of patients that could be referred directly to LCS from the community (although we did not confirm whether any were already within the programme). 50% of that group were current smokers supporting the need for reliable access to SCC from the CDC. Further work aims to develop easy to use (e.g. QR code) information for CDC attendees on LCS and Smoking cessation with proactive referrals. Furthermore, we propose to assess all attendees at CDC to more broadly understand opportunistic LCS and TDS referrals.

## M7 5-YEAR OUTCOMES OF LUNGCAST: 'SMOKING CESSATION IN LUNG CANCER STUDY'

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10.1136/thorax-2025-BTSabstracts.491

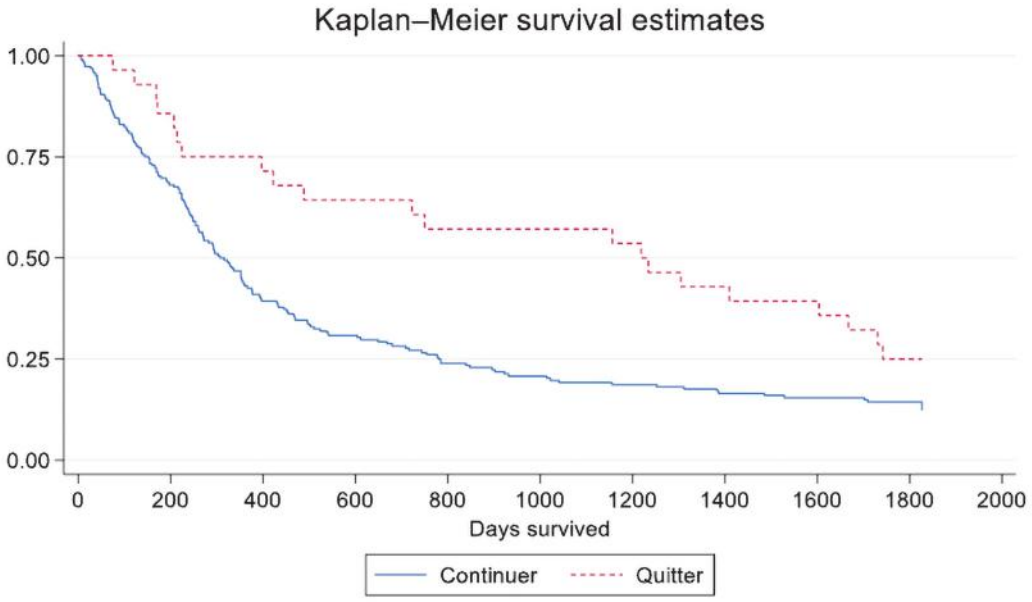
**Introduction** Our UK prospective study of 2751 patients with non-small cell lung cancer (NSCLC) showed that both smoking status at diagnosis and especially stopping smoking, were independently and significantly associated with better survival at 2 years[1] and that smoking cessation is cost-effective.[2]

**Objective** To compare longer-term (5 year+) outcomes.

**Design** We sought additional ethical approval to check vital status after a minimum of 5 years, in a subgroup of 526 participants (mean age 71 years, 54% male) from within our Health Board, that were recruited into LungCast between 2010–2020.

**Methods** Smoking status was assessed at 1, 3, 6, 12, 24, 60 months (confirmed by eCO) from date of diagnosis. Kaplan-Meier analysis and log-logistic models estimated the effects of smoking status at baseline and quitting on long-term survival.

**Results** 1. The 7% with NSCLC who had never smoked lived longer than the 55% who were ex-smokers (p=0.02) and the 37% who were smoking at baseline (p=0.02) but this was not statistically significant on multivariate analysis (p=0.06 and




**Abstract M7 Figure 1**

p=0.21 respectively) after accounting for age, treatment (surgery, radiotherapy, chemotherapy), cancer stage and ECOG performance status.

2. 28 from the 194 smokers (15%) became long-term quitters. They had roughly double the survival proportion (25% versus 13%, p<0.01) at 5 years, a longer median survival 723 days versus 260 days (p<0.02). Survival was statistically significant at univariate, multivariate and log-logistic regression (survival ratio 2.82, 95% CI 1.62–4.93, p<0.01) after accounting for the above confounders. See figure 1.

**Conclusion** A subgroup from the original study shows smoking status at baseline has a trend to towards predicting survival but the significant benefits of quitting if you are smoker are maintained beyond 5 years.

## REFERENCES

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## M8 TAILORING CYSTISINE'S DOSAGE IN SMOKERS WITH COMORBIDITIES AND UNSUCCESSFUL PREVIOUS EFFORTS

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10.1136/thorax-2025-BTSabstracts.492

**Introduction** Cytisine has been used as a smoking cessation medication for decades in Eastern countries. Its use has been extended in many European countries and now has a pending application in WHO to be used as a first line medication for smoking cessation.

**Aim** To evaluate smoking abstinence results of Cytisine including smokers with comorbidities. Patients >65 years old were included, with exclusion of those with renal failure. Data are derived from the Outpatient Smoking Cessation Clinic of the Pulmonary Department of First Intensive Care Unit of 'Evangelismos' Hospital during the period September 2024-March 2025. Notably, 46.9% had previously attempted to quit unsuccessfully.

**Method** 105 smokers participated. Demographic data, comorbidities, Fagerström Test and pulmonary function test were collected. Cytisine was provided free of charge at a daily dose of 9 mg until smoking cessation, typically between days 14 and 21 in highly dependent smokers with a smoking history more than 30 pack-year. Dose of 9 mg was maintained for one additional week, followed by a gradual tapering over a total treatment period of two months. Intensive behavioural support through five structured sessions was also provided.

**Results** Mean age of patients was 53.1 ± 10.79 years (45.7% men). Regarding comorbidities, 22.5% had COPD, 11.1% asthma, 24.7% hypertension, 18.8% depression and 13.8% anxiety. 56% of smokers were highly addicted (Fagerström Test score>7) and 57% of them achieved smoking abstinence, whereas 62% of those (44%) with Fagerström Test score<7 quitted smoking (p value=0.7). Overall, 68% of participants achieved smoking cessation in three months. The majority (73.1%) were using traditional tobacco products. There were no statistically significant differences in demographics or pulmonary function tests between those who quit smoking and those who did not. No major adverse events were reported. Extended use of Cytisine was associated with increased smoking cessation rates.

**Conclusion** Tailoring Cystisine dosage in highly addicted smokers with comorbidities that had previous failed, leaded to increased smoking cessation rates at three months. Reimbursement of smoking cessation drugs is contributing. There is urgent need for further larger studies with long-term follow up, in order to assess efficacy and smoking cessation rates achieved by Cytisine.

## M9 DO SMOKERS REQUIRING SECONDARY CARE SERVICES ENGAGE WITH COMMUNITY-BASED SMOKING CESSATION SUPPORT?

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10.1136/thorax-2025-BTSabstracts.493

**Introduction** Smoking cessation interventions vary considerably. Following a hospital inpatient or outpatient attendance many patients will be referred to community-based stop smoking services. We report the level of engagement in those seen and followed up in a hospital-based service compared to community follow up alone.

**Methods** Cardiff and Vale University Health Board has a hospital and community based 'Help me Quit' (HB-HMQ, CB-HMQ) smoking cessation service. HB-HMQ aims to offer an individualised smoking cessation programme for patients with chronic medical conditions. Between January and December 2024 all referrals from secondary care teams to HB-HMQ were either seen and followed up by HB-HMQ or referred directly to CB-HMQ without HM-HMQ involvement based on whether patients had underlying chronic medical conditions. We retrospectively reviewed the engagement and success levels in this patient cohort.

**Results** 1,254 patients were referred to the HB-HMQ service from secondary care services. 246 patients were referred to CB-HMQ however 211 (86%) did not engage. Among those who set a quit date, 18 (53%) remained smoke-free at the 1-month follow-up.

In comparison of the 1,008 chronic patients scheduled to be followed up by the HB-HMQ, 658 (65%) did not engage with the HB-HMQ. From those who set a quit date with HB-HMQ, 81 (83%) were smoke-free at 1 month.

**Discussion** Engagement with smoking cessation services is challenging for patients with chronic medical conditions and it has previously been shown that hospitalised patients do better with a HB-based program.[1] In our study patients seen in secondary care services without chronic medical conditions had lower levels of engagement and 1-month success rates with direct referral to CB-HMQ. These results are comparable to previous studies[1] and demonstrate the importance of continuity and a patient-centred, integrated approach in this patient group.

### REFERENCE

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## M10 EVALUATION OF PARTICIPANT EXPERIENCE IN THE NIHR HTA OPACE (OPTIMISING AZITHROMYCIN PREVENTION TREATMENT IN COPD TO REDUCE EXACERBATIONS) TRIAL – A PRAGMATIC TRIAL OF CONTINUED VS DISCONTINUED AZITHROMYCIN PROPHYLAXIS IN COPD

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10.1136/thorax-2025-BTSabstracts.494

**Background** The patient voice is vital to shaping research that reflects what matters most to patients. OPACE is a double-blind placebo-controlled randomised trial of continued azithromycin vs discontinuation trial in COPD patients on long-term azithromycin prophylaxis to reduce exacerbations. We performed a trial sub-study to gain feedback on participants' trial experiences.

**Methods** The trial is recruiting, and this optional sub-study involves a questionnaire at baseline and end of trial and a 3-month interview in a smaller group. OPACE is a pragmatic trial that uses patient information sheets, trial website and video as patient-facing materials. The trial design permits home or in-person participation and home delivery of trial medication. For people who declined trial participation, where possible, reasons for declining were collated.

**Results** Currently, 71 participants completed the baseline questionnaire, 18 participants completed the 3-month interview, none have yet completed their end of trial questionnaires.

Participants were from across the UK regions, 55% were male, median (interquartile range) age was 72 (64–77) years, 96% were white, 59% considered themselves to be disabled, 8% were carers, 32% lived alone. 75% of participants in the OPACE trial are taking part in research for the first time.

73% found the trial participant information sheet very useful, but only 11% watched the trial video, and only 14% had seen the OPACE website.

80% liked the choice of taking part in the trial from home or in-person, 60% preferred to take part from home, 30% preferred a combination of home/in-person consultations.

93% found it very helpful that trial medication was home delivered.

73% were very comfortable/comfortable that their azithromycin may be discontinued as part of the trial.

Analysis of interview data is ongoing but emerging themes to support trial participation include trial team support and ability to restart standard azithromycin in the trial.

Reasons for declining trial participation include not wanting to take part in research, carer responsibilities, too unwell and risk of exacerbation.

**Conclusion** Participants value the choice of taking part in the trial from home and are comfortable with the trial intervention. Future analysis from interviews and end of trial participation is planned.

## M11 REAL-LIFE OUTCOMES OF PATIENTS WITH COPD INITIATING BUDESONIDE/GLYCOPYRRONIUM/FORMOTEROL AFTER A PREVIOUS DUAL THERAPY IN SPAIN. ORESTES STUDY

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10.1136/thorax-2025-BTSabstracts.495

**Introduction** Since 2022, triple therapy (TT) with budesonide/glycopyrronium/formoterol (BGF) has been available in Spain for COPD treatment. Real-world evidence is essential to

complement data from clinical trials. The ORESTES study aimed to describe patients' characteristics at BGF initiation, and to report key clinical outcomes after 12 months. This abstract focuses on patients who escalated to BGF from dual therapy (DT).

**Methods** This retrospective chart review was conducted across 20 hospitals from Spain. A total of 718 patients with COPD (spirometry-confirmed) aged ≥40 years at BGF initiation were included.

**Results** Among all patients, 295 (41.1%) escalated to BGF from DT, 49.6% transitioned from prior TT, 3.3% from monotherapy, and 6.0% were treatment-naïve. Within the DT group, 38% escalated from ICS/LABA and 62% from LABA/LAMA. The mean age was 69.9 years (SD=9.9), with 31.2% being female. The average duration of COPD was 7.3 years (SD=5.7); 48.5% had a predicted FEV1 <50%, and 60.3% had experienced at least one COPD exacerbation (ECOPD) in the previous year. Additional baseline characteristics are presented in table 1.

Following initiation of BGF, reductions were observed in the incidence of ECOPD, as well as in the use of rescue medication (SABA), oral corticosteroids (OCS), and antibiotics (see

**Abstract M11 Table 1** Baseline characteristics and main outcomes of patients initiating BGF after dual therapy

| **Baseline Characteristics** | | |
|---|---|---|
| Age (years) - mean (SD) | | 69.9 (9.9) |
| Sex, male (%) | | 68.8% |
| Time since COPD diagnosis (years) - mean (SD) (N=290) | | 7.3 (5.7) |
| Current smokers - % | | 35.3% |
| Former smokers - % | | 62.0% |
| Pack-years – mean (SD) (N=621) | | 48.6 (26.1) |
| $FEV_1$ (L) – mean (SD) | N=126 | 1.34 (0.59) |
| $FEV_1$ (% predicted) – mean (SD) | | 50.4 (17.0) |
| < 30% predicted | | 12.9% |
| ≥ 30% - < 50% predicted | | 35.6% |
| ≥ 50% - < 80% predicted | | 48.5% |
| ≥ 80% predicted | | 3.0% |
| GOLD AEB classification | N=208 | |
| GOLD E (2 mod. ECOPD or 1 sev. ECOPD) | | 40.9% |
| GOLD A (0-1 mod ECOPD + mMRC 0-1) | | 13.9% |
| GOLD B (0-1 mod ECOPD + mMRC ≥ 2) | | 45.2% |
| Concurrent chronic diseases | | |
| ≥ 3 general - % | | 91.2% |
| ≥ 1 cardiovascular* - % | | 77.6% |

| **Outcomes** | | **Baseline†** | **Follow-up†** | |
|---|---|---|---|---|
| BGF treatment persistence - % | Persistence at 12 months | | 91.5% | |
| | Discontinuation before 12 months | | 4.1% | |
| | Deceased | | 4.1% | |
| | Loss to follow-up | | 0.3% | **Relative Difference (95%CIs)** |
| ECOPD | Total, ≥ 1 - % | 60.3% | 50.2% | -16.9% (-28.6%; -5.1%) |
| | Total - annualised rates | 1.06 | 0.94 | -11.6% (-13.3%; -9.8%) |
| | Severe, ≥ 1 - % | 29.5% | 25.1% | -14.9% (-35.5%; 5.6%) |
| | Severe - annualised rates | 0.37 | 0.36 | -3.9% (-5.5%; -2.3%) |
| | Moderate, ≥ 1 - % | 42.0% | 34.2% | -18.6% (-34.8%; -2.4%) |
| | Moderate - annualised rates | 0.69 | 0.58 | -15.7% (-17.2%; -14.2%) |
| ≥ 1 SABA use (actual)‡ - % patients | | 43.0% | 33.0% | -23.2% (-27.1%; -19.3%) |
| SABA, actual no. of used prescriptions‡ – annualised rates | | 1.00 | 0.75 | -25.0% (-26.9%; -23.2%) |
| ≥ 1 OCS use - % patients | | 39.3% | 35.3% | -10.3% (-27.6%; 6.9%) |
| ≥ 1 Antibiotic use - % patients | | 49.8% | 42.4% | -15.0% (-27.9%; -2.0%) |

*SD: Standard deviation; FEV1: Forced expiratory volume in 1 second; GOLD: Global Initiative for Chronic Obstructive Lung Disease; mMRC: modified Medical Research Council dyspnea scale; ECOPD: COPD exacerbation; OCS: oral corticosteroids; CI: confidence interval.*

** Includes: pulmonary hypertension, tachyarrhythmias and other arrhythmias, ischaemic or haemorrhagic stroke, aneurysm, ischaemic heart disease, myocardial infarction, heart failure, peripheral arterial disease, hypertension, obesity, dyslipidaemia and diabetes mellitus.*

*† Defined as 12-month period pre-BGF (baseline) or post-BGF (follow-up)*

*‡ Actual use of SABA was collected (not only prescriptions by clinicians)*

table 1). The proportion of patients experiencing at least one ECOPD decreased from 60.3% to 50.2%, and the annualised exacerbation rate declined from 1.06 to 0.94. SABA actual use dropped from 43.0% prior to BGF initiation to 33.0% afterwards, with the mean annualised number of actual prescriptions used per patient decreasing from 1.00 to 0.75. The proportion of patients requiring OCS decreased from 39.3% to 35.3%, while those needing antibiotics declined from 49.8% to 42.4%. Treatment persistence with BGF at 12 months was 91.5%.

**Conclusion** In the ORESTES study conducted in Spain, patients with COPD who escalated from dual therapy to BGF showed high treatment persistence at 12 months and experienced clinically meaningful improvements, including reductions in exacerbations and decreased use of additional treatments.

## M12 REAL-WORLD DISEASE CONTROL AND STABILITY IN PATIENTS WITH COPD TREATED WITH EXTRAFINE TRIPLE THERAPY WITH BECLOMETASONE DIPROPIONATE, FORMOTEROL, AND GLYCOPYRRONIUM

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10.1136/thorax-2025-BTSabstracts.496

**Background** Achieving disease control (DC) and stability (DS) in COPD could be valuable goals to evaluate treatment effectiveness in real-world settings. Composite endpoints may be useful tools to assess DC and DS and provide a more holistic assessment by combining multiple measures including lung function, symptoms, and exacerbations.

**Methods** This pooled analysis combined 4541-individual patient level data from six European, prospective, observational studies to assess the proportion of patients with COPD who met DC and DS outcomes following treatment with extra fine fixed dose combination of beclomethasone dipropionate, formoterol, and glycopyrronium (BDP/FF/G) in single inhaler. DC is defined as an improvement in Forced Expiratory Volume in the first second (FEV1) of $\geq$100 mL from baseline, a reduction of $\geq$2 points in the COPD Assessment Test (CAT) score, and 0 exacerbation. DS is stated as FEV1 increase or no change, CAT reduction or no change, and 0 exacerbations. DC and DS were analysed at 3 and 6 months of treatment with extra fine BDP/FF/G.

**Results** The full analysis set included 4541 patients (62.3% male), with mean (SD) CAT score 21.5 (7.5), mean (SD) FEV1 1.38L (0.59), and mean (SD) exacerbation rate 1.6 (1.0). At 3 months (n=1694), 23.3% and 45.2% of patients achieved DC and DS, respectively. At 6 months (n=1554), 24.7% and 42.2% achieved DC and DS, respectively.

**Conclusions** Treatment with extra fine single inhaler triple therapy BDP/FF/G represents an effective option to achieve disease control and stability in COPD patients in real-world setting, providing practical and patient-centric treatment goals.

## M13 ANTIBIOTIC AND SYSTEMIC CORTICOSTEROID USE IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND TYPE 2 INFLAMMATION RECEIVING DUPILUMAB

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**Introduction and Objectives** Systemic corticosteroids (SCS) and antibiotics are often co-prescribed for chronic obstructive pulmonary disease (COPD) exacerbations. However, prolonged use can be associated with adverse events. In the BOREAS and NOTUS trials, dupilumab reduced exacerbations and improved lung function in patients with COPD and type 2 inflammation. This post hoc analysis evaluated whether dupilumab reduced SCS and antibiotic use in patients from BOREAS and NOTUS.

**Methods** BOREAS (NCT03930732) and NOTUS (NCT04456673), phase 3, randomized, placebo-controlled trials, enrolled patients with COPD and type 2 inflammation (screening blood eosinophil counts $\geq$300 cells/μL) on triple therapy. Patients received add-on dupilumab or placebo for 52 weeks. Endpoints: mean SCS and antibiotic intake duration and adjusted annualized SCS and antibiotic courses for patients with $\geq$1 moderate or severe exacerbation during the treatment period.

**Results** Among patients with $\geq$1 moderate or severe exacerbation during the 52-week treatment period (dupilumab: n=338; placebo: n=394), mean (SD) SCS intake duration was 16.1 (22.1) and 22.2 (34.1) days for dupilumab and placebo, respectively; mean antibiotic intake duration was 23.0 (49.2) and 24.8 (49.4) days, respectively. In these patients, dupilumab vs placebo reduced the annualized number of total SCS courses by 20% (relative risk vs placebo of 0.80 [95% CI: 0.71, 0.91]; $P$=0.0004) and antibiotic courses by 11% (relative risk vs placebo of 0.89 [95% CI: 0.79, 1.00]; $P$=0.058). For patients using SCS for $\geq$1 severe exacerbation, mean (SD) SCS intake duration was 23.5 (21.3) and 34.9 (26.4) days for dupilumab and placebo, respectively; antibiotic intake duration was 17.8 (13.8) and 21.3 (20.0) days, respectively. In these patients, dupilumab vs placebo reduced the annualized number of SCS courses by 63% (relative risk vs placebo: 0.37 [0.19, 0.72]; $P$=0.0033) and of antibiotic courses by 62% (relative risk vs placebo: 0.38 [0.20, 0.74]; $P$=0.0043).

**Conclusions** In BOREAS and NOTUS, dupilumab compared with placebo significantly reduced the number of exacerbations. In the subgroup of patients who experienced exacerbations during the treatment period, dupilumab compared with placebo reduced the cumulative number of days of exposure to SCS or antibiotics and reduced the number of SCS and antibiotic courses, regardless of exacerbation severity.

## M14 ASSOCIATION BETWEEN EARLY LUNG FUNCTION IMPROVEMENT WITH DUPILUMAB AND SUSTAINED CLINICAL OUTCOMES IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CLINICAL PERSPECTIVE

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**Introduction and Objectives** Dupilumab, a human monoclonal antibody, blocks interleukin-4/13 signalling, key and central drivers of type 2 inflammation. In BOREAS and NOTUS, dupilumab reduced exacerbations and improved lung function in patients with chronic obstructive pulmonary disease (COPD) and type 2 inflammation. This post hoc analysis assessed whether early lung function improvement correlates with reduction in moderate exacerbations and symptom burden, and improved quality of life over 52 weeks in patients from BOREAS and NOTUS.

**Methods** BOREAS (NCT03930732) and NOTUS (NCT04456673), phase 3 randomized, double-blind, placebo-controlled trials, enrolled patients with COPD, moderate-to-severe airflow limitation, and type 2 inflammation (screening blood eosinophils $\geq$300 cells/μL) on triple therapy. Patients received add-on subcutaneous dupilumab 300 mg or matching placebo q2w for 52 weeks. Endpoints were change in annualized moderate or severe COPD exacerbation rates, comparing patients with Week 4 change from baseline in post-bronchodilator forced expiratory volume in 1 second ($FEV_1$) of <150 mL vs $\geq$150 mL (threshold identifying patients with above average $FEV_1$ improvement in the ITT population), as well as Pearson correlation coefficient (*r*) for Week 4 change of post-bronchodilator percent predicted (pp) $FEV_1$ and Week 52 clinical outcomes: moderate or severe exacerbation rate, St. George's Respiratory Questionnaire (SGRQ), and Evaluating Respiratory Symptoms in COPD (E-RS:COPD).

**Results** In dupilumab recipients, moderate or severe exacerbations were reduced by 22% (relative risk: 0.78 [95% CI: 0.61, 0.98]; $P$=0.032) in those with improvement of post-bronchodilator $FEV_1$ $\geq$150 mL at Week 4 compared with patients with <150 mL improvement. In patients receiving dupilumab, Week 4 change from baseline in post-bronchodilator $ppFEV_1$ also correlated with a reduction in moderate exacerbation rates ($r$=–0.1043; $P$=0.0017), and Week 52 improvement in SGRQ ($r$=–0.2201; $P$<0.0001), and E-RS: COPD total scores ($r$=–0.1543; $P$<0.0001); the placebo arm did not show significant correlations, except with Week 52 SGRQ total scores ($r$=−0.1405, $P$=0.0002).

**Conclusion** Patients with greater early lung function improvement had significantly greater reduction in moderate exacerbation. Early improvements in lung function were weak but significantly correlated with reductions in exacerbations and symptom burden at Week 52, as well as with improvements in quality of life.

## M15 DUPILUMAB EFFICACY ACROSS BASELINE EOSINOPHIL COUNTS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION

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**Introduction and Objectives** Type 2 inflammation (indicated by elevated blood eosinophil counts [BEC]) in patients with chronic obstructive pulmonary disease (COPD) often correlates with higher exacerbation risk. In BOREAS and NOTUS, dupilumab significantly reduced exacerbation rates and improved lung function. Safety was consistent with the known dupilumab safety profile. This post hoc analysis evaluated dupilumab efficacy in patients, grouped by baseline BEC $\geq$150 cells/μL or $\geq$300 cells/μL.

**Methods** BOREAS (NCT03930732) and NOTUS (NCT04456673), phase 3, randomized, placebo-controlled trials, enrolled 1,874 patients (40–85 years) with COPD, moderate-to-severe airflow limitation, and type 2 inflammation (screening BEC $\geq$300 cells/μL). Patients were randomized to dupilumab 300 mg or placebo q2w for 52 weeks. Baseline BEC for subgroups was measured at randomization. This analysis included patients from the intention-to-treat population, grouped by baseline BEC $\geq$150 cells/μL, or $\geq$300 cells/μL. Endpoints assessed included annualized moderate or severe exacerbation rate, change from baseline to Week 52 in pre-bronchodilator forced expiratory volume in 1 second ($FEV_1$), St. George's Respiratory Questionnaire (SGRQ), and Evaluating Respiratory Symptoms (E-RS): COPD total scores. Data are shown as least squares mean difference vs placebo (95% CI) at Week 52, unless stated otherwise.

**Results** Of 1,874 patients, 1,703 (dupilumab n=855; placebo n=848) had baseline BEC $\geq$150 cells/μL, and 1,135 (dupilumab n=573; placebo n=562) had baseline BEC $\geq$300 cells/μL. Dupilumab reduced annualized exacerbation rates vs placebo by 36.9% (BEC $\geq$150 cells/μL) and 35.8% (BEC $\geq$300 cells/μL). Dupilumab improved pre-bronchodilator $FEV_1$ by 76 mL (42, 110; BEC $\geq$150 cells/μL) and 92 mL (51, 133; BEC $\geq$300 cells/μL) and reduced patient-reported SGRQ scores at Week 52 by −3.1 points (−4.7, −1.4; BEC $\geq$150 cells/μL) and −4.0 points (−6.0, −1.9; BEC $\geq$300 cells/μL), as well as ERS:COPD scores by −1.0 point (−1.5, −0.4; BEC $\geq$150 cells/μL) and −1.0 point (−1.7, −0.4;BEC $\geq$300 cells/μL).

**Conclusions** In patients with COPD and type 2 inflammation, dupilumab reduced annualized exacerbation rates and symptom burden, and improved lung function and quality of life, with greater treatment effects observed in those patients with higher baseline BEC.

## M16 TREATMENT FAILURES DURING AN EOSINOPHILIC EXACERBATION FOLLOWING TREATMENT WITH BENRALIZUMAB OR PREDNISOLONE: DATA FROM ABRA

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**Introduction** The ABRA trial was a multicentre, phase 2, double-blind, double-dummy, placebo-controlled clinical trial. Adults with asthma or COPD exacerbations and blood eosinophils ≥300 cells/μL were randomised 1:1:1 to: (1) benralizumab + placebo (BENRA), (2) placebo + prednisolone (PRED), or (3) benralizumab + prednisolone (BENRA +PRED). Benralizumab was superior to prednisolone (standard of care) at reducing treatment failures, but treatment failures were still 45% at 90 days (compared to 74% with prednisolone).
**Aim** Describe the characteristics of patients who had a treatment failure following an eosinophilic exacerbation.
**Results** The characteristics of the patients who did and did not have any treatment failures are shown in table 1.

In the multivariable logistic regression model, steady-state% predicted $FEV_1$ was a statistically significant predictor of treatment failure (OR = 0.971, 95% CI: 0.945–0.999, p=0.042), indicating that higher baseline lung function was associated with reduced odds of repeat exacerbation. Additionally, treatment with prednisolone alone was associated with a more than threefold increase in the odds of treatment failure compared to benralizumab or benralizumab plus prednisolone (OR=3.16, 95% CI: 1.38–7.25, p=0.005). Other variables, including age, smoking status and eosinophil count and FeNO at time of exacerbation, were not significantly associated with treatment outcome.

**Abstract M16 Table 1** Patient characteristics for those patients who had treatment failure vs those who did not in the ABRA trial

| | Did have a treatment failure (n=87) | Did not have a treatment failure (n=71) |
|---|---|---|
| Women, n (%) | 49 (55.1) | 37 (52.1) |
| Age, mean (SD) | 58 (13.84) | 56.3 (16.85) |
| Asthma exacerbation, n (%) | 48 (53.9) | 40 (56.3) |
| COPD exacerbation, n (%) | 39 (43.8) | 31 (43.7) |
| Smoker, n (%) | 11 (12.4) | 4 (5.6) |
| Ex Smoker, n (%) | 44 (49.4) | 36 (50.7) |
| $FEV_1$% predicted at steady state, mean (SD) | 68.38 (23.29) | 75.9 (24.2) |
| Blood eosinophils at exacerbation, mean (SD) | 0.7 (0.41) | 0.68 (0.42) |
| Fraction exhaled nitric oxide at exacerbation, mean (SD) | 54.67 (46.86) | 59.5 (43.91) |
| Benralizumab alone treatment, n (%) | 25 (28.1) | 28 (39.4) |
| Benralizumab and prednisolone treatment, n (%) | 23 (25.8) | 29 (40.8) |
| Prednisolone treatment, n (%) | 39 (43.8) | 14 (19.7) |

**Conclusion** Reduced baseline lung function and monotherapy with prednisolone were key predictors of treatment failure in the ABRA study.

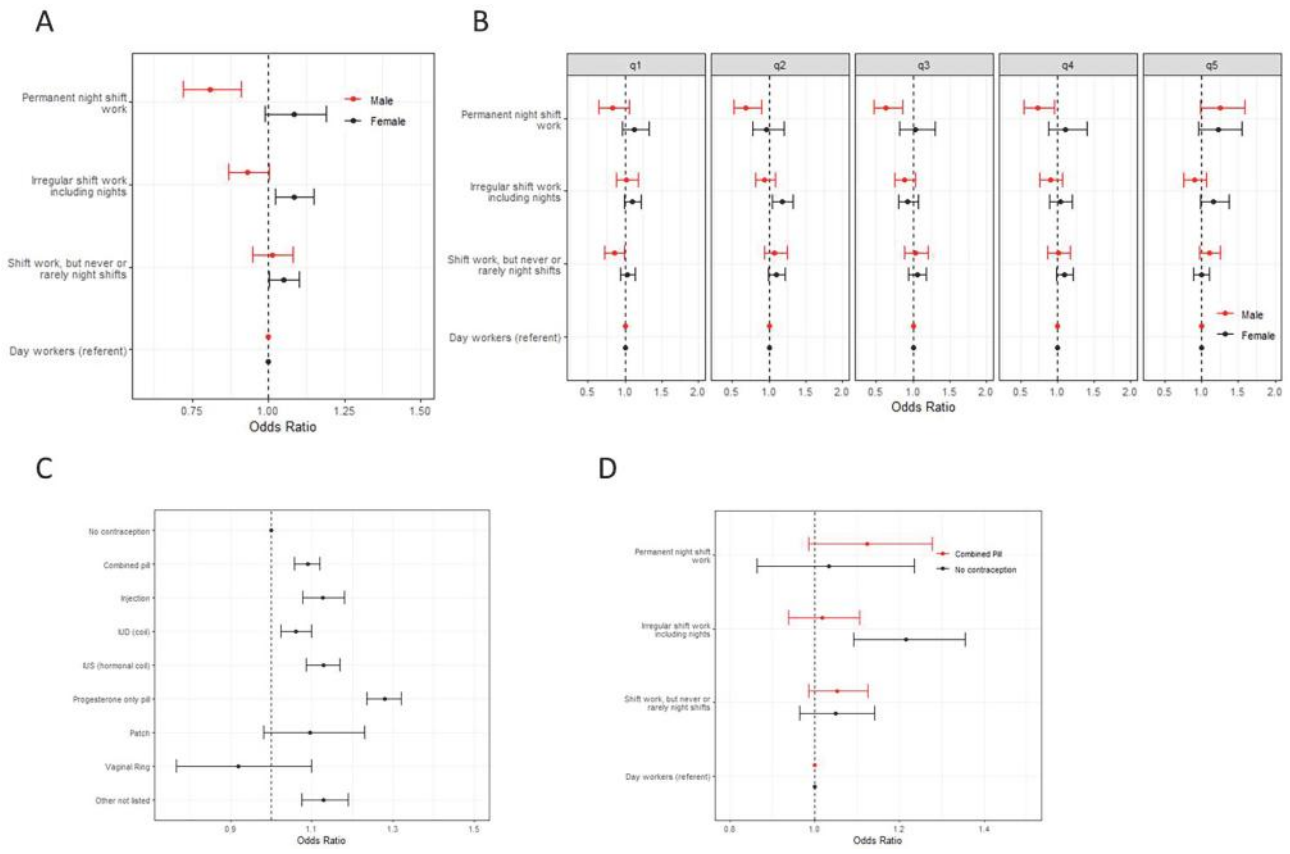
# The multi-morbidity of asthma

## M17 SEX DIFFERENCES IN THE RELATIONSHIP BETWEEN SHIFT WORK STATUS AND PREVALENT ASTHMA IN THE OUR FUTURE HEALTH COHORT

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**Introduction** More females than ever are taking up shift working. We have previously shown that asthma is more common in night shift workers and female night shift workers in particular are significantly more at risk. Why this is the case is unknown. We aim to investigate further in newly available Our Future Health (OFH) data by examining how these relationships vary by age and by use of hormonal contraception.
**Methods** We are one of the first groups to use the OFH dataset; a large cohort of over 1.7 million participants (17–95 years; 57.2% female; 10% non-White), representative of the UK in terms of ethnicity and age, recently made available for health research. OFH's large size and greater age and ethnic diversity, compared to other cohorts,make it well-suited to address our objectives. We use logistic regression, adjusted for demographic and lifestyle factors, to describe sex differences in prevalent asthma by shift work status, and explore interactions by age and contraceptive use.
**Results** Compared to female day workers, females working irregular shift work including nights had higher covariate-adjusted odds of asthma (**A;** OR: 1.08 (95% CI 1.02–1.15)) whereas this shift pattern was linked to a lower likelihood of asthma in males (OR 0.93 (95% CI 0.87–1.00); sex interaction p-value<0.01). Sex-stratified covariate-adjusted relationships between shift work status and prevalent asthma did not vary significantly across age groups (**B**), p-interaction: males p=0.07; females p=0.99. In female participants, use of hormonal contraception was associated with higher asthma prevalence when compared to non-users (**C**; e.g. combined pill, OR 1.09 (95% CI 1.06–1.12)), but use of the combined pill use did not fully explain the relationship between shift-work and prevalent asthma (**D**).
**Conclusion** We confirm that shift work is associated with higher likelihood of asthma in females and show, for the first time, lower likelihood of asthma in male shift workers compared to male day workers. Although contraceptive use was linked to a higher likelihood of asthma, contraceptive use did not explain the higher risk of asthma in female shift workers. Further research is required to understand the basis of observed sex differences.




**Abstract M17 Figure 1** Adjusted odds (95% CI) of asthma. **A:** By current shift work exposure and stratified by sex. **B:** By current shift work exposure and stratified by sex and quintiles of age. **C:** By contraception use. **D:** By current shift work exposure and stratified by use of the combined pill. All analyses adjusted for age (except for in B), ethnicity, household income, alcohol status and intake, days exercised (walked, moderate and vigorous), length of working week and chronotype

## M18 SHIFTING COPD AND ASTHMA CARE LEFT: ANALYSIS OF THE 'ONE STOP' COMMUNITY DIAGNOSTIC CLINIC IN THE WEST OF IRELAND

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**Introduction** In Ireland, the Sláintecare programme has identified chronic disease as a priority, aiming to transition asthma and chronic obstructive pulmonary disease (COPD) care from tertiary care to patient-centred primary care and community hubs. This includes community-based resources: GP-direct access spirometry, community diagnostic clinics (CDC's) and community pulmonary rehabilitation (PR). We created a 'one-stop' CDC for patients with suspected or confirmed asthma or COPD, close to where they lived. Patients availed of spirometry, consultant, clinical nurse specialist (CNS) and physiotherapist review at the one visit.

**Methods** A retrospective study of patients who attended the CDC was compiled from its inception in April 2023 to April 2025. The source of referral, spirometry, diagnosis, and investigations was analysed from their electronic health record.

**Results** 668 new patients were seen at the CDC. 64% referrals were from GP's directly to the CDC, avoiding hospital waitlists. Suspected COPD was the most common clinical query from GPs. After CDC, 302 patients (45.2%) were diagnosed with asthma, 151 were diagnosed with COPD (22.6%) & 70 diagnosed with ACOS (10.5%). Of the 151 COPD patients, 146 had diagnostic spirometry, whereby 118 (80.8%) had GOLD 1/2 COPD and 28 patients (19.2%) had GOLD 3/4 COPD. 21 patients received an alternate respiratory diagnosis (17 bronchiectasis, 3 lung cancer, 1 IPF). The remaining patients received an alternative diagnosis (normal, sinusitis, GORD or obesity).

89% of patients availed of CNS review, whilst 38.5% availed of physiotherapy services. 82 patients (12%) were referred to PR.

420 (62.9%) patients had medication changes, of which 327 (77.9%) were escalated and 93 (22.1%) were de-escalated.

Most patients were discharged medically after one visit, with respiratory CNS and physiotherapy follow-up arranged, where appropriate. 78 patients (11.7%) required further investigations including CT Thorax, full PFT's, bronchoscopy etc.

**Conclusions** The 'one stop' CDC is an effective patient-centred model, whereby patients avail of diagnosis, treatment and self-management plan at one community clinic visit. The 'shift left' model away from hospitals improves GP access to specialist respiratory care. GPs are considering COPD as a diagnosis for their dyspnoeic patients, highlighting the value of timely specialist respiratory care in community settings.

## M19 REAL-LIFE EVALUATION OF FORCED EXPIRATORY RATIOS WITH ASTHMA EXACERBATIONS AND SYMPTOMS

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**Background** Small airways dysfunction (SAD) is an important treatable trait in persistent asthma but remains poorly captured by conventional spirometry. The ($FEV_3$-$FEV_1$)/FVC and $FEV_3$/$FEV_6$ ratios have been proposed as novel markers of peripheral airflow limitation.

**Objective** We investigated the relationship between ($FEV_3$-$FEV_1$)/FVC and $FEV_3$/$FEV_6$ with severe exacerbation frequency and symptom control in patients with moderate-to-severe asthma.

**Methods** Clinical, physiological and biomarker data were collected from adults with GINA-defined moderate-to-severe asthma visiting a specialist severe asthma centre in Scotland. Associations between spirometric and oscillometric parameters with clinically relevant outcomes were retrospectively analysed using multivariate models.

**Results** 294 patients were included in the analysis. The ($FEV_3$-$FEV_1$)/FVC and $FEV_3$/$FEV_6$ ratios were not significantly associated with asthma symptoms or severe exacerbation rate. Conversely, traditional measures such as $FEV_1$/FVC [adjusted odds ratio (95%CI) 2.11 (1.27,3.50) p<0.01], $FEF_{25–75}$/FVC [2.11 (1.27,3.50) p<0.01], and oscillometric Rrs5–20 [OR 1.90 (1.14,3.16) p<0.05] were significantly associated with ≥2 exacerbations in the previous year (figure 1). High coefficient of determination ($r^2$) values were observed between $FEV_1$/FVC with (a) ($FEV_3$-$FEV_1$)/FVC $r^2$=0.61 and (b) $FEV_3$/$FEV_6$ $r^2$=0.83, suggesting overlapping aspects of lung function being measured. There was a weak correlation between $FEV_1$/FVC and Rrs5–20 values ($r^2$=0.08) indicating that these parameters assess unique aspects of lung function and mechanics.

**Conclusion** In moderate-to-severe asthma, ($FEV_3$-$FEV_1$)/FVC does not independently predict clinical outcomes and offers no advantage over conventional lung function assessments.

## M20 CAN MEASURES OF SMALL AIRWAY DYSFUNCTION AID WITH THE DIAGNOSIS OR MANAGEMENT OF ASTHMA EXACERBATIONS? – A SYSTEMATIC REVIEW

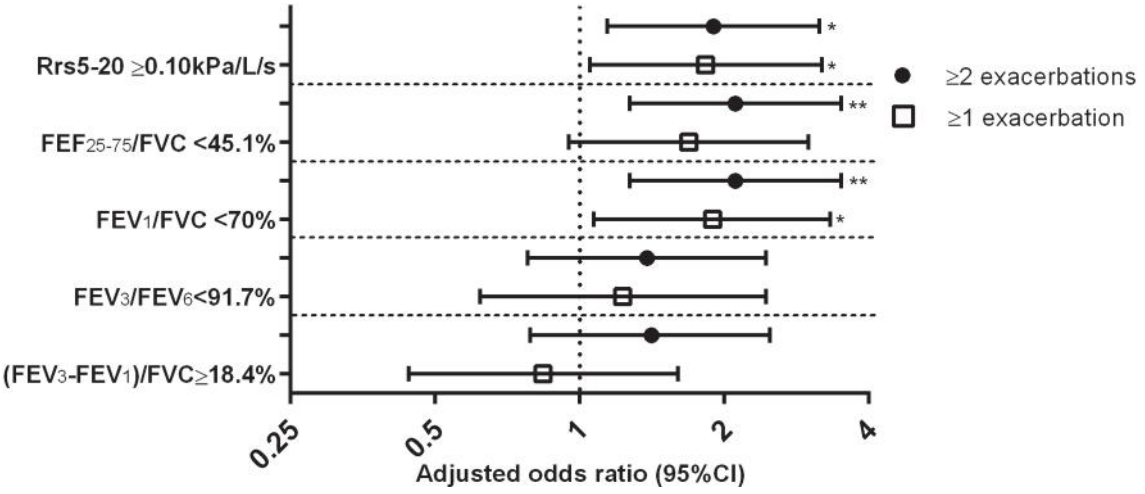
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10.1136/thorax-2025-BTSabstracts.504

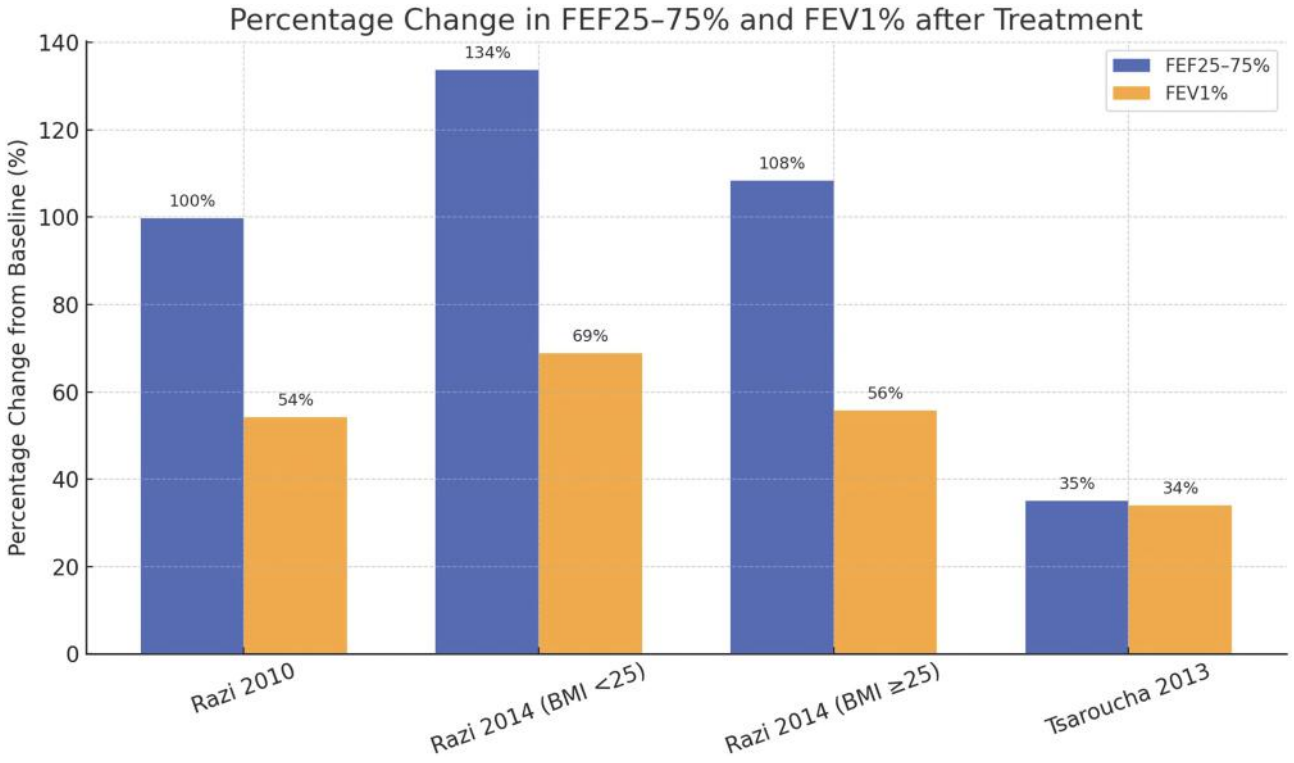
**Introduction** Asthma exacerbations are diagnosed and monitored using clinical features and forced airways manoeuvres which can be difficult for patients to perform. Tests assessing small airways function might provide more nuanced information about the exacerbation and response to treatment, and this systematic review (SR) evaluated the evidence to support these tests during adult asthma exacerbations.

**Methods** The SR followed PRISMA guidelines and was registered with PROSPERO (CRD42024494994). MEDLINE, EMBASE, CENTRAL, and CINAHL with no time or language restrictions. Risk of bias was assessed using RoB 2.0 (RCTs) and the Newcastle-Ottawa Scale (observational studies). A narrative synthesis was conducted due to methodological heterogeneity.

**Results** Seven studies (six observational, one RCT) involving 634 adults with asthma exacerbations met inclusion criteria. Five studies assessed Forced Expiratory Flow (FEF) at 25%–75% of the pulmonary volume, one reported FEF25/50/75, and one used multiple-breath washout (MBW). Most studies were exploratory and limited by small sample sizes, heterogeneous methodologies, and incomplete statistical reporting, including inconsistent use of group comparisons and varied follow-up durations. No study used small airway tests as a primary diagnostic tool, and statistical significance was inconsistently reported. Risk of bias was notable across all studies.




**Abstract M19 Figure 1** Adjusted odds ratios (95%CI) between spirometry and oscillometry parameters in relation to exacerbation frequency




**Abstract M20 Figure 1** Percentage change in FEF25–75 and FEV1 from pre- to post-treatment in three studies. Changes were calculated from reported group mean values unless otherwise specified. Units shown are percent predicted (% predicted)

All studies reported a good ability for patients to perform tests of small airways during exacerbations. All reported impaired small airway function during exacerbations with improvements at follow-up (taken 2 to 8 weeks after the initial presentation). Most reported a greater improvement in FEF25–75 than in FEV1 (Increments in FEF25–75 of 152%, 124%, and 99.7% and 80%, 62%, and 54% in FEV1). MBW indices were elevated during exacerbations and declined during recovery, with a strong inverse correlation between Sacin and $FEV_1$ ($\rho$=–0.67, p=.006). One RCT (n=36) evaluated bronchodilator response with or without non-invasive ventilation, showing modest post-treatment changes in FEF25–75.

**Conclusion** Tests of small airway function are feasible during asthma exacerbations and may detect clinically relevant changes. It remains unclear which test may be optimal and whether tests of small airways function during exacerbation enhance early diagnosis or treatment monitoring.

## M21 IMPACT OF THE SENTINEL PROGRAMME ON ASTHMA CARE ACROSS SIX PRIMARY CARE NETWORKS IN ENGLAND

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**Introduction** In the UK, short-acting β2-agonist (SABA) overuse in asthma remains common; however, this treatment strategy fails to address underlying inflammation and is associated with increased risk of exacerbations and asthma-related deaths. The SENTINEL Program aimed to identify and address SABA over-use through supported asthma guideline implementation within six English primary care networks (PCNs). Here we report on the effectiveness of SENTINEL to decrease SABA overuse and improve patient outcomes.

**Methods** Data were analysed for adult asthma patients within the six participating PCNs from November 2018 to October 2023. The analysis included three groups: i) the overall asthma population (Overall), ii) patients prioritized for clinic review due to SABA overuse (Review), and iii) patients newly prescribed MART during the review (MART). Outcomes (SABA use, exacerbations defined by acute OCS bursts) were assessed using electronic medical records (EMIS or SystmOne) before and after SENTINEL implementation. EMIS/SystmOne data were pooled where possible but reported separately for MART group and exacerbations due to coding differences. Descriptive statistics were used to describe prescription patterns. Three-level generalized linear mixed models were applied to evaluate changes pre- and post-implementation.

**Results** The Overall, Review, and MART groups included 14,202, 3,208 and 878 patients, respectively. In the Overall group, mean (SD) age was 52.1 (17.9) years, 58.9% were female, and 15.0%, 30.2% and 52.3% were current-, former- or never-smokers, respectively. Most common comorbidities were hypertension (27.5%), frailty (15.2%) and obesity (14.9%). Reduction in SABA prescribing was observed, with adjusted mean (95% confidence interval [CI]) reductions of 1.94 (1.92–1.97) and 4.15 (4.10–4.21) prescriptions per patient per year in Overall and Review groups, respectively (p<0.01). In the MART group, the reduction was 6.34 (6.19–6.48) in EMIS and 6.52 (6.38–6.67) in SystmOne (p<0.01) practices. Exacerbations decreased post-implementation in the Overall group by −7.6 (95% CI −5.8 to −9.4, p<0.01)

events per 100 patients per year in EMIS practices, and −3.6 (95% CI −2.7 to −4.4, p<0.01) events per 100 patients per year in SystmOne practices.

**Conclusions** SENTINEL improved prescribing patterns and reduced exacerbations, supporting the importance of implementing evidence-based guidelines into practice to achieve clinical benefits.

## M22 KEY INDICATORS OF CARE FOR PATIENTS HOSPITALISED WITH ASTHMA AND COPD WITH COMORBID MENTAL HEALTH OR COGNITIVE IMPAIRMENT: PRELIMINARY FINDINGS FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME (NRAP)

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10.1136/thorax-2025-BTSabstracts.506

**Introduction** Anxiety and depression are common respiratory comorbidities associated with poor disease control and risk of exacerbation.[1] Cognitive dysfunction and brain pathology have also been reported in people with chronic lung disease.[2] The link between these important extrapulmonary comorbidities and delivery of respiratory care is poorly understood.

**Aims** Describe the prevalence of mental health and cognitive impairment in a national cohort of hospitalised adults with asthma and COPD and explore associations with key performance indicators (KPIs) for the delivery of inpatient care.

**Methods** This analysis used data from the National Respiratory Audit Programme (NRAP), which reports on hospitalised adults in England/Wales with COPD or asthma exacerbations and discharged 01/04/2023–31/03/2024. We compared demographic variables and KPIs using t-tests for categorical variables and Kruskal-Wallis test for age. Preliminary, unadjusted logistic regression was performed with mental health/dementia as the independent variable (baseline=none), and dependent variable the delivery of KPI.

**Results** 38,969 adult COPD patients and 13,663 adult asthma patients were included. 26.1% asthma and 29.2% COPD patients had a documented history of mental health disease or cognitive impairment, with prevalence described in table 1. There was a female predominance in all groups. Patients with a mental health comorbidity were more likely to be current smokers. The severity of COPD exacerbations (based on NEWS2 score) was lower in the dementia group. Conversely, near-fatal asthma exacerbations were more frequent in the dementia group. Asthma and dementia/cognitive impairment were associated with lower documented delivery of specialist review, acute assessment and discharge bundle. In COPD, severe mental health and dementia/cognitive impairment was associated with lower documented spirometry, acute NIV, specialist review and discharge bundle.

**Conclusions** Up to one third of patients with asthma and COPD have a mental health or cognitive impairment documented during acute admission for exacerbation. The presence of these comorbidities is associated with lower odds of receiving basic respiratory care. Further work is needed to support

**Abstract M22 Table 1** Prevalence of documented diagnoses of anxiety, depression, severe mental health and dementia/cognitive decline in those hospitalised with exacerbations of asthma and COPD in NRAP between 01/04/2023–31/03/2024. Unadjusted logistic regression for the association between mental health diagnoses and delivery of KPIs

| | | Asthma n= 13663 | | | | | |
|---|---|---|---|---|---|---|---|
| | | None | Anxiety | Depression | Anxiety & Depression | Severe mental health | Dementia or Cognitive impairment |
| Total n (%) | | 10102 (73.9) | 793 (5.8) | 1125 (8.2) | 1020 (7.5) | 340 (2.5) | 283 (2.1) |
| KPI Unadjusted OR (CI 95%, P-value) | **1. Respiratory review <24hrs** | - | 1.13 (0.99–1.30) | 1.05 (0.94–1.18) | 1.29 (1.14–1.45) | 1.03 (0.85–1.25) | 0.55 (0.43–0.69) |
| | **2. PEF taken <1hr** | - | 1.18 (0.95–1.46) | 0.93 (0.76–1.13) | 1.01 (0.82–1.23) | 0.94 (0.65–1.31) | 0.44 (0.23–0.75) |
| | **3. Systemic steroids <1hr** | - | 1.01 (0.82–1.23) | 0.99 (0.83–1.18) | 1.00 (0.84–1.20) | 1.15 (0.85–1.54) | 0.58 (0.39–0.82) |
| | **4. Key components of DB on discharge** | - | 1.05 (0.92–1.20) | 0.99 (0.88–1.11) | 1.16 (1.03–1.30) | 0.83 (0.68–1.01) | 0.57 (0.45–0.71) |
| | **5. Smoking cessation offered** | - | 0.92 (0.68–1.27) | 0.90 (0.70–1.18) | 1.16 (0.90–1.52) | 0.90 (0.64–1.29) | 0.42 (0.18–1.02) |
| | **6. Discharged on ICS** | - | 0.86 (0.69–1.08) | 0.97 (0.80–1.19) | 0.95 (0.78–1.17) | 1.27 (0.89–1.89) | 1.16 (0.79–1.78) |
| | | COPD n= 38969 | | | | | |
| | | None | Anxiety | Depression | Anxiety & Depression | Severe mental health | Dementia or Cognitive impairment |
| Total n (%) | | 27600 (70.8) | 2806 (7.2) | 3025 (7.8) | 2466 (6.3) | 914 (2.3) | 2158 (5.5) |
| KPI Unadjusted OR (CI 95%, P-value) | **1. Acute NIV <2hrs** | - | 1.05 (0.82–1.35) | 0.91 (0.70–1.18) | 1.03 (0.79–1.34) | 0.64 (0.43–0.92) | 0.74 (0.51–1.06) |
| | **2. $O_2$ prescribed to targeted sats** | - | 1.79 (1.19–2.84) | 1.12 (0.80–1.61) | 1.54 (1.02–2.44) | 1.95 (0.95–4.96) | 0.91 (0.63–1.37) |
| | **3. Spirometry result available** | - | 1.19 (1.10–1.28) | 1.17 (1.09–1.26) | 1.17 (1.08–1.27) | 0.69 (0.60–0.79) | 0.65 (0.60–0.71) |
| | **4. Smoking cessation offered** | - | 1.05 (0.92–1.21) | 1.12 (0.99–1.26) | 1.23 (1.08–1.40) | 0.99 (0.83–1.18) | 0.72 (0.59–0.87) |
| | **5. Respiratory review <24hrs** | - | 1.28 (1.18–1.39) | 1.12 (1.04–1.22) | 1.23 (1.13–1.34) | 1.07 (0.94–1.23) | 0.65 (0.59–0.71) |
| | **6. Key components of DB on discharge** | - | 1.29 (1.19–1.40) | 1.06 (0.98–1.16) | 1.20 (1.10–1.32) | 0.94 (0.81–1.10) | 0.65 (0.58–0.72) |

CI, confidence interval; COPD, chronic obstructive pulmonary disease; DB, discharge bundle; ICS, inhaled corticosteroid; KPI, key performance indicators; NIV, non-invasive ventilation; OR, Odds ratio; PEF, peak expiratory flow; sats, peripheral oxygen saturations.

these findings with adjusted analyses and investigate impact on clinical outcomes.


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## M23 OBESITY AND ANXIETY IMPACT ON SYMPTOM BURDEN IN NEWLY DIAGNOSED AND MISDIAGNOSED ASTHMA

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**Introduction** Anxiety and obesity are associated with adverse asthma outcomes. We aim to describe their impact on symptom burden in newly diagnosed and misdiagnosed asthma patients.

**Method** We used data collected from two independent cohorts. Whilst symptomatic, untreated individuals with GP-suspected asthma were recruited into the RADicA study (Manchester, UK), the SMART study (Dublin, Ireland) included patients with a diagnostic label of asthma who have persistent symptoms despite inhaled corticosteroids (ICS), both undergoing diagnostic evaluation using objective testing. Symptom burden was assessed using the Asthma Control Questionnaire (ACQ) and Asthma Control Test (ACT). Asthma diagnosis was determined by clinical history, objective assessments (blood eosinophil count [BEC], fractional exhaled nitric oxide [FeNO], bronchodilator reversibility and bronchial challenge testing) and treatment response. All participants underwent anthropometric measurements and provided self-reported history of anxiety.

**Results** Of 137 symptomatic adults (>16 yrs, 60.6% female) recruited to the RADicA study, 59% had asthma. Anxiety and/or obesity (A/O) were more prevalent in symptomatic non-asthmatic group (60%) compared to asthmatic group (47%). Asthmatic individuals with A/O had higher symptom burden both pre- and post-ICS, independent of FeNO, BEC or lung function (table 1), and were less likely to achieve well-controlled symptoms post-ICS (OR [95% CI]: 0.28 [0.22–0.84], p = 0.021). In SMART study, of 113 individuals with a diagnostic label of asthma who remained symptomatic despite ICS, 44% did not have asthma. The prevalence of A/O was higher in symptomatic patients without asthma (60%) compared to asthmatic group (55%). Although ACT scores did not differ between asthmatic individuals with and without A/O (table), those with co-existing obesity self-rated their asthma control worse (p = 0.034). Symptoms became controlled in those without asthma after addressing treatable traits and ICS cessation/reduction (baseline ACT: 18[14–23] and post-ICS reduction: 21[15–24]).

**Conclusion** A/O are common in suspected, confirmed and misdiagnosed asthma, and are associated with greater symptom burden independent of objective disease markers. Symptom-based tools cannot reliably distinguish asthma from comorbidity-driven symptoms, leading to potential overtreatment. Addressing non-asthma related treatable traits can improve outcomes and reduce unnecessary steroid use. Objective biomarkers beyond subjective symptom measures are needed to enable personalised pathophysiology-based asthma management.

## M24 THE PREVALENCE OF PSYCHOLOGICAL SYMPTOMS IN SEVERE AND DIFFICULT ASTHMA

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**Background** The prevalence of anxiety and depression in severe and difficult asthma is poorly understood as validated assessment tools assessing psychological symptoms are infrequently used in this population. It is recognised that anxiety and depression are associated with increased respiratory symptoms and there is therefore a risk of increased asthma

**Abstract M23 Table 1** Impact of obesity and anxiety on symptom burden and object disease markers in adult patients with confirmed asthma in RADicA and SMART studies

| | RADicA (n=77) | | | SMART (n=63) | | |
|---|---|---|---|---|---|---|
| | No obesity and/or anxiety n=40 | Presence of obesity or/and Anxiety n=37 | p-value | No obesity and/or anxiety n=30 | Presence of obesity or/and Anxiety n=33 | p-value |
| | **Baseline (pre-ICS)** | | | **Baseline (at recruitment, on ICS)** | | |
| ACQ-5, points | 0.8 (0.4–1.6) | 1.6 (1–2.6) | **0.003** | | | |
| ACT scores | | | | 17 (12–21) | 16 (13–18) | 0.53 |
| BEC, x$10^9$ cells/L | 0.3 (0.16–0.7) | 0.26 (0.16, 0.41) | 0.333 | 0.33 (0.19–0.49) | 0.25 (0.16–0.35) | 0.25 |
| FeNO, ppb | 49.5 (23.8–92.2) | 48 (15–91) | 0.564 | 33 (22–50) | 24 (16–0) | 0.13 |
| FEV1% predicted,% | 94 (84.5–103) | 90 (81.5–97.5) | 0.209 | 85 (65–111) | 77 (65–119) | 0.80 |
| | **Post ICS (Flixotide 200 mcg twice daily)** | | | **Post ICS Relvar (92 or 184 mcg daily)** | | |
| ACQ-5, points | 0.2 (0–0.8) | 0.7 (0.2–1.2) | **0.020** | | | |
| ACT score | | | | 21 (15–23) | 19 (14–21) | 0.25 |
| BEC, x$10^9$ cells/L | 0.31 (0.14, 0.42) | 0.24 (0.14, 0.3) | 0.467 | 0.29 (0.23–0.50) | 0.23 (0.15–0.44) | 0.19 |
| FeNO, ppb | 21 (17–32) | 18 (10–31) | 0.233 | 22 (17–30) | 21 (14–27) | 0.41 |
| FEV1% predicted,% | 96 (86–106) | 95 (87–101) | 0.653 | 85 (72–94) | 80 (66–96) | 0.73 |

All results presented as ***Median (IQR)***

medication, healthcare utilisation and worse outcomes for asthma patients with co-morbid anxiety and depression.

**Methods** The Hospital Anxiety and Depression Scale (HADS) questionnaire was collected from patients with severe and difficult asthma referred to a specialist asthma clinic in the UK. Key demographic and clinical characteristics including ACQ-6 score and FeNO level were recorded and compared between patients reporting levels of anxiety and/or depression above the validated HADS threshold of 8.

**Results** 300 patients (63% female, median age 52) completed the HADS. The median (IQR) ACQ-6 score was 1.83 (0.83–2.83) and FeNO was 29.5ppb (17–53). A HADS of ≥8 was reported by 50% patients for anxiety, 37% for depression and 31% for both. Compared to individuals reporting no psychological symptoms, patients with anxiety were younger (45 [32–67] vs 58 [38–69], p<0.001), were more likely to be female (61% vs 39%, p=0.001) and had a higher ACQ6 2.33 [1.17–3.50] vs 1.17 [0.33–2.17] despite equivalent FeNO level (median 29ppb in both groups). In a multi-linear regression analysis anxiety increased the ACQ6 by 0.84 points.

**Conclusion** Co-morbid psychological symptoms are very common in severe and difficult asthma and significantly contribute to poor symptom control. A multi-disciplinary approach including psychological interventions has the potential to improve asthma symptom control, morbidity and to reduce asthma-related healthcare costs.

## M25 IMPACT OF SEVERE ASTHMA SERVICE ON BREATHLESSNESS: COMPARISON OF MEDIAN ACQ AT SERVICE ENTRY WITH LATEST AVAILABLE MEDIAN ACQ AFTER TREATMENT DID NOT REACH THE MINIMAL CLINICALLY IMPORTANT DIFFERENCE ACROSS 1628 PATIENTS WITHIN ASTHMA AND BREATH MASTERY LEARNING HEALTH SYSTEM

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Context: the challenge of unresolved breathlessness in asthma

Breathlessness is the predominant symptom driving the asthma pathway. In primary care, people with asthma who remain breathless despite treatment escalation are typically referred to secondary care. Notably, Type 2 (T2)-high patients may remain breathless even when biologic therapy successfully controls inflammatory biomarkers. In the Type-2 biomarker versus symptom-based treatment trial (Lancet Respir Med 2021; 9:57–61), a substantial number of patients with well-controlled biomarkers were reluctant to reduce treatment, highlighting the disconnect between biomarker control and symptom burden.

**Methods** We analysed data from 1628 patients who attended the Severe Asthma Service in autumn 2024. Information from each patient's earliest contact with the service was extracted to reflect asthma status at the time of referral, while the most recent available data were used to assess the impact of tertiary care treatment (HRA IRAS 342426). Breathlessness was evaluated using the Asthma Control Questionnaire (ACQ), comparing scores at entry with the latest score available to understand the impact of treatment. An ACQ of ≤0.75 typically indicates good asthma control, while ACQ of ≥1.5 is indicative of poor asthma control.

**Results** The latest overall ACQ median (IQR) of 2.2 (1.2–3.2) demonstrated a statistically significant change compared to ACQ at service entry 2.4 (1.4–3.3). Patients started on biologics also demonstrated a statistically significant change in ACQ: 2.7 (1.7–3.5) vs 2.3 (1.2 -3.3). In both cases, the difference did not meet the ACQ minimally clinically important difference (MCID) of 0.5. In 561 patients in which the highest ever eosinophil was <0.3, median ACQ remained unchanged at 2.3 at both entry and follow-up.

**Discussion** Comprehensive data from a single centre demonstrate a post treatment median ACQ of 2.2 which is well above the cut-off for poor control. Though there was a statistically significant fall in ACQ with treatment in patients started on biologics, this did not meet the ACQ MCID, and the ACQ of 2.3 was also above the ACQ cut-off for good control of 0.75. These data support the Lind Alliance Breathlessness consensus that interventions to address non-T2 high breathlessness in severe asthma clinics is a research priority.

**Abstract M25 Table 1**

| | All (n=1628) | On biologics (n=448) | Eosinophil <0.3 (n=561) | Eosinophil >0.3 (n=619) |
|---|---|---|---|---|
| Age in years, median (IQR) | 54.9 (41.3–65.0) | 56.4 (46.5–64.9) | 54.5 (39.0–64.4) | 54.1 (39.0–65.6) |
| Males, n, % | 510 (31.3) | 106 (37.3) | 132 (22.2) | 212 (32.0) |
| BMI in kg/m2, median (IQR) | 30.9 (26.4–36.7) | 31.2 (27.1–37.2) | 31.4 (26.7–37.6) | 30.3 (25.9–36.3) |
| Baseline FeNO, median (IQR) | 20.0 (11.0–43.0) | 26.5 (15.0–56.0) | 15.0 (8.0–24.0) | 23.0 (12.0–48.0) |
| Latest FeNO, median (IQR) | 21.0 (12.0–42.8) | 31.0 (17.0–55.0) | 15.0 (9.0–26.0) | 24.0 (13.0–44.0) |
| Baseline ACQ, median (IQR) | 2.4 (1.4–3.3)* | 2.7 (1.7–3.5)* | 2.3 (1.4–3.5) | 2.1 (1.0–3.0) |
| Latest ACQ, median (IQR) | 2.2 (1.2–3.2)* | 2.3 (1.2–3.3)* | 2.3 (1.3–3.3) | 2.0 (1.2–3.0) |

*Within–group comparisons of ACQ scores showed statistically significant differences for the overall cohort and for those on biologics (Wilcoxon signed–rank test, adjusted with Bonferroni correction)

## M26 ARE SYMPTOM QUESTIONNAIRES A USEFUL TOOL IN THE ASSESSMENT OF INDUCIBLE LARYNGEAL OBSTRUCTION

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**Introduction** Inducible Laryngeal Obstruction (ILO) is an inappropriate adduction of the vocal folds is frequently misdiagnosed as asthma due to symptom similarity. The current standard assessment tool for ILO is laryngoscopy.

In our airways service, as part of routine care, patients complete symptom questionnaires to assist in the differential diagnosis. However, with overlapping symptoms and patient awareness, there is uncertainty as to the value of questionnaires in predicting a diagnosis of ILO. Previous research has suggested symptom questionnaires lack such sensitivity.

**Aim** To assess the relationship between questionnaires and laryngoscopy-confirmed ILO diagnosis in referred patients.

**Methods** Data from our contemporaneous database was preliminarily analysed for 165 referred patients with symptoms of breathlessness over a 16-month period (November 2023-March 2025). Responses on the following questionnaires were compared with the outcome of the ILO assessment: Vocal Cord Dysfunction Questionnaire (VCDQ), Visual Analogue Scale (VAS), Newcastle-Laryngeal Hypersensitivity (N-LHS) and Leicester Cough Questionnaire (LCQ).

Patient demographic data and other comorbidities were also compared with outcome of laryngoscopy.

**Results** On laryngoscopy assessment, 68 (41%) patients were diagnosed with ILO via laryngoscopy, whereas 97 (59%) were not diagnosed with ILO following video-laryngoscopy.

Comparison of total questionnaire scores between ILO and non-ILO groups showed no significant differences: VCDQ ($U=1610.5$; $p=0.3541$); VAS ($U=2200.5$; $p=0.4411$); N-LHS ($U=2058$; $p=0.5360$); LCQ ($U=1610.5$; $p=0.3541$).

Demographic data showed no difference between these groups on age. There were more females than males in the ILO group, but this was not a significant difference ($\chi 2=2.89$; $df=1$; $p= 0.089$). Asthma was significantly more common in the ILO group (($\chi 2= 4.58$; $df=1$; $p=0.032$). There were no differences between groups for other comorbidities including reflux, rhinitis and obstructive sleep apnoea.

**Conclusions** There were no significant differences between the ILO and non-ILO groups on the assessment questionnaire scores in a population of adults referred to a tertiary centre for assessment. Preliminary analysis of this data indicates that symptom scores alone cannot predict a diagnosis of ILO. Such tools may guide clinical discussion or raise suspicion of ILO, acting as a prompt to refer for assessment with laryngoscopy.

## M27 A PAIN IN THE NECK? HEALTHY VOLUNTEER ACCEPTABILITY OF TRANS LARYNGEAL ULTRASOUND TO ASSESS VOCAL FOLD MOVEMENT IN MIMICKED INDUCIBLE LARYNGEAL OBSTRUCTION

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**Introduction** Inducible Laryngeal Obstruction (ILO) describes an inappropriate narrowing of the vocal folds on inspiration. ILO is frequently misdiagnosed as asthma, leading to significant patient morbidity and healthcare costs. Obtaining an accurate and timely diagnosis is essential.

The current reference standard for the diagnosis of ILO is a video-laryngoscopy, which is an invasive procedure that is costly, can be poorly tolerated, and timely access is often challenging. Consequently, developing alternative, less-invasive assessment methods is a research priority.

As highlighted by the MRC framework for complex interventions, acceptability is a critical consideration in the design and evaluation of healthcare interventions and can influence their successful implementation, as acceptability may impact on patient adherence and clinical outcomes.

**Aims** This study aimed to determine the acceptability of translaryngeal ultrasound (TLUS) among healthy volunteers. These data form the initial stage of a larger feasibility study investigating TLUS as a non-invasive screening tool for ILO.

**Methods** Healthy NHS staff (n=29; mean age 40; 77% female) with no history of asthma, ILO, laryngeal pathology, or neck surgery were recruited. Following informed consent, participants underwent a standardised TLUS protocol (designed and previously published by the author/researcher; Slinger 2024) to visualise the vocal folds, including during mimicked ILO. Participants anonymously completed an electronic questionnaire immediately post-procedure to evaluate the acceptability of the TLUS assessment.

**Results** Acceptability of TLUS was very high. All participants reported the procedure was comfortable, stated they would undergo it again, and would recommend it to others. Qualitative data analysis revealed key themes contributing to acceptability, including the non-invasive nature of the scan, the importance of clear explanations and reassurance from the clinician, and the overall ease and speed of the procedure.

**Conclusion** This study provides important preliminary evidence that TLUS is a highly acceptable method for laryngeal assessment among healthy volunteers. These preliminary findings will help anticipate and address potential acceptability barriers, supporting the progression to a future clinical trial to evaluate TLUS in patients with suspected ILO.

Ethical approval is in place for this study

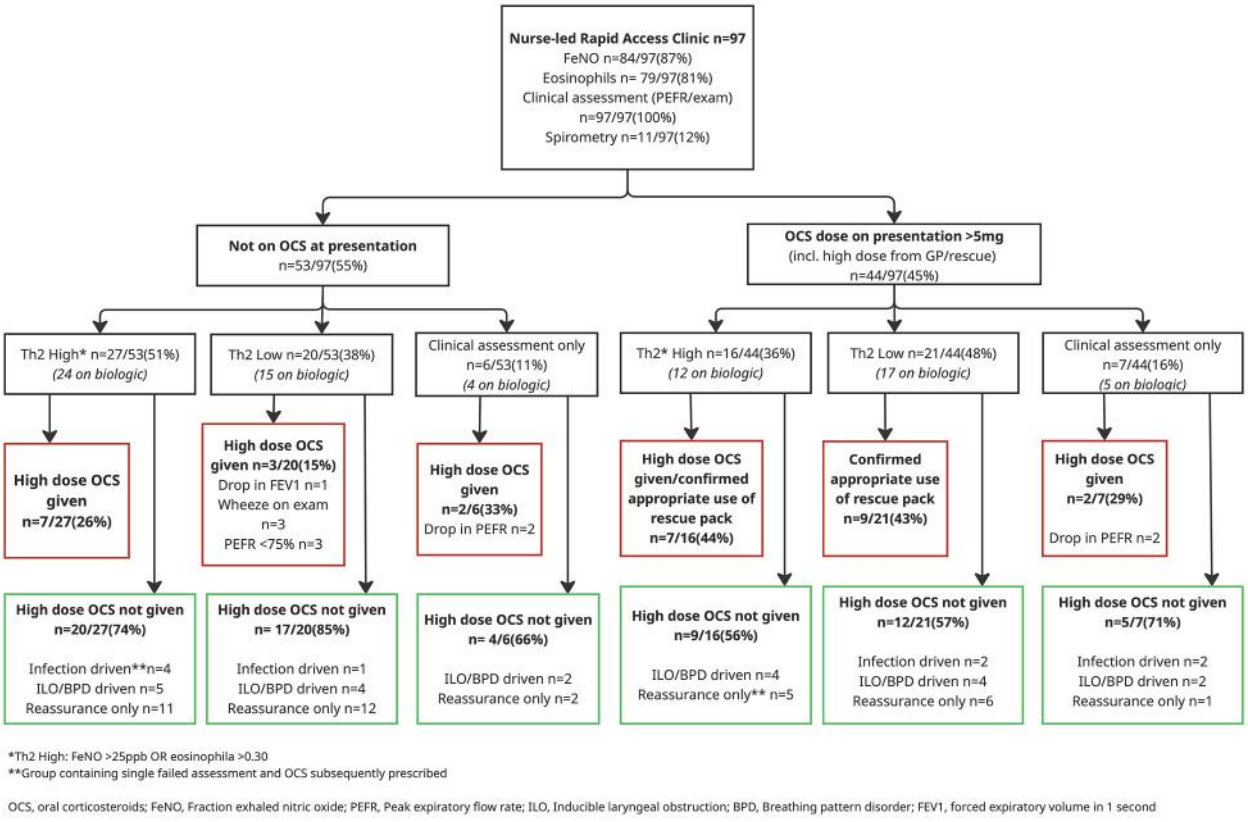
## M28 IMPACT OF BIOMARKER LED RAPID ACCESS CLINICS ON EXACERBATION MANAGEMENT IN A SEVERE ASTHMA POPULATION. FINDINGS FROM THE BRISTOL COMPLEX AIRWAYS SERVICE

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**Introduction** Biologic therapies have revolutionised asthma management by reducing acute exacerbations and oral corticosteroids (OCS) exposure. Asthma exacerbations are unpredictable, heterogenous events. Rapid biomarker assessment including type 2 inflammation (Th2) ensures targeted use of OCS and optimisation of treatable traits.[1] We describe a pragmatic, biomarker-led rapid access service at a regional complex airways service designed to enable timely access to objective specialist assessment.

**Methods** The Complex Airways service at North Bristol NHS Trust (UK) implemented a rapid access, face-to-face, nurse-led clinic for assessment of acute asthma exacerbations. Patients were assessed for clinical signs including wheeze, blood




**Abstract M28 Figure 1** Flowchart of exacerbation management by oral corticosteroid use prior to assessment

eosinophils, fraction exhaled nitrous oxide (FeNO), peak expiratory flow rate (PEFR) and spirometry when available. We reviewed outcome data for patients attending this clinic.

**Results** Between November 2023-March 2025, 97 patients were assessed. All patients presented with acute symptoms of exacerbation and had already initiated rescue high dose OCS or were due to start. 66% (n=64) were female and 78% (n=76) on an asthma biologic. Figure 1 describes biomarker results and subsequent OCS use following assessment. 69% (n=67) of patients were managed for one, or a combination of infection, inducible laryngeal obstruction (ILO) and breathing pattern disorder (BPD) and avoided high dose OCS. Only 2 patients, who were given antibiotics and no OCS, subsequently required rescue prednisolone. 31% (n=20) had already started high dose OCS and 9 patients were not adequately endotyped for assessment. 3 patients on no OCS and no evidence of Th2 inflammation were given high dose OCS (likely secondary to wheeze and drop in PEFR/FEV1). Other contributors to asthma symptoms including BPD and rhinosinusitis which were evident in 26% in the total population.

**Conclusions** Biomarker led rapid access clinics for asthma exacerbations reduces OCS use and identifies important treatable traits including infection, ILO and BPD. We have identified the need to standardise the biomarkers required for comprehensive assessment and recognise the importance of improving access to early intervention and education to prevent unnecessary use of rescue packs prior to assessment.

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## M29 THE EFFECTIVENESS OF BENRALIZUMAB IN THE MANAGEMENT OF CHRONIC EOSINOPHILIC PNEUMONIA

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**Introduction** Chronic eosinophilic pneumonia (CEP) is a rare condition associated with pulmonary and blood eosinophilia. Treatment to date has primarily comprised of prolonged courses of oral corticosteroids. Despite this, relapses are common and patients can often require treatment with recurrent courses or even maintenance oral steroids (mOCS) which is associated with significant morbidity. Limited data exists on the clinical effectiveness of eosinophil depleting biologic therapies in this patient cohort. Herein we report on our real-world experience utilising the anti-IL5R mAb benralizumab in patients with CEP.

**Methods** We performed a retrospective analysis of patients with severe eosinophilic asthma with a co-existing diagnosis of CEP who received treatment with benralizumab at our severe

asthma centre. CEP was diagnosed on the basis of CT evidence of subpleural consolidation in association with blood hyper-eosinophilia and exclusion of other causes. Baseline clinical characteristics and clinical outcomes at 1 year were evaluated. Data on the long-term CEP relapse rate was also collected.

**Results** 12 patients (58% female) with CEP were identified. The mean (SD) time from CEP diagnosis to biologic initiation was 32 (21.7) months. The mean (SD) number of CEP relapses prior to mAb initiation was 2.3 (1.8) which improved to 0.72 (1.3) (p=0.03) following mAb initiation with 83% patients having no CEP relapses. Median (IQR) BEC fell from 4.0 (1.7–4.7) to 0 (0–0) with benralizumab (p = 0.002). At baseline 83% of patients required mOCS primarily for relapsing CEP, with a mean dose of 14 mg (12.3). After 1 year, 60% of patients were able to discontinue mOCS therapy with only 4 patients requiring mOCS, all for adrenal insufficiency (p=0.004).

**Conclusion** Treatment with benralizumab resulted in a significant reduction in the rate of CEP relapse. In addition, there was a significant reduction in oral corticosteroid requirement with no patient requiring mOCS for CEP after 1 year of treatment. These findings support the use of eosinophil depletion with benralizumab as an effective steroid sparing treatment for patients with CEP.

## M30 DOUBLE OR NOTHING: EXPERIENCE OF DUAL MONOCLONAL ANTIBODY PRESCRIPTIONS AT A TERTIARY ASTHMA CENTRE

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10.1136/thorax-2025-BTSabstracts.514

**Introduction** Biological therapies, or monoclonal antibodies (MAbs) are increasingly used across a range of medical conditions, including severe asthma (SA). SA patients often have multiple co-morbidities, and it is increasingly common to see patients eligible for multiple MAbs for different diseases. At present, the risks of combining SA MAbs with those for other indications is unknown, with minimal evidence to support co-prescribing. We describe our experience of prescribing benralizumab alongside non-SA MAbs at a tertiary centre.

**Methods** We conducted a retrospective review of patients on SA Mab therapy alongside at least one other MAb, identifying patients through electronic prescribing records. We assessed duration of time on dual MAbs, adverse events (AEs), deterioration of disease control, and evidence of cross-team discussions.

**Results** We identified nine patients receiving a SA MAb alongside a non-SA MAb: four for osteoporosis, two for ankylosing spondylitis, and one each for rheumatoid arthritis, psoriasis, and inflammatory bowel disease (IBD). See table 1. Average time on dual MAb therapy was 25 months, and six patients (66%) remained on dual MAbs at time of writing.

One patient reported persistent flu-type symptoms, which is a documented adalimumab-related AE, and resolved on cessation of adalimumab. Eight patients retained good asthma control whilst receiving dual Mab therapy, with one patient stopping mepolizumab at 12 months due to poor asthma response. Only one patient had cross-directorate communication prior to commencement of dual MAbs.

**Conclusion** Dual MAb prescribing can be safe, with no reported AEs thought to be attributed to combined biological therapy in our cohort – specifically there were no reports of increased infection or concerning immunosuppressive features. Dual biologic prescribing, furthermore, did not appear to affect asthma control. Further real-world data are required, and use of national registry data could be used to generate more robust safety data.

**Abstract M30 Table 1** Table of dual Mab combinations

| SA MAb | Co-prescribed MAb | Indication | Duration on dual therapy (months) | AEs | Asthma control maintained? | Cross-directorate discussion |
|---|---|---|---|---|---|---|
| Benralizumab | Denosumab | Osteoporosis | 12 | Nil | Yes | No |
| Benralizumab | Denosumab | Osteoporosis | 24 | Nil | Yes | No |
| Benralizumab | Denosumab | Osteoporosis | 45 | Nil | Yes | No |
| Benralizumab | Denosumab | Osteoporosis | 44 | Nil | Yes | No |
| 5 Benralizumab | Adalimumab | Rheumatoid Arthritis | 24 | Nil | Yes | No |
| Benralizumab | Adalimumab | Ankylosing Spondylitis | 5 | Flu-like symptoms | Yes | No |
| Benralizumab | Bimekizumab | Ankylosing spondylitis | 11 | Nil | Yes | No |
| Mepolizumab | Secukinumab | Psoriasis | 12 | Nil | No – SA MAb stopped at 12 months | No |
| Benralizumab | Vedolizumab | IBD | 49 | Nil | Yes | Yes |

## M31 STRINGENT AMERICAN (ATS/ACAAI/AAAAI) ENDORSED 6-COMPONENT DEFINITION OF ASTHMA REMISSION IS NOT ACHIEVABLE IN A REAL-WORLD BENRALIZUMAB COHORT

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**Introduction** Goals for asthma treatment with biologics have progressed to clinical remission. General consensus[1] among experts is that remission should encompass absence of oral corticosteroids [OCS], well controlled symptoms and stable FEV1. A working group from the American Thoracic Society and College and Academy of Allergy, Asthma and Immunology has proposed a more stringent definition for remission on treatment.[2]

**Aim** To analyse ability of real-world asthma patients on benralizumab to achieve the stringent American remission criteria.

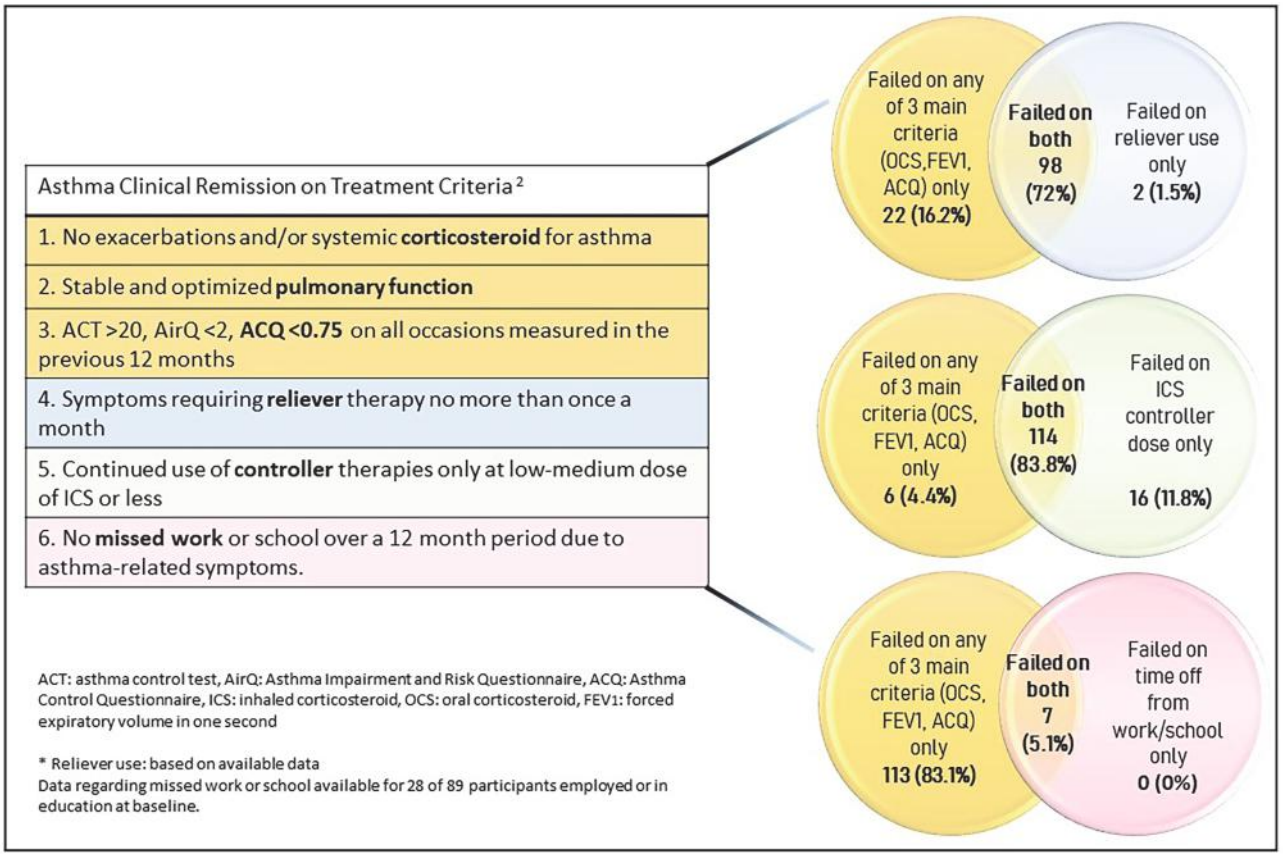
**Methods** BenRex is an open label, multicentre study of exacerbations while on benralizumab for severe eosinophilic asthma. Data from study visits were used to explore remission definitions.

**Results** At 1 year follow up, 0 of 136 subjects met the American remission criteria. Clinical response (50% reduction in OCS) was seen in 86%. If remission criteria were reduced to the 3 pertaining to OCS use, symptoms and FEV1, 16 (11.8%) would achieve remission. Figure 1 depicts numbers failing with each criteria.

**Conclusion** When a 6-component definition was used, no participant within this real-world biologic cohort achieved remission. If remission is to become a meaningful treatment goal in severe asthma, a more pragmatic definition may be required.

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**Abstract M31 Figure 1** Reasons for not meeting remission criteria using 6 component American remission definition

## M32 A NOVEL PATHWAY FOR PHARMACIST-LED STEPWISE DE-ESCALATION OF BACKGROUND ASTHMA THERAPY IN PATIENTS STABLE ON BIOLOGICS. EXPERIENCE FROM BRISTOL COMPLEX AIRWAYS SERVICE

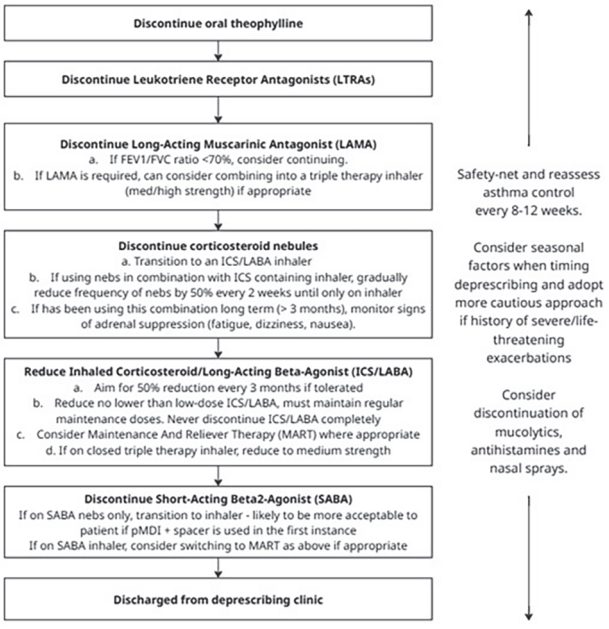
H Sakota, GW Nava, C Morgan, J Grenville, S Mulholland, N Leung, E Hunter, D Higbee, JW Dodd, R Shrimanker. *North Bristol NHS Trust, Bristol, UK*

10.1136/thorax-2025-BTSabstracts.516

**Introduction and Objectives** Biologic therapies have transformed severe asthma management, enabling improved control and reduced exacerbations. The Global Initiative for Asthma (GINA) guidelines[1] recommend considering stepwise reduction of background therapies in patients with sustained control on biologics but lacks tapering guidance for concurrent treatments. This novel and pragmatic pathway offers a strategy for de-escalation of multiple background therapies in well controlled biologic-treated patients within an NHS outpatient setting. The primary goal was to reduce treatment to the minimal effective dose of medication required to maintain clinical stability.

**Methods** A structured deprescribing pathway (SDP) was developed (see figure 1) and implemented in severe asthma patients stable on biologic therapy for >12 months in a pharmacist-led clinic. Deprescribing data was collected for patients reviewed between March 2024 and March 2025. The SDP begins with stepwise withdrawal of add-on controller therapies including montelukast, theophylline, long-acting muscarinic antagonists (LAMA) inhalers and nasal corticosteroids followed by incremental dose reduction of high-dose inhaled corticosteroids and long-acting β2-agonists (HD-ICS/LABA) inhalers and cessation of short-acting β2-agonists (SABA) and nebulisers. Patients were safety-netted, and a telephone review conducted at 8–12 weeks after each de-escalation to assess symptom control prior to the next reduction.

**Results** A total of 59 patients started the deprescribing pathway. So far, 100% (n= 11) theophylline, 98% (n= 45) montelukast, 82% (n= 49) LAMA inhalers, 85% (n= 13) steroid nasal sprays, 75% (n=4) of ICS nebulisers, 75% (n=4) SABA nebulisers were discontinued. 100% (n =8) SABAs were stopped as switched to Maintenance and Reliever Therapy (MART) and 91% (n=23) HD-ICS/LABAs were reduced to low or medium dose ICS/LABA.




**Abstract M32 Figure 1** Summary of deprescribing pathway

**Conclusions** This novel SDP appears to reduce polypharmacy associated with medication burden, side effects, and healthcare costs in patients well controlled on an asthma biologic. Next steps include expanding evaluation through quality metrics and patient reported outcomes.

### REFERENCE

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# TB: from bench to bedside

## M33 LOCALISATION AND ULTRASTRUCTURE OF MYCOBACTERIUM TUBERCULOSIS IN HUMAN MEDIASTINAL LYMPH NODES BY HIGH RESOLUTION ELECTRON MICROSCOPY

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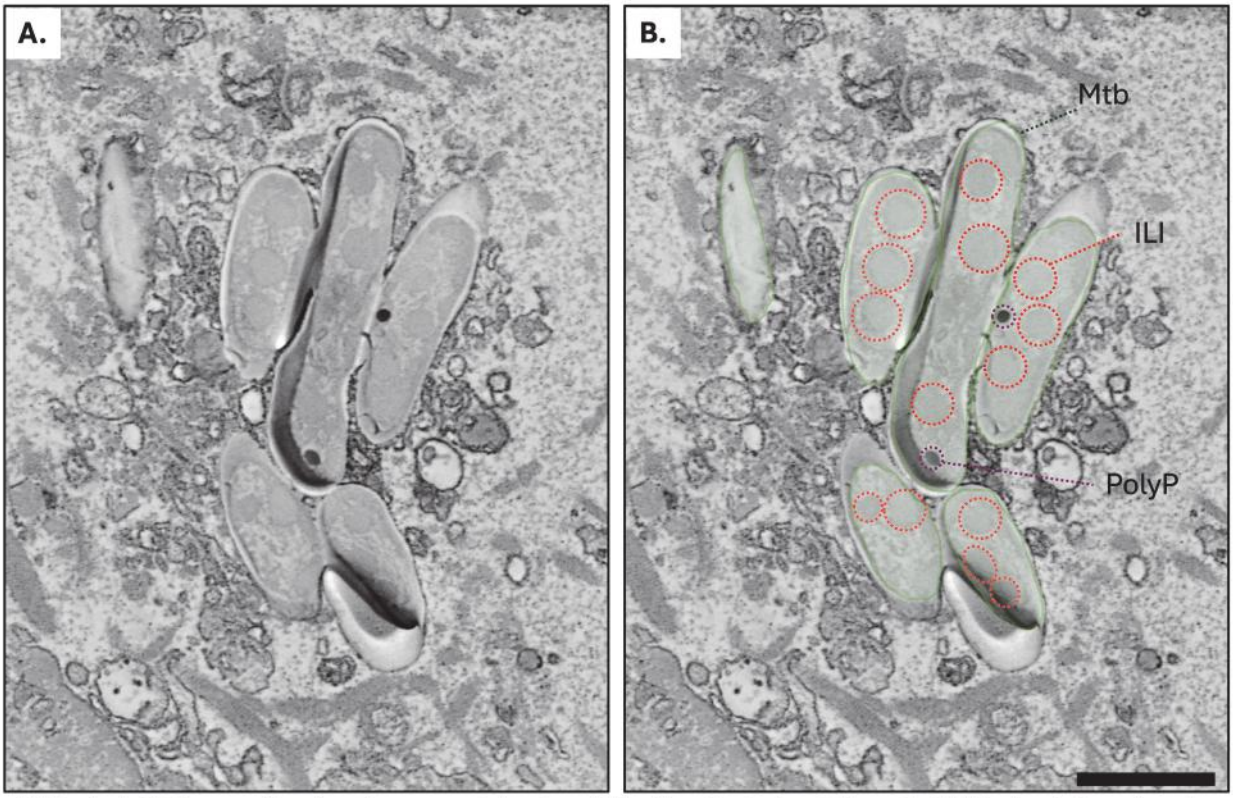
10.1136/thorax-2025-BTSabstracts.517

**Introduction** The mediastinal lymph node (LN) is a key site of *Mycobacterium tuberculosis* (Mtb)-host interaction. Though many studies have tried to localise and visualise Mtb in tissue samples from the infected human host, this has been elusive due to the challenges of obtaining and adequately preserving human tissue, precluding high resolution ultrastructural approaches such as electron microscopy.

**Methods** Following ethical approval we recruited individuals attending the Royal Free Hospital with suspected tuberculosis despite negative sputum Mtb PCR and culture, who were not on treatment and had planned diagnostic EBUS lymph node (LN) sampling. We consented participants for collection of 1–2 additional cores of mediastinal LN tissue for research analysis during the same diagnostic procedure. Research samples were placed in fixative 4%EM grade PFA with 0.1% glut in HEPES 200 mM pH 7.4 at the time of sampling and transferred to Francis Crick Institute where *en bloc* staining was performed and the sample embedded in UltraBed ultralow-viscosity resin. Serial-section scanning electron microscope array tomography and microCT were performed with volume reconstruction.

**Results** To date 11 individuals with possible tuberculosis have been recruited. 8 had mediastinal LN sampling, 5 of whom were Mtb PCR-positive and 3 PCR-negative. 3 of the 5 positive cases were AFB culture-positive and 4 demonstrated necrotising granulomatous lymphadenitis by histology. In a Mtb PCR-positive sample that has undergone extensive imaging analysis, extracellular bacteria were identified in areas of debris within the LN tissue. The bacteria themselves contain lipid droplets and granules characteristic of polyphosphates (figure 1). Analysis of this and other participants' LN samples is ongoing.

**Conclusion** We have established a clinical cohort leveraging expertise at Royal Free Hospital and Francis Crick Institute to visualise for the first time Mtb within mediastinal LN tissue.




(A.) A single SEM array slice showing suspected extracellular *Mycobacterium tuberculosis* infection in Lymph node tissue. (B.)SEM image overlayed with labels denoting bacteria (*Mtb bacilli, green),* intra-bacterial lipid inclusion (ILI, red), and inorganic polyphosphate (PolyP, Purple). Scale Bar = 500 nm

**Abstract M33 Figure 1**

This demonstrates the presence of bacteria with an energy source and a potential mechanism to survive oxidative stress in a patient with tuberculosis. This approach offers the possibility of furthering our understanding of clinically relevant aspects of Mtb-host interactions at the tissue, cellular and subcellular levels.

## M34 SEMI-AUTOMATED QUANTIFICATION OF PET-CT IN TB CONTACTS AND ITS ASSOCIATION WITH PROSPECTIVE OUTCOMES

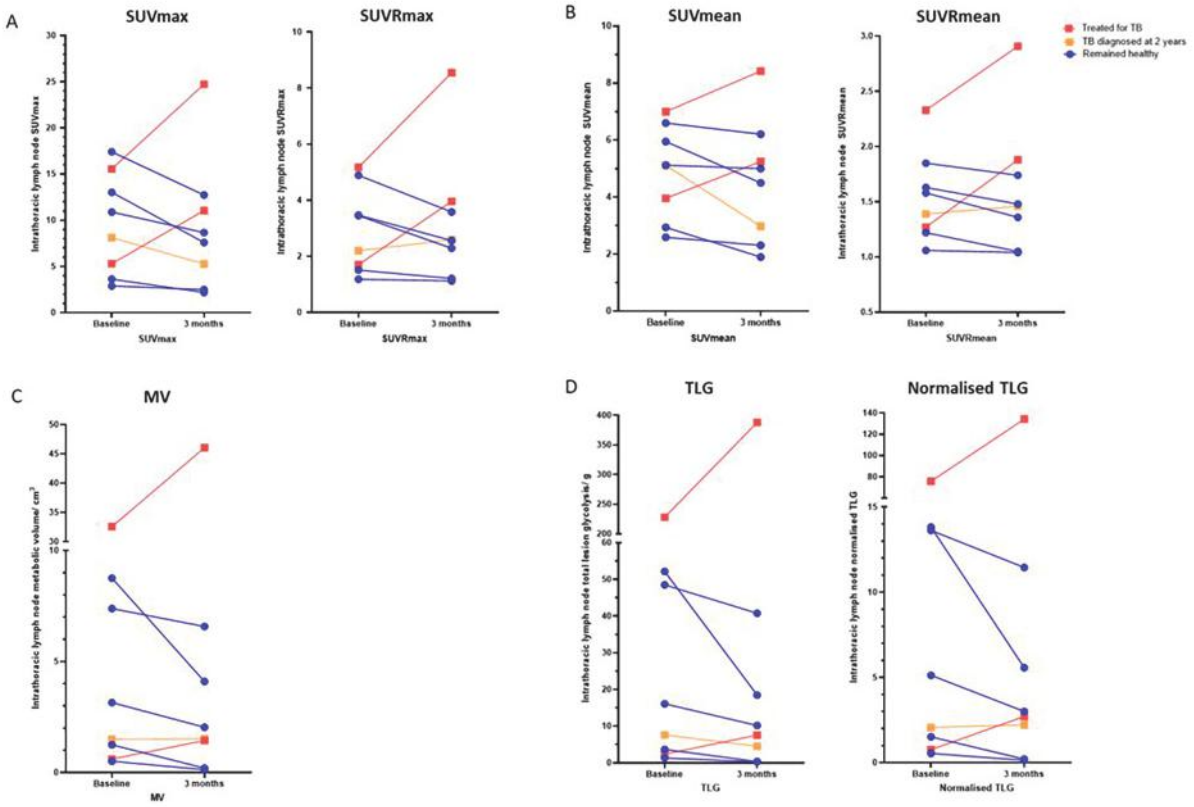
[1]JW Kim, [2]A Kamil, [2]J Lee, [2]I Novsarka, [1]P Haldar. *[1]NIHR Respiratory Biomedical Research Centre, University of Leicester, Leicester, UK; [2]University Hospitals of Leicester NHS Trust, Leicester, UK*

10.1136/thorax-2025-BTSabstracts.518

**Introduction** PET-CT has shown potential as a research tool for characterising heterogeneity in TB infection. Its prospective application requires standardisation of imaging protocols, interpretation and analysis to improve generalisability and enable data integration across discrete cohorts. We evaluated a semi-automated approach using open-source software to minimise inter-operator variability in image interpretation. We further assessed the impact of normalising PET metrics to a physiological reference organ and explored associations between metabolic change and prospective outcomes in recent pulmonary TB contacts.

**Methods** We quantified nineteen PET-CT scans with thoracic FDG uptake from eleven QuantiFERON-TB Gold-Plus (QFT)-positive TB contacts enrolled in a prospective observational study. Eight contacts underwent serial imaging at 3 months, providing data to evaluate metabolic trajectory. Imaging was analysed using a clinical platform (XD3) and 3D Slicer 5.4.0, an open-source research imaging platform that provides semi-automated segmentation and automated liver quantification. A primary reader extracted SUV-based and volumetric PET metrics using both platforms. A second reader independently extracted the data using 3D Slicer. Extracted metrics included maximum and mean standardised uptake value (SUVmax and SUVmean), metabolic volume (MV), total lesion glycolysis (TLG) in intrathoracic lymph nodes (ITLNs), and liver SUV-mean. PET metrics were normalised to liver SUVmean + 3 standard deviations (SDs).

**Results** All ITLN PET metrics from 3D Slicer showed strong agreement with the clinical platform (concordance correlation coefficient [CCC]>0.90) and moderate agreement with liver SUVmean+3SDs (CCC=0.85). Inter-reader agreement using 3D Slicer was excellent (intraclass correlation coefficient [ICC] >0.90). Three participants received TB treatment during the study period for incident TB; in two, TB was microbiologically confirmed, and in one, a new lung lesion developed on follow-up imaging that resolved with treatment. All three demonstrated increasing metabolic trajectory from baseline to 3 months when normalised to liver uptake (figure 1). In one case, liver normalisation revealed increasing metabolic activity otherwise obscured by lower physiological FDG uptake on the




SUVmax: maximum standardised uptake value. SUVRmax: SUV ratio maximum (SUVmax normalised to liver uptake), SUVmean: mean standardised uptake value, SUVRmean: SUV ratio mean, MV: metabolic volume, TLG: total lesion glycolysis

**Abstract M34 Figure 1**

second scan. Contacts remaining healthy showed decreasing PET metrics.

**Conclusions** Semi-automated PET quantification can reduce inter-operator variability. Normalising to reference organs enhances analytical sensitivity. We report preliminary findings that the metabolic trajectory in early infection is a promising indicator of prospective TB risk.

## M35 EVALUATING THE RISK-6 SIGNATURE AS A TB DIAGNOSTIC USING A NOVEL LABORATORY-BASED TUBERCULOSIS HOST IMMUNE RESPONSE ASSAY (TB-HIRA): A RETROSPECTIVE PILOT STUDY

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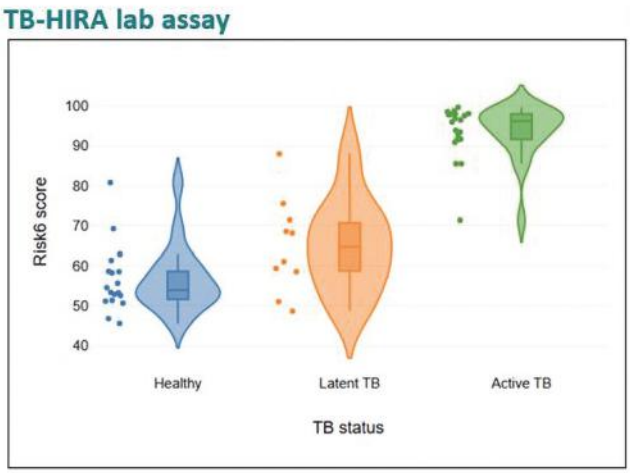
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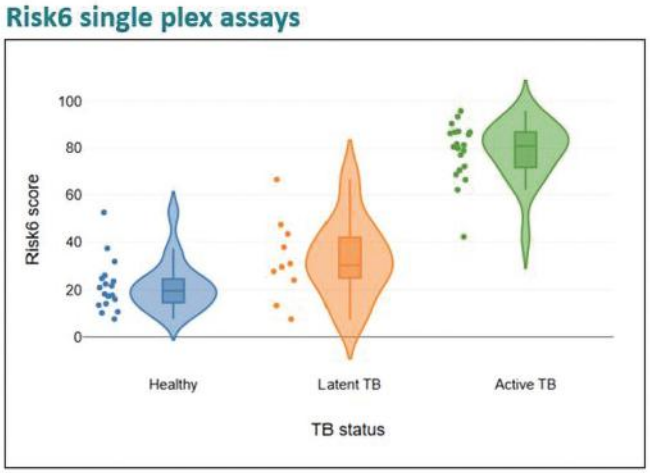
**Background** RISK6, a human-blood transcriptomic signature, has shown promise as a non-sputum-based tool for tuberculosis (TB) screening, triage and treatment monitoring among immunocompetent adults in high TB burden settings.[1] The TB-HIRA assay is a one-step multiplex qRT-PCR reaction that has been developed to streamline and accelerate the original multi-step singleplex protocol for measuring signature expression.

**Aim** To optimise the TB-HIRA qRT-PCR assay, determine concordance with the original RISK6 singleplex assays and determine performance of RISK6 scores using both methods to discriminate between active pulmonary TB (PTB), QuantiFERON-TB Gold (QFT) positive/latent TB (LTBI) and healthy (QFT negative) controls.

**Method** Venous blood samples stored in TempusTM RNA tubes from 20 individuals with active PTB, 10 with LTBI and 18 healthy controls were evaluated. Specimens were blinded and randomised before RNA extraction and throughout analysis. Both TB-HIRA and RISK6 assays were run in RT-PCR/PCR triplicates per sample. RISK6 scores were calculated in R using the risk6.R script[2] and expanded ROC analysis was performed in STATA to determine optimal cut-off thresholds.

**Results** There was excellent correlation between the original RISK6 score and the TB-HIRA assay (Spearman correlation coefficient 0.98. TB-HIRA systematically yielded higher RISK6 scores than the original singleplex assays. For both protocols, the RISK6 scores were significantly higher for active PTB compared with LTBI/healthy control groups with the optimal cut-off threshold distinguishing between them with 95% sensitivity and 96% specificity (AUC>0.99). There was no significant difference in scores between the healthy and LTBI groups (figure 1).




Dunn-Bonferroni-Tests

Copy

| | Test Statistic | Std. Error | Std. Test Statistic | p | Adj. p |
|---|---|---|---|---|---|
| Healthy - Latent TB | -6.59 | 5.52 | -1.19 | .233 | .698 |
| Healthy - Active TB | -25.84 | 4.55 | -5.68 | <.001 | <.001 |
| Latent TB - Active TB | -19.25 | 5.42 | -3.55 | <.001 | .001 |

Dunn-Bonferroni-Tests

Copy

| | Test Statistic | Std. Error | Std. Test Statistic | p | Adj. p |
|---|---|---|---|---|---|
| Healthy - Latent TB | -6.67 | 5.52 | -1.21 | .227 | .682 |
| Healthy - Active TB | -25.87 | 4.55 | -5.69 | <.001 | <.001 |
| Latent TB - Active TB | -19.2 | 5.42 | -3.54 | <.001 | .001 |

Adj. p: Values adjusted with Bonferroni correction.

**Abstract M35 Figure 1** Comparison of RISK6 score generated from the TB-HIRA vs singleplex assays

**Conclusion** In this proof-of-concept pilot study, the RISK6 signature offers robust discrimination of active PTB from LTBI and healthy subgroups, supporting potential utility as a non-sputum-based TB diagnostic test in a low TB burden setting. Strong concordance between the TB-HIRA multiplex assay and original singleplex assays, offers scope to develop a rapid near-patient TB triage test.

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## M36 PERIPHERAL BLOOD INFLAMMATORY AND IMMUNE BIOMARKERS AS CORRELATES OF TB INFECTION PHENOTYPES CHARACTERISED BY PET-CT: A PROSPECTIVE COHORT STUDY IN HOUSEHOLD CONTACTS

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10.1136/thorax-2025-BTSabstracts.520

**Introduction** TB infection (TBI) comprises a spectrum of infection states that may be characterised using FDG PET-CT by assessing the presence and burden of metabolic activity and structural change (PMID: 38914470). We evaluated whether routinely collected peripheral blood immunological and haematological inflammatory markers can inform the burden of PET-CT-defined inflammation in household contacts with TBI.

**Methods** We conducted a prospective cohort study of pulmonary TB contacts recruited between September 2021 and April 2024. Participants were recruited shortly after index case notification and underwent QuantiFERON-TB Gold Plus (QFT), full blood count, CRP, and PET-CT at baseline. QFT-positive contacts were prospectively followed for 2 years without receiving chemoprophylaxis. Monocyte-to-lymphocyte ratio (MLR), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), CRP, and quantitative TB1, TB2, and TB2-TB1 IFN-γ responses were investigated for associations with PET-CT-defined inflammation.

**Results** (see table 1).

Thirty-one of 56 QFT-positive contacts analysed exhibited FDG uptake consistent with metabolically active TBI: 26 with intra-thoracic lymph node (ITLN) involvement and 14 with lung involvement. Eight progressed to TB, including two diagnosed within 3 months of the index notification. PET lung positivity at baseline was significantly associated with TB progression ($\chi^2=19.4$, $p<0.001$).

TB1 and TB2 responses showed weak associations with the presence of ITLN positivity, but not with the extent of ITLN inflammation (SUVmax, total lesion glycolysis, data not shown). In contrast, MLR, NLR, PLR and TB2-TB1 did not associate with ITLN involvement, but did show moderate associations with the presence of PET lung positivity (AUCs: 0.81, 0.76, 0.70 and 0.69, respectively). PLR also correlated significantly with lung SUVmax ($\rho= 0.55$, $p=0.04$).

**Abstract M36 Table 1** Associations between peripheral blood markers with PET-CT-defined inflammation and TB progression

| | PET ITLN Positivity AUC (95% CI) | PET Lung Positivity AUC (95% CI) | Lung SUVmax ρ (p-value) | TB Events AUC (95% CI) |
|---|---|---|---|---|
| **Monocyte-to-lymphocyte ratio** | 0.63 (0.49 – 0.78) | **0.81 (0.65 – 0.97)** | 0.34 (0.24) | **0.75 (0.52 – 0.98)** |
| **Neutrophil-to-lymphocyte ratio** | 0.56 (0.41 – 0.71) | **0.76 (0.62 – 0.90)** | 0.46 (0.10) | 0.67 (0.46 – 0.89) |
| **Platelet-to-lymphocyte ratio** | 0.54 (0.39 – 0.70) | **0.70 (0.54 – 0.86)** | **0.55 (0.04)** | **0.78 (0.58 – 0.98)** |
| **CRP** | 0.53 (0.37 – 0.68) | 0.47 (0.23 – 0.67) | 0.38 (0.22) | 0.44 (0.21 – 0.67) |
| **QuantiFERON-TB Gold Plus** | | | | |
| **TB1** | **0.66 (0.52 – 0.80)** | 0.51 (0.35 – 0.67) | 0.17 (0.56) | 0.57 (0.36 – 0.78) |
| **TB2** | 0.64 (0.49 – 0.78) | 0.54 (0.39 – 0.70) | 0.12 (0.69) | 0.57 (0.36 – 0.78) |
| **TB2 – TB1** | 0.43 (0.27 – 0.59) | **0.69 (0.53 – 0.84)** | 0.18 (0.54) | 0.55 (0.33 – 0.78) |

Associations between peripheral blood markers with PET-CT-defined inflammation and TB progression were evaluated using the area under the receiver operating characteristic (ROC) curve (AUC). The relationship between peripheral blood makers and lung SUVmax was assessed using Spearman's rank correlation coefficient ($\rho$).
TB Antigen Tube 1 (TB1) detects CD4+ T-cell responses to TB antigens. TB Antigen Tube 2 (TB2) detects both CD4+ and CD8+ T-cell responses. TB1 and TB2 values were corrected for the Nil response.
AUC: area under the curve; ITLN: intrathoracic lymph node; PET: positron emission tomography; SUVmax: maximum standardised uptake value; TB: tuberculosis; $\rho$: Spearman's rank correlation coefficient, 95% CI: 95% confidence interval.

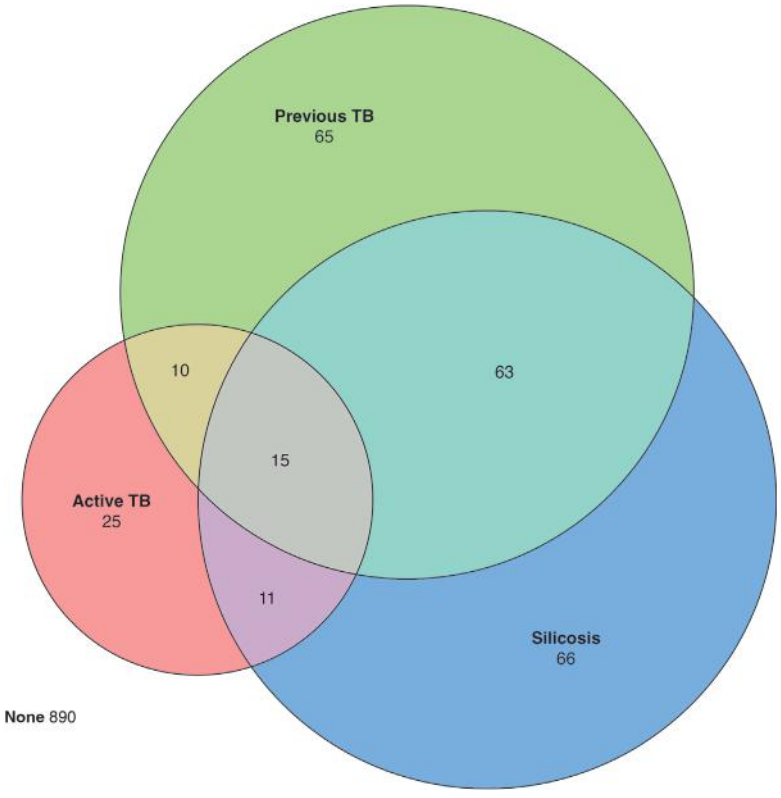
MLR, NLR, and PLR were moderately associated with TB progression (AUCs: 0.75, 0.67 and 0.78, respectively). CRP did not demonstrate associations with any evaluated outcomes.
**Conclusions** Peripheral blood inflammatory and immune markers are differentially associated with specific patterns of PET-defined inflammation in household contacts with TBI. Specifically, IFN-γ responses associate with the presence of ITLN activity, possibly indicative of a protective adaptive immune response. In contrast, haematological ratios associate with lung parenchymal inflammation that may be indicative of a failing protective immune response associated with an increased likelihood of TB progression.

## M37 THE BURDEN AND DETERMINANTS OF TUBERCULOSIS AMONG SMALL-SCALE MINERS AND COMMUNITY MEMBERS IN NORTHERN TANZANIA

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10.1136/thorax-2025-BTSabstracts.521

**Introduction** High tuberculosis (TB) disease prevalences are reported among 50 million small-scale miners globally. We describe TB risk factors and TB treatment outcomes among




**Abstract M37 Figure 1** A Euler Venn diagram of miner and ex-miner participants with silicosis, previous tuberculosis and active tuberculosis (n = 1145; complete cases)

small-scale miners, ex-miners and community members attending a screening program.

**Methods** Between 11th-24th March 2024, miners and community members completed a questionnaire, chest Xray and were offered HIV testing. All miners and ex-miners were offered sputum Xpert MTB/RIF Ultra (Xpert) tests and, in a nested cohort of 280 miners, Mycobacterium Growth Indicator Tube culture. Community members were offered Xpert if symptomatic. Follow up Xpert was performed if CXRs were abnormal. WHO tuberculosis outcomes were recorded 9-months following screening. Modelling of risk factors for TB and TB-associated mortality is ongoing.

**Results** Among 1341 participants, 59 (4.4%) had microbiological TB; these included 43/1122 (3.8%) current miners, 14/93 (15%) ex-miners, and 2/127 (1.6%) community members. Silicosis prevalence in current and ex-miners was 11% and 36%, respectively. No rifampicin resistant cases were identified. Microbiological TB prevalence in mines with >75% attendance was 15/277 (5.4%). Median work duration was higher among those with TB (13 years, IQR 8.5, 20 years) compared to those without TB (7.5 years, IQR 3, 15 years) and HIV was more common (10% vs 4%). Silicosis, TB and self-reported previous TB diagnoses clustered together (figure 1). At 9 months following treatment, 10/66 (15%) of participants diagnosed with TB had died. Of these, three refused to start treatment and subsequently died, five died within 6 weeks of starting treatment and two over 6 months after starting treatment. Two participants, both of whom died over 6 months after starting treatment, were HIV positive. Those who died on treatment were more likely to report weight loss at presentation, compared to those who did not die (86% vs 21%) but did not appear more likely to have silicosis (29% vs 44%).

**Conclusion** As expected from our previous systematic review, TB prevalence was very high and associated with work exposures and silicosis. More concerningly, and despite a largely active workforce sample with relatively low HIV prevalence, mortality was high. Limitations include possible selection bias and underdiagnosis of clinical TB.

## M38 EFFECT OF CHEST RADIOGRAPH REPORTING CODES ON TIME TO DIAGNOSIS OF SUSPECTED PULMONARY TUBERCULOSIS (PTB) – A RETROSPECTIVE COHORT STUDY

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**Background** Almost 1/3 of incident pulmonary TB (PTB) cases in England start treatment >4 months after symptom onset.[1] Leicester, a high incidence TB area (>40 cases/100,000 population) provides a centralised rapid access tuberculosis (RATB) pathway for suspected TB cases from primary and secondary care, facilitating urgent assessment. To expedite diagnosis, radiologists can assign urgent referral codes, such as the TB code, to streamline referrals to appropriate multidisciplinary teams.

**Aim** We investigated the accuracy of radiology reports for diagnosing PTB and the impact of reporting codes and keywords on time to PTB notification in patients referred to the RATB service.

**Methods** We assessed CXR reports of 1124 patients referred to the RATB service at University Hospitals of Leicester (Jan 2019–Dec 2023) for inclusion of TB or other urgent referral codes - lung cancer code (RAL), GPCTFU (GP CT follow-up), XRCCT (urgent CT follow-up) and TB keywords (TB*, tubercul*, mycobac*), and the intervals between CXR request, report and TB diagnosis.

**Results** There were 216 cases (19.2%) of PTB among the 1124 patients referred to RATB service. CXRs were performed promptly (median same-day), but median reporting times varied by requesting source - 2 days (A&E), 2 days (primary care), 6 days (inpatient), 11 days (outpatient) ($p<0.001$). Urgent codes or TB keywords were reported in 138 (63.9%) of the PTB cases. Interestingly, 22 PTB cases (10.2%) had reports containing TB keywords but no referral code assigned, and 41 cases (21.2%) had other abnormalities reported but no link to TB diagnosis was made. The diagnostic performance of different codes and TB keywords is noted in table 1. Inclusion of the RATB code or inclusion of TB keywords was associated with a significantly shorter median notification delay than non-coded cases (9 vs. 46.5 days ($p<0.001$)).

**Conclusion** Accurate reporting and inclusion of a TB code on CXR reports is a key driver of accelerated PTB diagnosis for RATB pathways. However, the code was applied to only a subset of cases, suggesting TB-related CXR changes lack specificity. These data provide a useful reference for evaluating AI-driven approaches to CXR reporting and coding for TB.

### REFERENCE

1. https://www.gov.uk/government/publications/tuberculosis-in-england-2023-report-data-up-to-end-of-2022/tb-treatment-and-outcomes-england-2022

**Abstract M38 Table 1** Effect of CXR coding and reporting of TB keywords on time to TB notification

| | CXR CODING CATEGORIES IN PATIENT REFERRALS TO RATB | | | | |
|---|---|---|---|---|---|
| *Variable* | *RATB coded* | *RAL, GPCTFU or XRCCT urgent codes applied (RATB excluded)* | *Non-urgent X-ray follow-up code (RATB and urgent codes excluded)* | *Non-coded scans including TB keywords* | *Non-coded scans with no TB keywords* |
| **Number of CXRs in category (% total CXRs)** | 308 (27.4) | 103 (9.2) | 19 (1.7) | 79 (7.0) | 615 (54.7) |
| **Number of PTB cases in the category (% of given category)** | 93 (30.2) | 23 (22.3) | 4 (21.1) | 22 (27.8) | 74 (12.0) |
| **Sensitivity (%) [95% CI]** | 43.1 [36.4–49.9] | 10.6 [6.9–15.5] | 1.9 [0.5–4.7] | 10.2 [6.5–15.0] | 34.3 [28.0–41.0] |
| **Specificity (%) [95% CI]** | 76.3 [73.4–79.1] | 91.2 [89.2–93.0] | 98.3 [97.3–99.1] | 93.7 [91.9–95.2] | 40.4 [37.2–43.7] |
| **Median days from CXR report to PTB notification (IQR)** | 9 (6–15) | 29 (11–45) | 51.5 (29–63) | 10 (5.5 – 24.5) | 46.5 (15–78) |

## M39 A PIECE OF THE DIAGNOSTIC PUZZLE: COULD HISTOLOGICAL RESULTS BE USED TO TRIGGER A SPECIALIST TB REVIEW?

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**Background** In 2024 5,480 people were treated for TB disease in England.[1] Early diagnosis and treatment reduces disease-associated morbidity and mortality. A typical histological pattern of necrotising granulomatous inflammation is key diagnostically, due to long delays in positive culture results. We reviewed all biopsies coded with this finding to determine whether active appraisal of these patient cases could reduce diagnostic delay of TB.

**Methods** We undertook a retrospective cohort analysis of all biopsy reports of 'necrotising granulomatous inflammation' from April 2023–2024 at two TB services in an urban area. Data were collected from the electronic patient health record and pathology database and cross-referenced with the national TB database to verify patients diagnosed with active TB.

**Results** 64 cases were found; 18 had reports where the search term was mentioned as a relevant negative finding in the histology report. Of the remaining 46 cases, 4 had a known cause for the necrotising granulomatous inflammation and they were excluded, resulting in a total of the 42 cases that were studied in further detail.

Patient characteristics are referenced in table 1.

In the TB patient cohort, median time from biopsy result to treatment initiation was 2 days (IQR 0 – 47 days). 9 patients received treatment within 7 days of biopsy result; of the remaining 5 patients, median time from result to treatment initiation was 91 days (IQR 27–109 days).

**Discussion** We suggest that active review of histology samples reported as 'necrotising granuloma' may reduce diagnostic delay. A significant proportion of patient biopsies with this finding were subsequently diagnosed with TB, and the overall numbers do not suggest that a streamlined pathway prompting review of the patient notes within the TB service would be a strain on resources. Prolonged time to diagnosis can have consequences for the individual, the health service and the public; a fast-track system directing pathology reports with key phrases to the TB service could translate into measurable benefit, and help avoid detrimental delay.

**Abstract M39 Table 1**

| | Whole cohort (n=42) | TB diagnosis (n=14) | Not TB (n=28) |
|---|---|---|---|
| Age [years] median (IQR) | 57.4 (44.7 – 70.0) | 44.6 (35.0–59.0) | 57.6 (52.6–74.6) |
| **Sex** | | | |
| Male | 18 (43%) | 6 (43%) | 12 (43%) |
| Female | 24 (57%) | 8 (57%) | 16 (57%) |
| **Biopsy site** | | | |
| Pulmonary (%) | 10 (23%) | 4 (28%) | 6 (21%) |
| Lymph node (%) | 12 (29%) | 5 (36%) | 5 (18%) |
| Soft tissue (%) | 20 (48%) | 5 (36%) | 17 (61%) |
| **Acid fast bacilli** | | | |
| **Positive** | 6 (14%) | 6 (43%) | 0 |
| **Negative** | 25 (60%) | 8 (57%) | 16 (57%) |
| **Non-diagnostic** | 11 (26%) | | 12 (43%) |
| **Necrosis present on sample** | | | |
| **Positive** | 25 (60%) | 14 (100%) | 11 (39%) |
| **Negative** | 17 (40%) | | 17 (61%) |

### REFERENCE

1. National quarterly report of tuberculosis in England: quarter 4, 2024, provisional data - GOV.UK

## M40 WHEN DOES TB PCR CHANGE MANAGEMENT? – A RETROSPECTIVE CASE SERIES EXAMINING ROLE OF PCR IN INITIAL AND FINAL TB DIAGNOSIS IN A GENERAL HOSPITAL

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Early diagnosis of tuberculosis brings clinical and public health benefits. The NICE guidance for TB (not substantially updated since 2016) states that for HIV-negative individuals TB PCR is an additional test available on the primary specimen 'if it would alter management'.[1] At a time of general cost saving on pathology tests it has become necessary to justify the use of TB PCR. We looked at 127 consecutive cases treated for TB in our hospital over 30 months from Jan 2022 to August 2024 to determine how many cases were diagnosed on the basis of PCR (table 1), lead time advantage over WGS, subsequent tests avoided, and early diagnosis of rifampicin resistant disease.

Among 36 pulmonary/miliary cases, 8 were sputum smear negative/PCR positive, and 2 were BAL smear negative, PCR positive. We estimated that the availability of early diagnosis through PCR avoided 3 thoracoscopies, 2 bronchoscopies, 7 repeat biopsies and 1 repeat EBUS. The average delay between positive PCR and receiving final WGS result was 67.2 days for pulmonary cases and 44.8 days for non pulmonary cases. 4 cases of rifampicin resistant disease were diagnosed on PCR.

**Conclusion** In 127 consecutive cases (January 2022 to August 2024) PCR provided the initial diagnosis in 46.4% of cases and the final diagnosis in 15.7%. It provided the initial diagnosis in 91% pulmonary cases (excluding resected pulmonary nodules). PCR is particularly helpful in sputum, BAL, pus and fluid samples where there is not an option

**Abstract M40 Table 1** Initial (final) basis of diagnosis for consecutive TB cases Jan 2022 - Aug 2024

| | PCR | Culture | Histology | Clinical | Total |
|---|---|---|---|---|---|
| extrathoracic LN | 14 (8) | 5 (14) | 17(14) | 0 | 36 |
| pulmonary | 31 (6) | 0 (25) | 3 (3) | 0 | 34 |
| bone/spinal | 4 (2) | 3 (7) | 4 (2) | 0 | 11 |
| pleural | 3 (3) | 1 (1) | 2 (2) | 4 (4) | 10 |
| intrathoracic LN | 3 (1) | 0 (3) | 5 (4) | 0 | 8 |
| ocular | 0 | 0 | 0 | 8 (8) | 8 |
| abdominal | 2 (0) | 0 (3) | 5 (4) | 0 | 7 |
| miliary | 2 (0) | 0 (2) | 0 | 0 | 2 |
| other | 0 | 0 | 7 (7) | 4 (4) | 11 |
| Total | 59 (20) | 9 (55) | 43 (36) | 16 (16) | 127 |

for histological diagnosis; and in early diagnosis of rifampicin resistance.

## REFERENCE

1. NICE Guideline Tuberculosis NG33 Published 13th January 2016.

## M41 HIGH FREQUENCY OF ASYMPTOMATIC MEDIASTINAL LYMPH NODE TB DIAGNOSED VIA EBUS: A RETROSPECTIVE COHORT STUDY FROM A UK TB-NETWORK

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**Introduction** In 2023 extrapulmonary tuberculosis accounted for 62% of TB diagnoses in England, with mediastinal lymph node tuberculosis (MLN-TB) responsible for 14%.[1] MLN-TB symptoms vary, and it may be incidentally detected on chest imaging. Mediastinal lymphadenopathy is now routinely evaluated using endobronchial ultrasound-guided needle aspiration (EBUS-TBNA).

We investigated MLN-TB diagnosed with EBUS-TBNA to estimate the frequency of asymptomatic presentations in our TB population.

**Methods**

- We conducted a retrospective study of EBUS-diagnosed MLN-TB in North Central London (NCL) TB Network between 2018–2024.
- Cases were classified as asymptomatic (no TB-suggestive symptoms, MLN incidentally noticed on imaging) or symptomatic (if typical TB symptoms, or TB-related symptoms led to imaging).
- A TB diagnostic score was recorded based on the level of evidence contributing to diagnosis, as previously.[2]

**Abstract M41 Table 1** Demographic, clinical, and microbiological characteristics of patients with asymptomatic and symptomatic MLN-TB from NCL TB network

| | Asymptomatic MLN-TB (N=49) | Symptomatic MLN-TB (N=86) | Total (N=135) | p-value |
|---|---|---|---|---|
| **Clinical presentation** | | | | |
| Isolated mediastinal TB | 27 (55%) | 29 (34%) | 56 (42%) | **0.01** |
| MLN-TB + disease elsewhere | 20 (41%) | 55 (64%) | 75 (56%) | |
| No data | 2 (4%) | 2 (2%) | 4 (3%) | |
| Sex (M) | 31 (63%) | 48 (56%) | 79 (59%) | 0.4 |
| Age (mean [SD]) | 51.2 [20%] | 40.0 [17.98] | 44.1 [19.60] | **<0.05** |
| **Ethnicity** | | | | |
| *Asian (All subgroups)* | 24 (49%) | 34 (40%) | 58 (43%) | 0.08 |
| *Black (African / Caribbean / Black British)* | 10 (20%) | 34 (40%) | 44 (33%) | |
| *White* | 6 (12%) | 12 (14%) | 18 (13%) | |
| *Arab* | 3 (6%) | 1 (1%) | 4 (3%) | |
| *Mixed/Multiple ethnic groups/Other* | 4 (8%) | 2 (2%) | 6 (5%) | |
| No data | 2 (4%) | 3 (4%) | 5 (4%) | |
| **Referral route EBUS** | | | | |
| Inpatient referral | 3 (6%) | 23 (27%) | 26 (19%) | **<0.05** |
| Two week wait referral | 6 (12%) | 17 (20%) | 23 (17%) | |
| Respiratory/ID clinic | 13 (27%) | 8 (9%) | 21 (16%) | |
| TB clinic | 8 (16%) | 6 (7%) | 14 (10%) | |
| Hospital specialist referral | 10 (20%) | 4 (5%) | 14 (10%) | |
| ED referral | 2 (4%) | 3 (4%) | 5 (4%) | |
| Community clinic (GP/migrant) | 2 (4%) | 2 (2%) | 4 (3%) | |
| No data | 5 (10%) | 23 (27%) | 28 (21%) | |
| **Comorbidities** | | | | |
| Diabetes | 8 (16%) | 11 (13%) | 19 (14%) | 0.61 |
| CKD | 5 (10%) | 9 (11%) | 14 (10%) | 0.96 |
| Chronic lung disease | 11 (24%) | 2 (3%) | 13 (12%) | **<0.05** |
| Malignancy | 8 (16%) | 2 (2%) | 10 (7%) | **<0.05** |
| *Lung tumour* | 3 (7%) | 0 (0%) | 3 (2%) | |
| HIV | 0 (0%) | 3 (4%) | 3 (2%) | 0.27 |
| Sarcoidosis | 1 (2%) | 0 (0%) | 1 (1%) | 0.41 |
| **Microbiology/histology - EBUS** | | | | |
| TB PCR +ve | 27 (55%) | 52 (61%) | 79 (59%) | 0.67 |
| No data | 5 (10%) | 7 (8%) | 12 (9%) | |
| Histology granuloma +ve | 39 (80%) | 61 (71%) | 100 (74%) | 0.13 |
| No data | 4 (8%) | 5 (6%) | 9 (7%) | |
| Culture +ve | 31 (63%) | 53 (62%) | 84 (62%) | 0.86 |
| No data | 1 (2%) | 2 (2%) | 3 (2%) | |
| **TB microbiologically confirmed (PCR/culture)** | 42 (86%) | 71 (83%) | 113 (84%) | 0.81 |
| TB non microbiologically confirmed | 7 (14%) | 16 (19%) | 23 (16%) | |
| Histology positive | 5 (10%) | 10 (9%) | 15 (11%) | |
| Histology negative | 2 (4%) | 6 (7%) | 8 (6%) | |
| *TB-positive sample from other site* | 1 (2%) | 5 (6%) | 6 (4%) | |
| *All samples TB-ve, but TB still likely* | 1 (2%) | 1 (1%) | 2 (2%) | |

**Results**

- 135 cases were included in the study (table 1): 61 from the Royal Free (RFH), 50 from University College of London (UCLH), and 24 from North Middlesex (NMH) hospital.
- 49/135 (36%) were identified as asymptomatic MLN-TB, with the remainder symptomatic.
- Asymptomatic MLN-TB patients were significantly older (mean age 51.2 vs 40.0 years, p=0.002) and more likely to have a history of chronic lung disease (CLD) or cancer than symptomatic individuals.
- Asymptomatic MLN-TB was more frequently limited to MLN involvement, compared to symptomatic TB (55.1% vs 33.7%, p=0.01).
- Microbiological confirmation (molecular test or culture) was obtained in similar proportions of asymptomatic and symptomatic individuals (85.7% vs 82.6%)
- MLN-TB symptoms varied significantly by referral route (p=0.001): inpatient referrals were mostly symptomatic, while two-week wait and other outpatient services captured more asymptomatic cases.

**Conclusions** Approximately 36% of our EBUS-diagnosed MLN-TB occurred in asymptomatic individuals having imaging for unrelated indications. Asymptomatic patients were older and more often had prior lung disease or malignancy.

Referral route seems to differ according to the clinical presentation of MLN-TB. The growing use of thoracic CT imaging—especially through non-TB-specific pathways—may facilitate incidental detection; mediastinal lymphadenopathy on imaging should prompt consideration of TB and early testing, even in the absence of traditional symptoms and risk factors.

## REFERENCES

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## M42 A MULTI-CENTRE AUDIT OF THE DIAGNOSIS AND MANAGEMENT OF LYMPH NODE TUBERCULOSIS

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**Introduction and Objectives** Lymph node tuberculosis represents a common manifestation of tuberculosis (TB). Diagnosis can be challenging, particularly if the condition is not suspected.

Multiple complications are associated with TB treatment, including development of paradoxical reactions. Corticosteroids are not routinely recommended for treatment of lymph node TB, however may be used in clinical practice. We aimed to conduct a multi-centre audit to review diagnostic procedures and treatment complications during management of lymph node TB, referring to NICE guidance.[1]

**Methods** Three UK NHS Trusts identified patients diagnosed with confirmed (culture-confirmed, acid-fast bacilli or molecular diagnostic positive, or histological evidence) or presumed lymph node tuberculosis between 1st January 2019 to 31st December 2023. A retrospective case note review was performed.

**Results** Data was available on 406 individuals who were diagnosed with lymph node tuberculosis. Results are summarised by site in table 1. 174 (43%) were female. 269 (66%) were microbiologically confirmed. In 213 (52%), presentation was lymph node involvement only, while in 191 (47%), presentation was lymph node TB with other sites involved.

**Abstract M42 Table 1**

| | Trust 1 | Trust 2 | Trust 3 | Total |
|---|---|---|---|---|
| Number of patients identified, n | 61* | 144 | 201 | 406 |
| Age, median years (IQR) | 33 (27 – 47) | 37 (28- 49) | 43 (33 - 55) | |
| Female, n (%) | 18 (30) | 56 (39) | 100 (50) | 174 |
| LN TB only, n (%) | 28 (46) | 96 (67) | 89 (44) | 213 (52) |
| LN with other site involvement, n (%) | 31 (51)** | 48 (33) | 112 (56) | 191 (47) |
| Number of procedures, median (IQR) | 2 (1–2) | 1 (1 – 1) | 1 (1–1) | 1 |
| Microbiologically confirmed, n (%) | *Hospital: 19 (90)<br>*Lab: 40 (100) | 79 (55) | 131 (65) | 269 (66) |
| Diagnosed/Treated as Paradoxical reaction, n (%) | 8 (13) | 11 (8) | 2 (1) | 21 (5) |
| Corticosteroids during treatment, n (%) | 12 (20) | 4 (3) | 9 (4) | 25 (5) |
| Treatment outcome = completed treatment, n (%) | 55 (90) | 139 (97) | 197 (98) | 391 (96) |
| Treatment outcome = care transferred or lost to follow-up, n (%) | 4 (7) | 1 (0.7) | 1 (0.5) | 6 (1) |
| Treatment outcome = treatment failure or died, n (%) | 0 (0) | 4 (2.7) | 0 (0) | 4 (1) |

*In Trust 1, records were identified by hospital admission coded as lymph node tuberculosis or if microbiology laboratory records identified a positive Mtb culture from a non-sputum sample. Lymph node TB managed entirely in the outpatient setting may not have been included.
**Included if significant lymph node disease

The median number of procedures for diagnosis was 1, (min=0, max =7). 269 (66%) cases were microbiologically confirmed. Overall, in 21 (5%) cases a paradoxical reaction on anti-tuberculous therapy (ATT) was diagnosed. However, this differed by site (Site 1 = 13%, Site 2 = 8%, Site 3 = 1%, p≤0.01), whilst 25 (6%) patients were managed with corticosteroids during ATT. There were high rates of treatment success.

**Conclusions** Lymph node TB is responsible for considerable morbidity. Specific standards for diagnosis and management of lymph node TB and guidance for use of corticosteroids in this condition are lacking.[1] However, our data suggests achieving microbiological diagnosis is challenging. Patients usually have at least one procedure, but may undergo multiple diagnostic procedures. In a third of our cases, diagnosis was not microbiologically confirmed. Paradoxical reactions occur during treatment, although due to lack of specific diagnostic tests or consensus definitions, these may be difficult to recognise. Diagnosis of paradoxical reaction and treatment with corticosteroids was relatively uncommon but may vary by setting.

## REFERENCE

1. Tuberculosis (2016) NICE guideline NG33. Last updated 16/02/2024.

## M43 HOSPITALISATION FOR TUBERCULOSIS: A RETROSPECTIVE EVALUATION OF BURDEN AND LENGTH OF STAY FOR A UK TB HOTSPOT

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**Introduction** Despite availability of rapid TB diagnostics and referral pathways, hospitalisation rates for TB remain high across Europe, contributing significantly to TB treatment costs.[1] While several European countries have published data on TB hospitalisation costs,[2] such data is lacking for the UK.

**Objectives** To quantify the proportion of TB cases hospitalised, assess average length of stay (ALOS), identify factors influencing hospitalisation and ALOS, and estimate the proportion of potentially avoidable admissions in Leicester.

**Method** This was a retrospective cohort study of all TB patients admitted between Jan-Dec 2023. The World Health Organization (WHO) criteria for hospitalisation guided selection of corresponding locally-recorded factors (table 1). Turnaround times (TAT) along the care cascade (time to sampling, results, treatment initiation and discharge) were extracted from patient records.

**Result** Table 1 Of 199 TB cases notified, 67 (33.7%) were hospitalised contributing 74 discrete hospitalisation episodes - 15 (22%) patients had pulmonary TB, 11 (16%) extrapulmonary TB, and 41 (61%) multi-compartmental disease. Four patients (5.9%) died.

Key hospitalisation factors identified were disease severity (31%), co-morbidities (36%), and malnutrition (34%). Thirteen patients (19%) had 1 factor for hospitalisation, 40 patients (60%) had >1 factors, while 14 (21%) had no identifiable factors. The ALOS was 33 days (median 21, IQR 11–37), comprising a median TAT from admission to diagnostic result of 9 days, and 13 days from starting treatment to discharge. For diagnosis, Xpert-Ultra MTB-RIF was used in 61 patients (91%) and 54 (81%) tested positive. Median length of stay was unaffected by the number of hospitalisation factors (19–21 days across groups with 0, 1 and >1 factor identified), suggesting predominance of operational factors.

**Conclusion** In 2023, one-third of TB patients in Leicester were hospitalised, with ALOS exceeding 30 days - longer than in comparable European countries like Sweden or the

**Abstract M43 Table 1** WHO* and local TB hospitalization criteria

| Main Criteria | Corresponding variables collected at Leicester | Number of patients meeting criterion |
|---|---|---|
| **Severe TB-** Meningitis, vertebral bone TB, Pericarditis, Miliary TB, Severe lung disease( Respiratory distress/failure, sepsis | Disease type (no change)<br>NEWS | Severe TB-21(31%)<br>NEWS->/=7-10(15%) |
| **Complications of TB-**requiring surgical interventions, such as haemorrhage, pneumothorax and pleural effusion | Procedure done (Y/N) | Procedure done-15(22%) |
| **Serious comorbidities with TB-**Severe Malnutrition, untreated HIV, liver disease, renal disease and Uncontrolled Diabetes | Diabetes<br>Cancer<br>HIV (new / high viral load)<br>CKD eGFR<30 / AKI 1<br>MUST score >/=2<br>Clinical Frailty Score >/=5 | Rest of co-morbidities/ coinfection-24(36%)<br>MUST Score-23(34%)<br>CSF Score-7(10%) |
| **Extreme of age-**Very young or old <5 years and >65 | No change | 10(15%) |
| **Life-threatening and serious medical events resulting from adverse reactions to anti-TB drugs** (*e.g.* life-threatening arrhythmias, psychosis, renal failure and hearing loss | Interruption to medication due to adverse effects | 10(15%) |
| **Additional Criteria** | | |
| 1)Patients for whom effective and safe anti-TB treatment cannot be ensured in an outpatient, community or home setting (*i.e.* **homelessness, overcrowding,** exposure of children aged <5 years and pregnant women in the household)<br>2)When there are accessibility problems (*i.e.* patient lives far from an outpatient facility<br>3)Where there **is nonadherence to treatment** (this can be considered in some settings as a last resort once all other care options have been used/applied exhaustively, based on the legal framework in force) | Social Risk Factors | 11(16%) |

*Source: WHO Operational Handbook on TB 2022; Consensus Document of WHO Regional Office Europe

Netherlands (ALOS <20days) with similar hospitalisation rates.[2] One-fifth of admissions were potentially avoidable as no factor for admission could be identified, while in-patient TATs were prolonged despite use of molecular diagnostics. We conclude TB-healthcare costs may be significantly reduced by developing strategies to prevent unnecessary admission and streamlining inpatient pathways.


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## M44 ABSTRACT WITHDRAWN

## M45 PERSISTENTLY POSITIVE SPUTUM SMEARS WITH SUB-THERAPEUTIC RIFAMPICIN LEVELS IN PULMONARY TUBERCULOSIS – A CALL FOR CHANGE?

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10.1136/thorax-2025-BTSabstracts.528

**Background** Rifampicin plays a vital bactericidal role in the treatment of Tuberculosis (TB). Achieving the WHO recommended dose of 10 mg/kg can be limited by fixed-dose preparations, which carry the risk of underdosing. Several studies have revealed low rifampicin concentrations at the site of pulmonary infection when 10 mg/kg dosing is used (van Ingen et al., 2011). Although low serum rifampicin levels are associated with treatment failure, relapse, and acquired drug resistance, rifampicin levels are not monitored routinely during TB treatment (Holden et al., 2022).

**Objectives**

- To explore if patients who are initially smear positive with low rifampicin (<8 mg/L) are as likely to remain smear positive after 2 weeks of treatment, as those with therapeutic rifampicin levels (>8 mg/L)
- The final dose (mg/kg) of rifampicin required to achieve therapeutic rifampicin levels (8 mg/L or more).

**Method** We conducted a retrospective review of all adult patients who initiated treatment for Pulmonary Tuberculosis between October 2021 and March 2025, identifying 118 patients. 20 patients who were not able to complete treatment were excluded. Patients' weights, smear results, initial and final rifampicin doses, and dose adjustments were collected from patient records. Rifampicin levels (2 hours post-dose) were checked two weeks after treatment initiation and after any dose adjustment. Levels below 8 mg/L were considered subtherapeutic.

**Results** 47 (out of 98) patients were smear-positive at treatment initiation, and 14 patients stayed smear-positive at two weeks of treatment. Of note, 12 patients who were smear positive at 2 weeks, had subtherapeutic (<8 mg/L) rifampicin levels. The mean rifampicin dose (mg/kg) required to achieve therapeutic levels of rifampicin in this cohort was 14.47 mg/kg.

About one-third (27%) of patients with subtherapeutic rifampicin levels were on a dosage of 450 mg or below.

Rifampicin levels 4.0–8.0 mg/L were accepted in two patients due to age and patient concerns.

Interestingly, monitoring rifampicin levels identified two patients who were non-compliant with treatment and directly-observed therapy brought rifampicin levels in range.

**Conclusion** While fixed-dose preparations aid in treatment compliance, the WHO-recommended dose is limited by the fixed-dose preparations. 86% of patients who stayed smear-positive at two weeks after treatment initiation had subtherapeutic (<8 mg/L) rifampicin levels.


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2. Holden A, Dhadda S, Obisi N, French K, Thickett K, Carter R. P149 Routine monitoring of rifampicin levels reveals subtherapeutic levels in 76% of patients with fully sensitive TB. *'Die Hard II' – Antibiotic Resistance and Challenges in TB* [online] 2022;A162.2–A163. doi:HYPERLINK "https://gbr01.safelinks.protection.outlook.com/?url=https%3A%2F%2Fdoi.org%2F10.1136%2Fthorax-2022-btsab-stracts.284&data=05%7C02%7CAbstracts%40brit-thoracic.org.uk%7C4118d6a78e744d122afe08ddf048cf2f%7Cdd0ce28c0d1a4210a53096d-ba109cdba%7C0%7C0%7C638930917711050999%7CUnknown%7CTWFpbGZsb3d8eyJFbXB0eU1hcGkiOnRydWUsIlYiOiIwLjAuMDAwMCIsIlAiOiJ-XaW4zMiIsIkFOIjoiTWFpbCIsIldUIjoyfQ%3D%3D%7C0%7C%7C%7C&sdata=SG-GIY3UEpCv3YHrlPUNttWZxAQRhAAudyRhXMO5huUw%3D&reserved=0"https://doi.org/10.1136/thorax-2022-btsabstracts.284


## M46 A META-ANALYSIS OF TB TREATMENT OUTCOMES IN INDIVIDUALS WITH SILICOSIS AND SILICA EXPOSURE

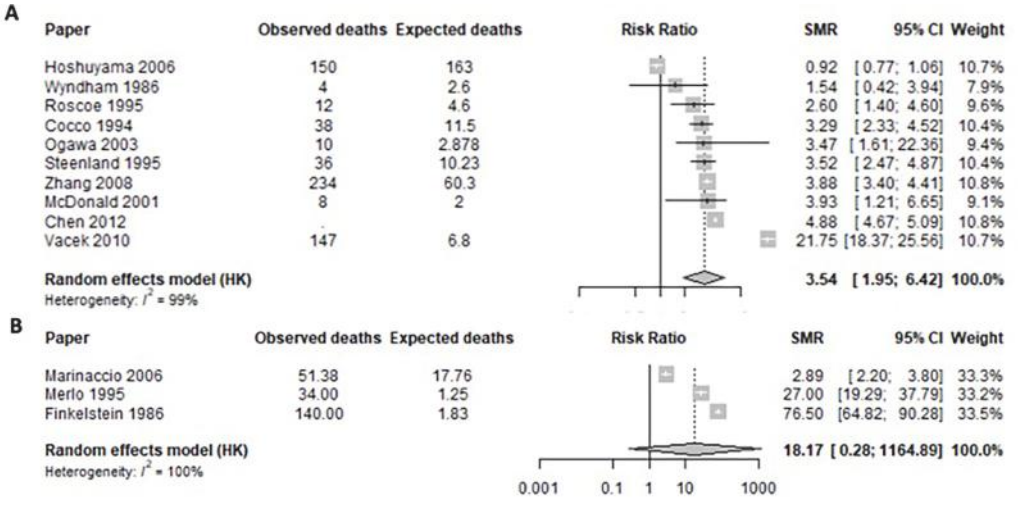
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**Background** Silicosis is caused by the inhalation of respirable crystalline silica. Silica-exposed individuals have a two-fold, and those with silicosis a four-fold, increased risk of tuberculosis (TB).[1] To determine if these groups also experience worse TB outcomes, we reviewed studies that described TB-related mortality and TB treatment outcomes among individuals with silica exposure or silicosis.

**Methods** Medline, Embase, Cinahl, Web of Science and Scopus were searched on 6 March 2025. Studies including TB treatment success, failure, relapse or mortality outcomes from adult populations described as silica-exposed or with a radiologically confirmed diagnosis of silicosis were included. We pooled standardised mortality ratios (SMR) using a random effects meta-analysis. Methodological and outcome differences precluded meta-analysis for TB treatment outcomes.

**Results** Of 26 eligible studies, 13 contributed SMR data from 264,617 workers. The pooled SMR for silicosis-exposed studies was 3.54 (95% confidence interval (CI) 1.95, 6.42, n = 10) (figure 1). Outlier analysis demonstrated that very high heterogeneity ($I^2$ = 99%) was largely attributable to two studies. SMRs for three silicosis studies were 76.5 (95% CI 63.6, 89.1), 27.0 (95% CI 18.8, 38.8) and 2.9 (95% 2.2, 3.8). TB treatment success rates ranged from 23% to 99% in 11 silica-




**Forest plots with pooled SMR values displayed for (A) Silica-exposed, undefined silicosis status and (B) Silicosis group. (A) Pooled SMR of 3.54 [1.95; 6.42] (n = 10), heterogeneity displayed using $I^2$ = 99% (p < 0.01). (B) Pooled SMR of 18.17 [0.28; 1164.89] (n = 3), heterogeneity displayed using $I^2$ = 100% (p < 0.01).**

**Abstract M46 Figure 1** Forest plots displaying pooled SMR value for silica-exposed and silicosis group

exposed groups, and 3% to 99% in 7 silicosis groups; 13/18 (72%) study groups reported treatment success rates below the 90% World Health Organisation benchmark. There was a greater relative risk of unfavourable outcomes in four of six studies that directly compared higher to lower exposures or silica-exposed to non-silica exposed groups.

**Discussion** Despite study heterogeneity, TB-related mortality appears more common among silica-exposed individuals than the general population, with a higher risk among those with silicosis. This excess TB mortality, greater than expected from increased incidence alone, and poor treatment success suggest silica exposure and silicosis lead to worse TB outcomes. Reducing silica exposures would lead to lower TB incidence and, to a greater extent, improve TB outcomes, including mortality.

## REFERENCE

1. Ehrlich R, *et al*. The association between silica exposure, silicosis and tuberculosis: a systematic review and meta-analysis. *BMC Public Health.* 2021 May 20;**21**(1).

## M47 POST-TB CARE IN THE UK: CROSS-SECTIONAL ANALYSIS FROM A NATIONAL SURVEY

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**Introduction** Global research has shown high rates of morbidity among people who have previously experienced active tuberculosis (TB) disease. Guidelines in other countries now include standards for assessing and managing post-TB morbidity.

Current UK guidance (NICE) does not include guidance around end of treatment assessment, and does not consider follow up after treatment for drug-susceptible TB to be cost effective due to low rates of relapse.

**Abstract M47 Table 1** Survey responses among TB services in the UK to questions about post-TB morbidity, end of treatment assessments and challenges to providing post-TB care (n=113)

| | Number of services (%) |
|---|---|
| **Reported types of post-TB morbidity encountered** | |
| Post-TB Lung Disease | 93 (82%) |
| Post-TB cardiovascular and pericardial disease | 20 (18%) |
| Post-TB neurological morbidity | 46 (41%) |
| Problems with financial wellbeing | 78 (69%) |
| Problems with psychological wellbeing | 75 (66%) |
| Problems relating to social vulnerabilities | 89 (79%) |
| None of the above | 5 (4%) |
| **Reported end of TB treatment assessments** | |
| Symptom screen | 111 (98%) |
| Activities of daily living screen | 55 (49%) |
| Anxiety/depression screen | 26 (23%) |
| Cardiovascular risk screen e.g. QRISK | 1 (1%) |
| Pulmonary function testing (if PTB) | 25 (22%) |
| Chest X-ray (if PTB) | 108 (96%) |
| Diabetes screen | 15 (13%) |
| Asking about financial stability | 48 (42%) |
| **Reported challenges to providing post-TB care** | |
| Not applicable – not intending to improve or provide post-TB care | 6 (5%) |
| Lack of funding | 67 (59%) |
| Limited healthcare staff capacity | 88 (78%) |
| Limited clinic capacity e.g. space or availability of appointments | 79 (70%) |
| Lack of mention in guidelines | 36 (32%) |
| Lack of evidence supporting the need for post-TB services in the UK | 46 (41%) |
| Lack of evidence supporting the approach to care for post-TB morbidity | 29 (26%) |
| Lack of clinical expertise to manage post-TB morbidities | 23 (20%) |
| Lack of clear clinical need, within our patient population | 23 (20%) |
| Other | 0 (%) |
| Not sure | 4 (4%) |

We conducted a survey to understand current approaches to post-TB care among UK TB services.

**Methods** We identified and contacted 135 TB services by liaising with key national stakeholders in all four nations.

An e-survey was disseminated (February - May 2025) by email to specialists (Consultants and TB Nurses) representing TB services, with follow-up via reminder telephone calls and emails. We selected one response per service from the most senior clinician.

Survey responses were analysed descriptively.

**Results** Responses were received from 83.7% (113/135) of TB services.

The majority had encountered patients with post-TB lung disease (82%; 93/113) and problems relating to social vulnerabilities (79%; 89/113) (table 1).

End of treatment chest X-rays (for pulmonary TB, PTB) and symptom screening were reported by 98% (111/113) and 96% (108/113) of services. Few reported routinely offering pulmonary function testing after PTB (22%; 25/113) or screening for common comorbidities including diabetes (13%; 15/113) and cardiovascular risk (1%; 1/113).

Fewer than half (41%; 46/113) reported providing medical care routinely after treatment and only 11% (12/113) ongoing psychosocial support. TB services routinely providing post-TB support offer this at the discretion of the individual clinician (51%; 27/53), both informally (38%; 20/53) and via specific clinics (36%; 19/53).

The main reported challenges to providing post-TB care were resource-related, including limited healthcare staff (78%; 88/113), clinic capacity (70%; 79/113) and funding (59%; 67/113).

**Discussion** Specialists in TB services regularly encounter patients with physical and non-physical post-TB morbidity. Provision of post-TB care is currently limited and heterogenous in scope and delivery. There is interest in improving provision but resource-related challenges are a key limitation.

There is a need to identify patterns of morbidity among UK TB survivors, to guide standards for clinical practice.

## M48 REVEALING THE HIDDEN WORKLOAD OF TB SERVICES: USING ELECTRONIC PATIENT RECORDS TO SUPPORT WORKFORCE PLANNING

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Accurate planning of TB workforce and services requires recognition of all activity contributing to TB care. The 2024 GIRFT Respiratory Medicine report emphasises the need to align workforce models with the full scope of service delivery, stating: *'Many TB services lack visibility of the true workload*




**Abstract M48 Figure 1** Increase in number of TB attendances coded on electronic patient records from 1st April 2024 to 31st March 2025

*undertaken by nurses and consultants outside of formal clinics.'*

A substantial volume of service provision is performed outside standard clinics, yet this activity is often undocumented in formal service metrics. Additionally, workload generated outside of 'active TB treatment' is excluded from local and national reporting systems.

We aimed to capture the 'hidden' work carried out by our TB service. Using TB referral data, we retrospectively compared categories of new referrals made to the service and numbers started on active and latent TB treatment from 1st April 2024 to 31st March 2025. Coding data which captured new and follow up appointments recorded on electronic patient records (EPRs) was also reviewed.

In total, 1252 referrals were made to the TB service. Reasons for referral were available for Q2-Q4 (n=1014): symptomatic or abnormal result (46%) immunosuppression screening (26%), contact screening (20%), occupational health & other (8%). During this period, 147 patients were started on active and 203 on latent TB treatment. Additionally, a subset of referral work was found to take place during MDTs & email correspondence which is not reported as clinical activity on the EPR.

A total of 7060 TB attendances were coded (figure 1). E95.8 and E95.9 (TB attendances) were found to be the most commonly used codes by clinicians at e-checkout during clinics. There was a 34% increase in total clinic coding from April-Sep 2024 [n=3020] to October-March 2025 [n= 4040], this reflected roll out of enhanced recording and coding of clinical activity.

Hidden TB workload can be identified using multiple sources and is essential to deliver a safe, sustainable service. A live data reporting system nested within our clinical work flows will allow continuous monitoring of referrals, comparison with regional and national trends, and provide accurate data for quality improvement and audit to enable real-time responses to changes in patterns locally.

# Winter Meeting 2025 Declarations of interest

## T1 RETROSPECTIVE EVALUATION OF ARTIFICIAL INTELLIGENCE (AI) SOFTWARE FOR THE DETECTION OF ACUTE PULMONARY EMBOLISM (PE) IN CT PULMONARY ANGIOGRAMS (CTPA): A PRELIMINARY GROUND-TRUTH ANALYSIS FROM THE AID-PE STUDY

The AID-PE study (NCT06093217) received funding from the Artificial Intelligence in Health and Care Award. This is part of the NHS AI Lab, which has made funding available to accelerate the testing and evaluation of artificial intelligence technologies which meet the aims set out in the NHS Long Term Plan.

## T3 LUNG EOSINOPHILS IN EOSINOPHILIC COPD PATIENTS DIFFER FUNCTIONALLY BUT NOT PHENOTYPICALLY

This project is supported by Asthma and Lung UK Early Career Grant.

## T4 SEX DIFFERENCES IN ASTHMA: A LARGE-SCALE MULTI-OMICS STUDY

TSCH has received grants from Pfizer Inc., grants from the University of Oxford, grants from the Wellcome Trust, grants from the Medical Research Council, grants from The Guardians of the Beit Fellowship, and grants from the NIHR Oxford Biomedical Research Centre during the conduct of the study; and personal fees from Amgen, AstraZeneca / Pieris, Chiesi and Sanofi outside the submitted work.

## T5 REDUCED AIRWAY REMODELLING AND INCREASED EPITHELIAL BARRIER INTEGRITY IN BRONCHIAL BIOPSIES FROM PATIENTS WITH T2 HIGH SEVERE ASTHMA FOLLOWING MEPOLIZUMAB TREATMENT.

GSK funded.

## T6 GENETIC CHARACTERISATION OF PSEUDOMONAS AERUGINOSA ISOLATES FROM BRONCHIECTASIS PATIENTS IN A RANDOMIZED, DOUBLE-BLIND PLACEBO-CONTROLLED TRIAL OF A BISPECIFIC MONOCLONAL ANTIBODY TARGETING PSL AND PCRV (GREAT-2)

GREAT-2 is an investigator-initiated research study proposed by the EMBARC consortium. Financial and logistical support is provided by AstraZeneca and by the European Respiratory Society (ERS). Drug supply and PK analysis are by AstraZeneca.

## S1 RANDOMISED, PLACEBO-CONTROLLED TRIAL OF ASTEGOLIMAB FOR COPD WITH FREQUENT EXACERBATIONS: ALIENTO

Funding Genentech, Inc., a member of the Roche Group.
This abstract was submitted to ERS 2025.

## S9 A MATTER OF LIFE AND BREATH: UNDERSTANDING THE NEEDS OF PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The project was supported by AstraZeneca, GSK and Roche, but the funders had no influence on the design, recruitment, analysis or interpretation of the data

## S11 SPIRO-AID: A RANDOMIZED CONTROLLED TRIAL OF AI-ASSISTED SPIROMETRY INTERPRETATION IN PRIMARY CARE

The study is being funded by National Institute for Health Research (NIHR) through an AI Award in Health and Care (Phase 3- Application: Grant number AI_AWARD02204).

## S12 IMPROVING SAFETY AND TREATMENT PRECISION FOR ACUTE COPD EXACERBATIONS (TRACE): FEASIBILITY OF POINT-OF-CARE TESTING IN COMMUNITY PHARMACIES. A QUALITATIVE STUDY.

Funded by Dunhill Medical Trust Academy Ignition Award

## S25 IS AGEING ASSOCIATED WITH EXERTIONAL VENTILATORY CONSTRAINT?

Giovanni Cenerini has received funding from the European Respiratory Society Clinical Training Fellowship 2024

## S26 EVALUATION OF AN AUTOMATED SOFTWARE APPLICATION VERSUS CLINICIAN INTERPRETATION OF CARDIOPULMONARY EXERCISE TESTS

CB Cooper is the founder of enhancedCPETanalytics LLC used in this study.

## S28 FACTORS ASSOCIATED WITH BREATHLESSNESS AND DYSREGULATED BREATHING POST-COVID-19 AMONGST REFERRALS TO A RESPIRATORY CLINIC.

Charitable donation via Campaign & Alumni Relations Office, University of Nottingham and NIHR Nottingham BRC

## S29 PERIPHERAL MUSCLE OXYGEN EXTRACTION IS COMPROMISED DURING SUBMAXIMAL EXERCISE IN PREVIOUSLY HOSPITALIZED COVID-19 SURVIVORS

This work was submitted in BACPR Conference 2025

## S30 OXYGEN REQUIREMENT RISES EARLIER THAN THE TOTAL NATIONAL EARLY WARNING SCORE 2 (NEWS2) SCORE PRIOR TO DETERIORATION

PW holds grants from the National Institute for Health Research (NIHR) and has held grants from Wellcome and Sensyne Health (now Arcturis Data). He is supported by the NIHR Biomedical Research Centre, Oxford. He was previously Chief Medical Officer for Sensyne Health (now Arcturis Data), holds shares in the company and provides consultancy. He sits on the NIHR HTA General Funding Committee. There are no other competing interests reported. Funding: This study has been delivered through the National Institute for Health and Care Research (NIHR) Oxford Biomedical Research Centre (BRC). The views expressed are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care.

## S33 HARNESSING EXHALED BREATH FOR LUNG CANCER EARLY DETECTION. THE EXPEL STUDY RESULTS

Exhalation Technologies (Owner of inflammacheck) provided in-kind contributions to this study, including equipment.

## S34 DOES PROFILING PLEURAL FLUID CIRCULATING TUMOUR DNA (CTDNA) HAVE A ROLE IN IMPROVING DIAGNOSTIC YIELD IN MALIGNANT PLEURAL EFFUSION?

This work was presented at the UK pleural society 26th June 2025.

## S37 MOLECULAR DETECTION AND QUANTIFICATION OF PSEUDOMONAS AERUGINOSA IN BRONCHIECTASIS: DATA FROM THE GREAT-2 RANDOMIZED CONTROLLED TRIAL

GREAT-2 is an investigator-initiated research study proposed by the EMBARC consortium. Financial and logistical support is provided by AstraZeneca and by the European Respiratory Society (ERS). Drug supply and PK analysis are by AstraZeneca. Development and validation of the RNA-qPCR assay was funded by Lifearc.

## S38 EFFECTS OF BRENSOCATIB ON NEUTROPHIL SERINE PROTEASE LEVELS IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: A PHARMACODYNAMIC ANALYSIS OF THE ASPEN TRIAL

Mark L. Metersky received consulting fees from AN2 Therapeutics, Boehringer Ingelheim, Insmed Incorporated, Paratek, Renovion, Tactile Inc., and Zambon; participation on a Data Safety Monitoring Board for Verona Pharma, UK. Anthony De Soyza received grants and fees from 30 Technology, AstraZeneca, Bayer, Boehringer Ingelheim, Chiesi, Gilead Sciences, GSK, Infex, Insmed Incorporated, LifeArc, and Zambon. Pierre-Régis Burgel received grants from Vertex and GSK, outside the submitted work. Dr Burgel also reports clinical trials: advisory activity for AstraZeneca, Chiesi, GSK, Insmed Incorporated, MSD, Viatris, Vertex, Zambon; substantial financial contributions to the budget of his institution: Association Vaincre la Mucoviscidose, Filière MUCO-CFTR, GSK, Société Française de la Mucoviscidose, and Vertex. Charles L. Daley received grant support, advisory board fees, and consulting fees from Insmed Incorporated. Dr. Daley also reports grant support from AN2 Therapeutics, Bugworks, Juvabis, and Paratek Pharmaceuticals; advisory board work with AN2 Therapeutics, AstraZeneca, Cepheid, Galapagos, Hyfe, MannKind, Matinas Biopharma, NobHill, Spero Therapeutics, and Zambon; consulting with Genentech and Pfizer; data monitoring committee work with the Bill & Melinda Gates Foundation, and Otsuka Pharmaceutical. David Mauger received grants from NHLBI and drugs for NIH-funded clinical trials from Genentech, GSK, OM Pharma, Sanofi-Regeneron. Charles S. Haworth: consultancy/speaker fees from 30 Technology, AstraZeneca, BiomX, Chiesi, Infex, Insmed Incorporated, LifeArc, Pneumagen, and Zambon; advisory board work with 30 Technology, Insmed Incorporated, and Zambon; board member for the European Cystic Fibrosis Society. Ariel Teper, Carlos Fernandez, Kevin Mange, Chunpeng Fan, and Xiangmin Zhang are employees and shareholders in Insmed Incorporated. James D. Chalmers received grants and personal fees from AstraZeneca, Boehringer Ingelheim, GSK, Insmed Incorporated, and Zambon; a grant from Gilead; and personal fees from Chiesi and Novartis. This study was funded by Insmed Incorporated. Medical writing support was provided by Brooke Bartram (BSc) Hons, MSc, of Envision Spark, an Envision Medical Communications agency, and funded by Insmed Incorporated.

This abstract was presented at CHEST Annual Meeting; October 19–22, 2025. Boston, MA, USA

## S39 POLYPHARMACY IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: THE BRONCHUK OBSERVATIONAL STUDY

We acknowledge the original funding obtained from the MRC (MR/L011263/1) and ongoing financial support from the COPD Foundation. BronchUK was further supported by educational grants for network activities from Bayer, Chiesi, Forest, Teva and GSK. BronchUK is sponsored by the Newcastle upon Tyne NHS Foundation Trust.

## S40 BRONCHIECTASIS & MULTIMORBIDITY: A DATA ANALYSIS OF THE BRONCHUK REGISTRY TO DETERMINE THE ASSOCIATIONS BETWEEN MULTIMORBIDITY AND PATIENT OUTCOMES

Funded by the Medical Research Council and educational grants from GSK, Chiesi, Bayer and Teva

Some data in this abstract was presented at the 2025 European Respiratory Society Congress.

S41 **MORBIDITY IN PATIENTS WITH NCFBE AND EXACERBATIONS IN THE THIN UK DATABASE**

Michael Loebinger has received honoraria from Insmed and consulting fees from Armata, 30T, Astra Zeneca, Parion, Insmed, Chiesi, Zambon, Electromed, Recode, Boehringer Ingelheim, Ethris, Mannkind, and AN2 Therapeutics. Jennifer Quint received funding from Insmed for contributing to this work. Marko Obradovic, Deval Gor, and Joseph Feliciano are employees and shareholders of Insmed Incorporated. Dionisio Acosta-Mena, Alexia Callet, Idir Ghout, and Charlotte Renaudat, are employees of Cedegim, a paid consultant for Insmed Incorporated. This study was funded by Insmed Incorporated. Medical writing support was provided by Rosie Morley, PhD, of Envision Value & Access, a part of Envision Medical Communications, and funded by Insmed Incorporated

This abstract was previously presented at the 2025 European Respiratory Society Congress.

S42 **THE RESOURCE IMPACT OF BRONCHIECTASIS AND ASSOCIATED EXACERBATIONS: BASED ON A 5-YEAR PROSPECTIVE OBSERVATIONAL COHORT STUDY (BRONCHUK)**

This work was funded by an unrestricted medical education grant from Insmed to London School of Economics; De Soyza has received educational grants from Insmed, GSK, Chiesi, AstraZeneca

S43 **LONGITUDINAL TRENDS IN ASTHMA MORTALITY AND LIFE-THREATENING ASTHMA IN CHILDREN IN ENGLAND.**

Funded by Midlands Asthma and Allergy Research Association, with matched funding from NIHR, and Asthma + Lung UK. Aleksandra Gawlik-Lipinski: paid honoraria from Chiesi and AstraZeneca.

S44 **ARE CHILDREN ADMITTED WITH NEAR-FATAL ASTHMA RECEIVING THE CORE ELEMENTS OF CARE AT DISCHARGE? RESULTS FROM NRAP, THE NATIONAL RESPIRATORY AUDIT PROGRAMME.**

Aleksandra Gawlik-Lipinski: paid honoraria from Chiesi and AstraZeneca

S47 **THE UTILITY OF BLOOD EOSINOPHILS, IGE AND SKIN PRICK TESTING IN PAEDIATRIC ASTHMA DIAGNOSIS**

This abstract was presented at EAACI 2025

S48 **MEASUREMENT OF CHILDREN AND YOUNG PEOPLE'S ASTHMA MEDICATION ADHERENCE BY PATIENT/FAMILY REPORT, ELECTRONIC MONITORING DEVICES AND PRESCRIPTION REFILL RATE**

Rebecca Dobra is funded by the National Institute of Health Research as a Clinical Lecturer

S52 **AGREEMENT BETWEEN FREQUENT HOME-BASED OR CLINIC-BASED MONITORING OF FENO IN ASTHMA: A SECONDARY ANALYSIS OF THE INFORM ASTHMA TRIAL.**

The INFORM ASTHMA Trial was funded was provided through an investigator-initiated research grant from AstraZeneca.

S56 **MACHINE LEARNING AND ENRICHMENT ANALYSIS REVEAL PERSISTENT PROTEIN SIGNATURES IN LONGITUDINAL POST-COVID SYMPTOM TRAJECTORIES.**

Medical Research Council and National Institute for Health and Care Research

S57 **EP395, A NOVEL NON-ANTIBIOTIC MACROLIDE, ENHANCES ANTIVIRAL RESPONSES IN BRONCHIAL EPITHELIAL CELLS FROM ASTHMA PATIENTS**

Part of this work was sponsored by EpiEndo Pharmaceuticals

S59 **EFFECTS OF BACTERIA AND HEME ON NEUTROPHIL-ENDOTHELIAL INTERACTIONS IN COPD**

Funding provided by British Association of Lung Research (BALR) summer studentship.

S61 **PROTECTIVE EARLY INNATE IMMUNE ACTIVATION BY MITOCHONDRIAL ANTIVIRAL SIGNALLING PROTEIN (MAVS) IN BACTERIAL PNEUMONIA**

This work was supported by grants from the Wellcome Trust (104553/z/14/z, 211050/Z/18/z) and the National Institute for Health Research (NIHR) Oxford Biomedical Research Centre (BRC) (to TSCH). YL is supported by the Chinese Scholarship Council (CSC) and the Chinese Academy of Medical Sciences (CAMS) Innovation Fund for Medical Science (CIFMS), China (Grant Number: 2018-I2M-2-002). The views expressed are those of the authors and not those of the NHS or NIHR.

This abstract was presented in Poster at 2024 ERS, Vienna, in a preliminary form.

S69 **REDUCTION IN SERUM MARKERS OF INFLAMMATION IN PEOPLE WITH CYSTIC FIBROSIS WHO ARE CHRONICALLY INFECTED WITH PSEUDOMONAS AERUGINOSA IS SEEN AFTER TREATMENT WITH ELEXACAFTOR/TEZACAFTOR/IVACAFTOR**

This abstract will be presented at ECSF Milan 2025

S66 **A BINARY PROGNOSTIC SCORE, INCORPORATING FIBULIN-3, PREDICTS 1-YEAR SURVIVAL IN PLEURAL MESOTHELIOMA**

This abstract is due to be presented at ERS as an Oral Presentation in 2025

S67 **ASSOCIATION OF AIRFLOW OBSTRUCTION IDENTIFIED AT LUNG CANCER SCREENING WITH MAJOR ADVERSE CARDIOVASCULAR EVENTS**

Funding of first author fellowship from AstraZeneca.

S68 **LONG-TERM EFFECTS OF ELEXACAFTOR/TEZACAFTOR/IVACAFTOR ON CLINICAL OUTCOMES AND QUALITY OF LIFE AMONG ADOLESCENTS AND ADULTS AGED 12+ WITH CYSTIC FIBROSIS: INTERIM RESULTS FROM THE TRAJECTORY STUDY**

This study was funded by Vertex Pharmaceuticals Incorporated.

This abstract was accepted for presentation at the North American Cystic Fibrosis Conference (NACFC) taking place in Seattle, WA from Oct 22-25, 2025.

S70 **OXYGEN ENHANCED (OE-)MRI AND MULTIPLE BREATH WASHOUT WITH SHORT EXTENSION (MBWSHX) REVEAL LUNG DISEASE PROGRESSION IN PEOPLE WITH CYSTIC FIBROSIS DESPITE TRIPLE CFTR MODULATOR THERAPY**

This abstract was presented at ECFS 2025, Milan, Italy

S76 **THE CLINICAL IMPACT OF 3D PRINTED POSITIVE AIRWAY PRESSURE (PAP) THERAPY INTERFACES (3DPIPPIN): A RANDOMISED CONTROLLED TRIAL**

Funded by the NIHR.

This abstract was previously presented at the 2025 European Respiratory Society Congress.

S77 **TRANSCUTANEOUS ELECTRICAL STIMULATION-INDUCED FATIGUE IN THE TIBIALIS ANTERIOR: A SURROGATE MODEL FOR UPPER AIRWAY MUSCLE FUNCTION**

Governmental Fund (Saudi Arabian Cultural Bureau in London, KCL Student ID: 21133372/1, 21st July 2024)

S81 **EPIDEMIOLOGY, PROGNOSIS, AND HEALTHCARE RESOURCE USE OF PATIENTS WITH PULMONARY HYPERTENSION ASSOCIATED WITH LUNG DISEASE AND/OR HYPOXIA IN THE UK**

This study was supported by Ferrer Internacional, S.A

This work was presented at ISPOR - The Professional Society for Health Economics and Outcomes Research.

S86 **INTEGRATION OF IPF-ASSOCIATED SNPS WITH DIFFERENTIAL DNA METHYLATION AND GENE EXPRESSION SHOWS CELL-TYPE SPECIFIC MECHANISMS IN AIRWAY AND PARENCHYMAL FIBROBLASTS**

This work was supported by the Medical Research Council [Grant Number: MR/W007002/1].

S87 **TARGETING AKAP13 RHOGEF ACTIVITY AMELIORATES PRO-FIBROTIC PHENOTYPES DRIVEN BY THE IPF ASSOCIATED AKAP13 RISK VARIANT**

Supported by APF Mike Bray fellowship.

S91 **DEFINING THE ANTIFIBROTIC MECHANISMS OF TREPROSTINIL IN PULMONARY FIBROSIS**

JM receives funding via an MRC CRTF MR/X001814/1

S94 **THE LIVED EXPERIENCES OF PATIENTS USING POSITIVE AIRWAY PRESSURE (PAP) THERAPY: A NESTED PHENOMENOLOGICAL STUDY WITHIN THE 3DPIPPIN RANDOMISED CONTROLLED TRIAL**

Funded by the NIHR

S99 **CARDIAC ARRHYTHMIAS DURING STABLE COPD**

The project is part funded by AstraZeneca

S101 **IMPACT OF DUPILUMAB IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH CARDIOVASCULAR OR METABOLIC COMORBIDITIES: BOREAS AND NOTUS TRIALS**

Research sponsored by Sanofi and Regeneron Pharmaceuticals Inc. ClinicalTrials.gov Identifiers: NCT03930732 and NCT04456673. Hurst JR: AstraZeneca – grants; AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Regeneron Pharmaceuticals Inc., Sanofi – educational and/or advisory work. Rogliani P: AstraZeneca, Boehringer Ingelheim, Chiesi Farmaceutici, GSK, Menarini Group, Novartis, Recipharm, Regeneron Pharmaceuticals Inc., Roche, Sanofi – lecturer, scientific advisor, and courses; Arcede Pharma, Boehringer Ingelheim, Chiesi Farmaceutici, Novartis, Verona Pharma, Zambon – funding to department. Rabe KF: AstraZeneca, Boehringer Ingelheim, Chiesi, Gilead, GSK, Novartis, Pearl Pharmaceuticals, Sanofi,

Teva – consultant, speaker fees, advisory board member; rnatics, Germany – co-founder Hawkins NM: AstraZeneca – grants, speaker, advisor; Boehringer Ingelheim, Chiesi, GSK – educational and/or advisory work. Hansel NN: AstraZeneca, GSK, Regeneron Pharmaceuticals Inc., Sanofi – consultant; AstraZeneca, COPD Foundation, GSK, NIH – grants Vogelmeier CF: Aerogen, AstraZeneca, Boehringer Ingelheim, Chiesi, CSL Behring, Grifols, GSK, Insmed, Menarini, Novartis, Nuvaira, Roche, Sanofi – presentations at symposia and/or served on scientific advisory boards. Bhatt SP: The author is supported by NIH grants R01HL151421 and UH3HL155806. Apreo, AstraZeneca, Boehringer Ingelheim, Chiesi, Genentech, GSK, Merck, Polarean, Regeneron Pharmaceuticals Inc., Sanofi, Verona – advisory boards or consultant; Horizon CME, illuminate.health, Integritas Communications, Integrity CE, Medscape – honoraria; Genentech, Nuvaira, Regeneron Pharmaceuticals Inc., Sanofi – funds paid to institute for research. Xia C, Soliman M: Regeneron Pharmaceuticals Inc. – employees and shareholders. Heble J: Sanofi – employee, may hold stock and/or stock options in the company.

## S102 THE POTENTIAL EFFECT OF GLUCAGON-LIKE PEPTIDE 1 RECEPTOR AGONISTS (GLP1S) ON CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) EXACERBATIONS, A CASE CONTROL ANALYSES

Bernardino Alcázar reports grants and personal fees from GSK, grants, personal fees and non-financial support from AstraZeneca, personal fees and non-financial support from Boehringer Ingelheim, personal fees and non-financial support from Chiesi, grants, personal fees and non-financial support from Laboratorios Menarini, personal fees from Bial, personal fees from Zambon, personal fees from MSD, personal fees from PulmonX corporation and personal fees from Sanofi, outside the submitted work. Naveed Sattar has consulted for and/or received speaker honoraria from Abbott Laboratories, AbbVie, Amgen, AstraZeneca, Boehringer Ingelheim, Carmot Therapeutics, Eli Lilly, GlaxoSmithKline, Hanmi Pharmaceuticals, Menarini-Ricerche, Metsera, Novartis, Novo Nordisk, Pfizer, and Roche; and received grant support paid to his University from AstraZeneca, Boehringer Ingelheim, Novartis, and Roche outside the submitted work. Alan Kaplan is a member of the advisory board of, or speakers bureau for, ALK, AstraZeneca, Belus, Boehringer Ingelheim, Covis, Eisai, GlaxoSmithKline, Idorsia, Merck Frosst, Moderna, Novo Nordisk, Novartis, Pfizer, Purdue, Sanofi, Teva, Trudel and Valeo. Heath Heatley, John Townend, Derek Skinner and Victoria Carter are employees of Observational & Pragmatic Research Institute, Singapore. Tan Tze Lee is an advisory Board Member for Boehringer Ingelheim, AstraZeneca, Takeda, GlaxoSmithKline, Merck Sharp & Dohme, Mundipharma, and Janssen. Honoraria were received for these advisory boards. Honoraria were received for speaking at CMEs for AstraZeneca in the past. Conference sponsorships from AstraZeneca, Boehringer Ingelheim, Merck Serono, GlaxoSmithKline, Norvatis, Mundipharma and Merck Sharp & Dohme. Research grants from Merck Serono (Concor Study), Merck Sharp & Dohme (Apbord study). Mariko Siyue Koh reports grant support from AstraZeneca, and honoraria for lectures and advisory board meetings paid to her hospital (Singapore General Hospital) from GlaxoSmithKline, AstraZeneca, Novartis, Sanofi, and Boehringer Ingelheim, outside the submitted work. David B. Price has advisory board membership with AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Novartis, Viatris, Teva Pharmaceuticals; consultancy agreements with AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Novartis, Viatris, Teva Pharmaceuticals; grants and unrestricted funding for investigator-initiated studies (conducted through Observational and Pragmatic Research Institute Pte Ltd) from AstraZeneca, Chiesi, Viatris, Novartis, Regeneron Pharmaceuticals, Sanofi Genzyme, and UK National Health Service; payment for lectures/speaking engagements from AstraZeneca, Boehringer Ingelheim, Chiesi, Cipla, Inside Practice, GlaxoSmithKline, Medscape, Viatris, Novartis, Regeneron Pharmaceuticals and Sanofi Genzyme, Teva Pharmaceuticals; payment for travel/accommodation/meeting expenses from AstraZeneca, Boehringer Ingelheim, Novartis, Medscape, Teva Pharmaceuticals.; owns 74% of the social enterprise Optimum Patient Care Ltd (Australia and UK) and 92.61% of Observational and Pragmatic Research Institute Pte Ltd (Singapore); is peer reviewer for grant committees of the UK Efficacy and Mechanism Evaluation Programme, and Health Technology Assessment; and he was an expert witness for GlaxoSmithKline.

## S104 BLOOD AND AIRWAY EOSINOPHILS IN SEVERE EOSINOPHILIC ASTHMA ARE PHENOTYPICALLY AND FUNCTIONALLY DISTINCT

Study is investigator led, Industry sponsored (AstraZeneca)

## S105 THE AIRWAY TRANSCRIPTOME IN TYPE2 CYTOKINE BIOMARKER-HIGH AND -LOW SEVERE ASTHMA

PB has received research funding from Genentech via the University Hospitals of Leicester NHS Trust; consultancies for Boehringer-Ingelheim, Genentech, and Celldex Therapeutics via the University of Leicester. Support to attend scientific meetings from Chiesi, Teva and Sanofi Genzyme.

## S106 SPATIAL TRANSCRIPTOMIC PROFILING OF AIRWAY INFLAMMATION AND STRUCTURAL CHANGES IN ASTHMA

Funded by the NIHR and Wellcome Trust

## S108 MEPOLIZUMAB TREATMENT INFLUENCES AIRWAY REMODELLING AND IMPROVES EPITHELIAL CELL BIOLOGY IN SEVERE ASTHMA AS DETERMINED BY SINGLE CELL TRANSCRIPTOMICS

Funded by GlaxoSmithKline (GSK)

## S109 B CELL RECEPTOR REPERTOIRE SEQUENCING TO CHARACTERISE ANTIBODY SUBCLASS EXPRESSION AND SOMATIC HYPERMUTATION IN SEVERE EOSINOPHILIC ASTHMA AND EGPA

Funding for this study was provided by GSK [NCT04671446]. GSK was provided the opportunity to

review a preliminary version of this publication for factual accuracy, but the authors are solely responsible for final content and interpretation. PEP has attended advisory boards for AstraZeneca, GlaxoSmithKline and Sanofi; has given lectures at meetings/webinars, with/without honoraria, supported by AstraZeneca, Chiesi and GlaxoSmithKline; has attended international conferences with AstraZeneca; has taken part in clinical trials sponsored by AstraZeneca, GlaxoSmithKline, Regeneron and Sanofi; is conducting research funded by GlaxoSmithKline for which his institution receives remuneration and quality improvement activity at his institution supported by AstraZeneca. The remaining authors have no COI to report.

Half of the data is due to be presented at ERS 2025. The other half of the data has not been previously presented / accepted for presentation elsewhere. None of the data has been published / submitted for publication.

## S114 LONGITUDINAL CHANGE IN RESPIRATORY FUNCTION IN FIREFIGHTERS; RESULTS FROM THE GRENFELL FIREFIGHTER STUDY

The Grenfell Firefighter Study was funded by the Colt Foundation

## S124 DEVELOPMENT AND VALIDATION OF CHRONIC PULMONARY ASPERGILLOSIS HEALTH ASSESSMENT QUESTIONNAIRE (CPA-HAQ).

AS has received research grant funding from Pfizer and Astra Zeneca and funds for consultancy and advisory board work from Mundipharma.

## S125 NAVIGATING THE BOAT TRIAL DEVELOPMENT: A PPIE-LED JOURNEY TO IMPROVE PATIENT PARTICIPATION IN BRONCHIECTASIS RESEARCH

Funding for this study was obtained through NIHR Study within a trial (SWAT) funding stream

## S127 EXPLORING THE EFFECT OF DIFFERENT RESPIRATORY DISEASES ON PATIENTS' PERSPECTIVE OF ANTIMICROBIAL RESISTANCE: AN AMR-LUNG/ EUROPEAN LUNG FOUNDATION GLOBAL PATIENT SURVEY

There are no conflicts of interest pertaining to the data this abstract. It was produced as part of the clinical research collaboration with the European Lung Foundation.

## S132 ACHIEVING ASTHMA REMISSION WITH EXTRAFINE BECLOMETHASONE DIPROPIONATE, FORMOTEROL FUMARATE, AND GLYCOPYRRONIUM BROMIDE (BDP/FF/GB) PMDI IN ADULTS WITH MODERATE-TO-SEVERE ASTHMA: A POST-HOC ANALYSIS OF THE TRIMARAN AND TRIGGER STUDIES

Chiesi Ltd

This work was presented at AAAAI.

## S133 IMPROVEMENTS IN FEV1 AND ST GEORGE'S RESPIRATORY QUESTIONNAIRE (SGRQ) AT 4 AND 16 WEEKS ON BENRALIZUMAB ARE STRONGLY ASSOCIATED WITH CLINICAL REMISSION AT 1 YEAR

Acknowledgements: AstraZeneca [BenRex funder], BenRex TSC, RASP-UK, Vitalograph Ltd [UK], BOSCH [Germany] and the Innovative Medicines Initiative 2 Joint Undertaking (JU) under grant agreement No. 831434 (3TR) [support to J Logan].

This abstract will be presented as a poster at the 2025 ERS Congress.

## S135 TWICE-YEARLY DEPEMOKIMAB DEMONSTRATES EFFICACY IN PATIENTS WITH ASTHMA ACROSS BASELINE MEDIUM- AND HIGH-DOSE ICS SUBGROUPS: PHASE III SWIFT-1/2 STUDIES

Funding: GSK (206713/213744; NCT04719832/ NCT04718103). IDP in the last 5 years has received speaker's honoraria for speaking at sponsored meetings from AstraZeneca, Aerocrine, Almirall, Sanofi/Regeneron, Menarini and GSK, and payments for organising educational events from AstraZeneca, GSK and Sanofi/Regeneron. He has received honoraria for attending advisory panels with Sanofi/Regeneron, AstraZeneca, GSK, Merck, Circassia, Chiesi, Upstream Bio and Areteia. He has received sponsorship to attend international scientific meetings from GSK, AstraZeneca and Sanofi/Regeneron. MB has received research grants, advisory board, expert consultancy and lecture fees from AstraZeneca, Chiesi, Grifols, GSK, Lallemand, Lusofarmaco, Menarini, Omron and Sanofi. DJ, NB and AV are employees of GSK and hold financial equities in GSK. PC is an advisor for Boehringer Ingelheim, Boston Scientific, GSK, AstraZeneca, Novartis, Teva, Chiesi, Sanofi and SNCF; has received advisory board fees and/or honoraria from Boehringer Ingelheim, GSK, AstraZeneca, Novartis, Teva, Chiesi, Boston Scientific, ALK, AB Science, Argenx and Sanofi; has received grants from Roche, Boston Scientific, Boehringer Ingelheim, GSK, AstraZeneca, ALK and Novartis; is the elected president of the Scientific Committee de la Fondation du Souffle (2020-2025).

This is an encore abstract whose data was accepted at the European Respiratory Society - 35th Annual Congress; September 27–October 1, 2025; Amsterdam, Netherlands.

S136 **IMPLEMENTATION OF THE BTS CLINICAL STATEMENT ON PULMONARY REHABILITATION 2023: EVIDENCE FROM A COMMUNITY COPD SERVICE.**

T.N. has received speaker and travel grants from AstraZeneca, Chiesi and Sanofi, and a research grant from AstraZeneca; D. S. has received personal fees from Adovate, Aerogen, Almirall, Apogee, Arrowhead, AstraZeneca, Bial, Boehringer Ingelheim, Chiesi, Cipla, CONNECT Biopharm, Covis, CSL Behring, DevPro Biopharma LCC, Elpen, Empirico, EpiEndo, Genentech, Generate Biomedicines, GlaxoSmithKline, Glenmark, Kamada, Kinaset Therapeutics, Kymera, Menarini, MicroA, OM Pharma, Orion, Pieris Pharmaceuticals, Pulmatrix, Revolo, Roivant Sciences, Sanofi, Synairgen, Tetherex, Teva, Theravance Biopharma, Upstream and Verona Pharma. A.G.M has received honoraria from GlaxoSmithKline and Sanofi and has received non-financial support from Verona pharma, all not related to this work. T.J, J.K and A.B report no COI.

S137 **PREDICTORS OF MEETING THE MINIMAL CLINICALLY IMPORTANT DIFFERENCE IN PULMONARY REHABILITATION OUTCOMES USING A UK NATIONAL DATASET**

This work was funded in whole by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme (223512/Z/21/Z).

S139 **COMPARING THE EFFECTIVENESS OF CENTRE-BASED AND HOME-BASED PULMONARY REHABILITATION USING A UK NATIONAL DATASET**

This work was funded in whole by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme (223512/Z/21/Z).

S143 **STRATOSPHERE 1 STUDY - A NOVEL BMP TARGET ENGAGEMENT BIOMARKER PANEL FOR USE IN CLINICAL TRIALS DEMONSTRATES SOTATERCEPT ALTERS GENE EXPRESSION IN PAH**

Funding: MRC MR/T025646/1. I disclose no COIs. I couldn't see a box for this - I am applying for the early career prize and have mitigation for my age as I took an extended maternity/childcare break. The abstract here relates to my PhD that I have been doing part time (again child care related) and am at near completion.

This abstract was submitted as a late-breaking abstract to the 2025 ERS Congress

S145 **INTERROGATION OF THE GLYCOLYTIC AXIS IN PULMONARY ARTERIAL HYPERTENSION MODELS AND HOW MODIFYING THESE CAN LEAD TO POTENTIAL THERAPEUTIC OPTIONS.**

Funded by BHF and MRC

S146 **INFLAMMATION AND OBESITY CORRELATE IN PULMONARY HYPERTENSION BUT ASSOCIATE WITH DIVERGING OUTCOMES**

No COI (EDB is supported by the Gates Cambridge Trust - Bill & Melinda Gates Foundation Grant Number OPP1144)

This abstract is due to be presented at ERS - September 2025 Amsterdam.

S150 **REDUCE 2: A RANDOMISED FEASIBILITY STUDY COMPARING RECURRENT THERAPEUTIC THORACENTESIS TO INDWELLING PLEURAL CATHETER INSERTION, DAILY DRAINAGE AND TALC INSTILLATION FOR THE MANAGEMENT OF MEDICALLY-RESISTANT CARDIOGENIC PLEURAL EFFUSIONS.**

This study was funded by an unrestricted grant from Becton, Dickinson and Company. The funder had no role in study concept, design or analysis.

S151 **WHEN TO TEST FOR BIRT-HOGG-DUBÉ SYNDROME? UK NATIONAL DATA**

This abstract will be presented at ERS 2025

S160 **IMPROVED QUALITY OF LIFE, EARLY SYMPTOM CONTROL AND REDUCED SURGERY WITH MEPOLIZUMAB IN CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSWNP): RESULTS FROM THE SYNAPSE STUDY**

Funding: GSK (205687/NCT03085797). Acknowledgements: Medical writing support and coordination support for the original abstract were provided by Nathan Ley, PhD, at Fishawack Indicia Ltd, UK, part of Avalere Health, and was funded by GSK. Editorial support for the encore abstract was provided by Dr. Alap P. Chavda, an employee of GSK. This is an encore abstract whose data was accepted and earlier presented at the European Academy of Allergy and Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, Scotland. Conflict of interests/Disclosures: MW has received personal fees from ALK-Abelló, AstraZeneca, Genzyme, GSK, Allergie, InfectoPharm, LETI Pharma, Med update, Novartis, Regeneron, Sanofi and Stallergenes, and is a member of the executive committee (Treasurer), Head of the Section Otorhinolaryngology of the Deutsche Gesellschaft für Allergie und klinische Immunologie (German Society of Allergology and Clinical Immunology). Their institution has received payments from ALK-Abelló, AstraZeneca, GSK, Novartis, Regeneron, Sanofi-Aventis and Takeda. EDC has received lecture fees and participations in experts board meeting of GSK, Novartis, Sanofi and AstraZeneca. LW, LZ, ARS and PH are employees of GSK and hold financial equities in GSK. JM has received research grants from and attended speaker bureaus and/or advisory boards for Almirall, AstraZeneca, Glenmark, GSK, Lilly, Menarini, MSD, Noucor/Uriach, Regeneron Pharmaceuticals Inc., Sanofi-Genzyme and Viatris/MEDA Pharma.

This is an encore abstract whose data was accepted and earlier presented at the European Academy of Allergy and

Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, Scotland.

## S161 TEZEPELUMAB REDUCES INFLAMMATORY BIOMARKERS IN ADULTS WITH SEVERE CHRONIC RHINOSINUSITIS WITH NASAL POLYPS WITH AND WITHOUT COMORBID ASTHMA: RESULTS FROM THE PHASE 3 WAYPOINT STUDY

Joseph K Han has received consultancy fees from AstraZeneca, Genentech, GSK, Novartis, Regeneron Pharmaceuticals and Sanofi. Brian J Lipworth reports non-financial support (equipment) from GSK; has received grants, personal fees (consulting, talks and an advisory board) and other support from AstraZeneca, Boehringer Ingelheim, NIOX Group (Circassia) and Teva Pharmaceuticals; has received personal fees (consulting and talks) from Sanofi/Regeneron;has received grants, personal fees (consulting and talks) and other support from Chiesi; personal fees (consulting) from Glenmark, Lupin and Sandoz; has received grants and personal fees (consulting talks and advisory board) from AstraZeneca; and the son of Brian J Lipworth is currently an employee of AstraZeneca. Amin Allahyar, Mathias Cardner, James Cartwright, Greet De Baets, Michael Lehmann, Ting Li, Vaishali Mankad, Daniel Muthas, Adam Platt, Daniel Sutton and Joseph D Sherrill are employees of AstraZeneca and may own stock or stock options in AstraZeneca. Julie McLaren is an employee of Amgen and owns stock in Amgen. This study was funded by AstraZeneca and Amgen Inc. Medical writing support was provided by Freyja McClenahan, PhD, of PharmaGenesis London, London, UK, with funding from AstraZeneca and Amgen Inc.

This abstract will be presented at the European Respiratory Society (ERS) International Congress, 27 September–1 October 2025, Amsterdam, Netherlands.

## S162 EMERGING BIOLOGIC USE MAY REDUCE THE NEED FOR CORTICOSTEROID TREATMENT AND SURGERY FOR CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSWNP) IN FRANCE, GERMANY, ITALY, SPAIN AND THE UK

Funding: GSK (217519). Acknowledgements: Medical writing support and coordination support for the original abstract were provided by Avalere health, funded by GSK. Editorial support for the encore abstract was provided by Dr. Alap P. Chavda, an employee of GSK. This is an encore abstract whose data was accepted and earlier presented at the European Academy of Allergy and Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, Scotland. Conflict of interests/Disclosures: WA, JH, AE and LW are employees of GSK and hold financial equities in GSK. AH, LT and FB are employees of Adelphi Group, which received funding from GSK for the conduct of this study.

This is an encore abstract whose data was accepted and earlier presented at the European Academy of Allergy and Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, Scotland.

## S164 MEPOLIZUMAB IMPROVES AND SUSTAINS ASTHMA CONTROL AND LUNG FUNCTION IN PATIENTS WITH SEVERE ASTHMA, INDEPENDENT OF BASELINE IGE AND FENO IN THE REAL WORLD: 2-YEAR RESULTS FROM REALITI-A

Funding: GSK (204710). GWC has received research grants and fees from A. Menarini, AstraZeneca, Chiesi Farmaceutici, Faes, Firma, Genentech, Guidotti, GSK, Hal Allergy, Innovacaremd, OmPharma, RedMaple, Regeneron, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma.. MCL has received grants for clinical trials from AstraZeneca, Boehringer Ingelheim, GSK and Gossamer Bio, Sanofi and personal fees for participation in advisory boards from AstraZeneca, GSK and Gossamer Bio. CP has received fees for advisory boards, speaker meetings and research grants from GSK, AstraZeneca, Chiesi, Novartis, Teva and ALK-Abelló. JKL has received grants and research support from AbbVie, Allakos, Amgen, Celldex, Regeneron, GSK, Sanofi-Genzyme, Novartis, AstraZeneca, Takeda and Medexus; honoraria for speakers' bureau from AbbVie, Amgen, Sanofi-Genzyme, AstraZeneca, Novartis, Medexus, Aralez, Merck and GSK; and consulting fees from AbbVie, Amgen, GSK, Regeneron, Sanofi-Genzyme, Novartis and AstraZeneca. RC has received lecture fees from GSK, AstraZeneca, Teva, Chiesi, Sanofi and Novartis; honoraria for Advisory Board Meetings from GSK, AstraZeneca, Teva, Chiesi, Novartis, Celltrion; sponsorship to attend international scientific meetings from Chiesi, Napp, Sanofi and GSK and a research grant to her Institute from AstraZeneca for a UK multicentre study. SG, LZ, RA-C and PH are employees of GSK and hold financial equities in GSK.

This is an encore abstract where data was accepted and earlier presented as a Poster at the European Academy of Allergy and Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, UK.

## S168 NEUTROPHILS DISPLAY PHENOTYPIC AND FUNCTIONAL CHANGES INDICATIVE OF ACTIVATION IN A NOVEL IN VITRO MODEL OF IDIOPATHIC PULMONARY FIBROSIS

Funding received from the Wolfson Foundation / Royal College of Physicians (Wolfson Intercalated Award) and the British Association for Lung Research (BALR) / Action for Pulmonary Fibrosis (APF) (BALR/APF Summer Studentship)

## S169 INVESTIGATING THE EFFECT OF BARICITINIB ON NEUTROPHILIC INFLAMMATION IN AN EX VIVO LUNG PERFUSION (EVLP) MODEL OF ACUTE RESPIRATORY DISTRESS SYNDROME

Belfast Health and Social Care Trust Charitable Funds

## S170 NEUTROPHIL ELASTASE AND NETS CONTRIBUTE TO DISEASE EXACERBATION IN PLEURAL INFECTION

This abstract was presented at the European Respiratory Society Congress 2025.

## S174 REDUCTION OF EXACERBATIONS ACCORDING TO TYPE 2 INFLAMMATORY BIOMARKERS WITH DUPILUMAB TREATMENT IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Couillard S: AstraZeneca, bioMérieux, National Institute for Health and Care Research Oxford Biomedical Research Centre, Quebec Respiratory Health Research Network, Sanofi-Regeneron Pharmaceuticals Inc. – nonrestricted research grants; Association Pulmonaire du Québec – Research Chair in Respiratory medicine; AstraZeneca, GSK, Sanofi-Regeneron Pharmaceuticals Inc., Valeo Pharma – speaker honoraria; AstraZeneca, FirstThought, GSK, Sanofi-Regeneron Pharmaceuticals Inc. – consultancy fees; AstraZeneca – sponsorship to attend/speak at international scientific meetings; Biometry Inc. (a company developing a FeNO device [myBiometry]) – advisory board member and will have stock options. Ramakrishnan S: Charlie's Research Foundation, National Institute for Health and Care Research – salary support; AstraZeneca, Chiesi, GSK – speaker fees; AstraZeneca, Boehringer Ingelheim – conference travel support. Pavord ID: Aerocrine AB, Almirall, AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Novartis, Regeneron Pharmaceuticals Inc., Sanofi, Teva Pharmaceuticals – speaker fees; AstraZeneca, GSK, Regeneron Pharmaceuticals Inc., Sanofi, Teva Pharmaceuticals – payments for organization of educational events; Almirall, AstraZeneca, Boehringer Ingelheim, Chiesi, Circassia, Dey Pharma, Genentech, GSK, Knopp Biosciences, Merck, MSD, Napp Pharmaceuticals, Novartis, Regeneron Pharmaceuticals Inc., RespiVert, Sanofi, Scherring-Plough, Teva Pharmaceuticals – consultancy fees; AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Napp Pharmaceuticals, Regeneron Pharmaceuticals Inc., Sanofi, Teva Pharmaceuticals – international scientific meeting sponsorship; Chiesi – research grant. Çolak Y: Sanofi – grants; AstraZeneca, Boehringer Ingelheim, GlaxoSmithKline, Sanofi – personal fees. Buhl R: Boehringer Ingelheim, GSK, Novartis, Roche – grants to Mainz University; AstraZeneca, Berlin-Chemie, Boehringer Ingelheim, Chiesi, Cipla, GSK, Novartis, Roche, Sanofi, Teva – personal fees; all outside the submitted work. Deslée G: AstraZeneca, Chiesi, GSK, Sanofi – consulting fees. Bauer D, Heble J: Sanofi – employees, may hold stock and/or stock options in the company. Soliman M: Regeneron Pharmaceuticals Inc. – employees and shareholders.

This abstract was presented at the CHEST Annual Meeting; October 19-22, 2025; Chicago, Illinois.

## S175 TRACKING EOSINOPHILS FOLLOWING PREDNISOLONE OR BENRALIZUMAB AT THE TIME OF AN EXACERBATION IN THE ABRA TRIAL

The ABRA study was funded by AstraZeneca as an unrestricted investigator initiated research grant to the senior investigator

## P1 CURRENT PRACTICES, PERCEPTIONS AND FUTURE CHALLENGES IN COPD MANAGEMENT.

This work was previously presented at the 2025 European Respiratory Society Congress.

## P2 DISEASE STABILITY, MANAGEMENT AND TREATMENT GOALS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE AMONG NEWLY DIAGNOSED VS LONG-TERM DIAGNOSED PATIENTS: A PATIENT SURVEY

David Halpin reports personal fees from Aerogen, AstraZeneca, Boehringer Ingelheim, Berlin-Chemie, Chiesi, CSL Behring, GSK, Inogen, Menarini, Novartis, Pfizer and Sanofi. Alberto Papi reports grants or contracts from Chiesi, AstraZeneca, GSK, Sanofi and Agenzia Italiana del Farmaco, outside the submitted work; consulting fees from Chiesi, AstraZeneca, GSK, Novartis, Sanofi, Avillion and Elpen Pharmaceutica, outside the submitted work; payment or honoraria for lectures, presentations, speakers' bureaus, manuscript writing or educational events from Chiesi, AstraZeneca, GSK, Menarini, Novartis, Zambon, Mundipharma, Sanofi, Edmond Pharma, Iqvia, Avillion and Elpen Pharmaceuticals, outside the submitted work; and participation on advisory boards for Chiesi, AstraZeneca, GSK, MSD, Novartis, Sanofi, Iqvia, Avillion and Elpen Pharmaceuticals, outside the submitted work. Tharishini Mohan, Anurita Majumdar, and Mina Al-Ameri are employees of, and or hold financial equities in, GSK.

The original abstract was presented at the American Thoracic Society 2025 congress, San Francisco, CA, USA, May 16–21 (poster P262).

## P8 REAL-WORLD STUDY ON CAUSES OF DEATH IN PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) IN ENGLAND

The study was funded by AstraZeneca. Hana Mullerova, the presenting author, is an employee of AstraZeneca and may hold stock and/or stock options in the company

This abstract was accepted at ERS 2025, September 27 - October 1, Amsterdam, Netherlands

## P10 BREATHLESS SYSTEMS: COPD PRESSURES IN HOSPITALS ACROSS ENGLAND

This work was funded by AstraZeneca. Professor M Bafadhel has received speaker honoraria and grant funding from AstraZeneca. Dr R Russell has received speaker honoraria from AstraZeneca.

## P13 SEASONAL VARIATION IN EXACERBATION RISK FOLLOWING SEVERE AECOPD

Study was investigator initiated but funding from GSK

## P14 UNDER PRESSURE: PATIENT CHARACTERISTICS ASSOCIATED WITH PRIMARY CARE RESPIRATORY TREATMENT REVIEWS BEFORE RESPIRATORY WINTER PRESSURES IN THE UK

This study was funded by AstraZeneca. PEP has attended advisory boards for AstraZeneca, GlaxoSmithKline and Sanofi; has given lectures at meetings/webinars, with/without honoraria, supported by AstraZeneca, Chiesi and GlaxoSmithKline;

has attended international conferences with AstraZeneca; has taken part in clinical trials sponsored by AstraZeneca, GlaxoSmithKline, Regeneron and Sanofi; is conducting research funded by GlaxoSmithKline for which his institution receives remuneration and quality improvement activity at his institution supported by AstraZeneca. AM has received honoraria for lectures, conference travel and advisory boards from AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Roche and Sanofi. JGM, KS, MW and MH are employed by, and may hold stock in, AstraZeneca.

P17 **IMPACT OF VIRTUAL NURSE/PHARMACIST-LED MONITORING IN PATIENTS WITH INTERSTITIAL LUNG DISEASE (ILD) ON ANTIFIBROTIC TREATMENT IN A UK ILD SPECIALIST CENTRE**

This abstract is due to be presented at the ERS Congress in 2025

P23 **NINTEDANIB IN SYSTEMIC SCLEROSIS-ASSOCIATED INTERSTITIAL LUNG DISEASE: REAL-WORLD MULTICENTER COHORT STUDY ON TOLERABILITY AND DISCONTINUATION**

Funding: none; Conflict of interest statement: C.P.D. reports consulting fees from Janssen, GlaxoSmithKline, Boehringer Ingelheim, Roche, CSL Behring, Corbus, Acceleron, Horizon, Arxx Therapeutics, Lilly, Novartis, Certa Therapeutics, Zurabio and research grants from Abbvie, GlaxoSmithKline, Horizon, Arxx Therapeutics and Servier. A.U.W.: consulting fees from Boehringer Ingelheim, Roche, Veracyte, and Chiesi, lecture fees from Boehringer Ingelheim, and is the President of WASOG. V.H.O.: speaker fees from Boehringer Ingelheim. E. A.R.: advisory board and lecture fees from Boehringer Ingelheim and Roche, and lecture fees from Mundipharma, paid to her institution. The remaining authors have declared no conflicts of interest.

P30 **EFFECT OF DIETARY NITRATE SUPPLEMENTATION ON EXERCISE PERFORMANCE IN HYPOXIC IPF: THE EDEN-OX 3 STUDY**

Funder: 1. The Moulton Medical Foundation. 2. Saudi Arabian Cultural Bureau - UK, Ministry of Education – Riyadh, Saudi Arabia.

P31 **A QUALITATIVE EXPLORATION OF THE PERCEPTION OF MAINTENANCE NEEDS THREE MONTHS AFTER REHABILITATION IN PATIENTS WITH LONG COVID**

KK is undertaking a PhD funded by the Wellcome Trust

P32 **PREDICTING ENROLMENT TO PULMONARY REHABILITATION IN THE UK**

This work was funded in whole by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme (223512/Z/21/Z).

P36 **IMPROVING THE UPTAKE AND COMPLETION OF PULMONARY REHABILITATION (PR): DEVELOPMENT AND EVALUATION OF A TRAIN-THE-TRAINER PROGRAMME TO ESTABLISH A LAY HEALTH WORKER SERVICE.**

Funded by the National Institute for Health Research (NIHR). Funding grant number: NIHR130999.

P38 **PATIENT, FAMILY MEMBER, AND CLINICIAN EXPERIENCES OF PHYSIOTHERAPY AIRWAY CLEARANCE TECHNIQUES DURING HOSPITAL ADMISSION: A SCOPING REVIEW.**

The author KJ is a PhD Fellow, and this review is one of the outputs of her PhD Project. The work is funded by the Northern Ireland Public Health Agency (Research and Development Division) [EAT/5781/24] as part of the Doctoral Fellowship Programme. The authors BON, JB and BC are also authors of one of the studies included in this review. To mitigate bias, screening, eligibility confirmation, data extraction, and analysis were conducted independently by other team members. These authors participated only in aggregate-level analysis and synthesis, with no involvement in the assessment of their own study. BON, JB and BC are the academic supervisor of KJ. In addition, BC declares research leadership roles in UK organisations (UK Critical Care Research Group, Intensive Care Society, NIHR Critical Care National Specialty Group).

P43 **IMPACT OF INTEGRATED PULMONARY AND CARDIAC REHABILITATION: A HOLISTIC APPROACH TO PATIENT CARE IN A 10-WEEK PILOT STUDY**

Local non recurrent funding received from the integrated care board.

This work was presented at the Association of Cardiovascular Nursing and Allied Professionals (ACNAP) conference, a sub division of European Society of Cardiology presented on 13/06/2025 in Sophia Antipolis, France

P50 **A PILOT DIGITAL PATHWAY FOR REMOTE DETECTION OF SLEEP DISTURBANCE IN CARDIOMETABOLIC DISEASE**

This abstract will be presented at NIHR UKCRF Birmingham Conference (Mid july 2025)

P61 **BARRIER AND FACILITATORS TO PRIMARY CARE PRACTITIONER PRESCRIBING AND PATIENT ADOPTION OF ANTI-INFLAMMATORY, MAINTENANCE AND RELIEVER THERAPY (MART) FOR ADULTS WITH ASTHMA**

Sponsored and funded by AstraZeneca

P66 **IMPROVING ASTHMA OUTCOMES IN LANCASHIRE AND SOUTH CUMBRIA (L&SC) THROUGH SABA REDUCTION AND MART IMPLEMENTATION WITH SENTINEL PLUS.**

This is a joint working collaboration with AstraZeneca and the Lancashire and South Cumbria ICB

P67 **WHAT PROPORTION OF PATIENTS ATTENDING A&E WITH ACUTE ASTHMA COULD BE DISCHARGED ON MART/AIR?**

The AMEND quality improvement project was funded by AstraZeneca through an Education Grant and AstraZeneca was not involved in the development of the programme educational materials. The views expressed are those of the authors and not necessarily those of AstraZeneca. The AMEND project builds upon IS's PhD studentship from AUKCAR, which was funded by Asthma+Lung UK as part of the Asthma UK Centre for Applied Research [AUK-AC-2012-01 and AUK-AC-2018-01]. PEP has attended advisory boards for AstraZeneca, GlaxoSmithKline and Sanofi; has given lectures at meetings/webinars, with/without honoraria, supported by AstraZeneca, Chiesi and GlaxoSmithKline; has attended international conferences with AstraZeneca; has taken part in clinical trials sponsored by AstraZeneca, GlaxoSmithKline, Regeneron and Sanofi; is conducting research funded by GlaxoSmithKline for which his institution receives remuneration and quality improvement activity at his institution supported by AstraZeneca. Remaining authors have no COI to report.

P68 **ARE HOSPITAL ORGANISATIONAL FACTORS ASSOCIATED WITH THE DELIVERY OF THE ADULT ASTHMA DISCHARGE BUNDLE? AN ANALYSIS OF DATA FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME.**

This work was supported by the National Respiratory Audit Programme (NRAP) which is hosted by the Royal College of Physicians (RCP) and commissioned by the Healthcare Quality Improvement Partnership (HQIP) as part of the National Clinical Audit Patient Outcomes Programme (NCAPOP).

P69 **INTENSIVE CARE ADMISSIONS FOR ASTHMA ARE CHARACTERIZED BY FREQUENT NON-ATTENDANCE TO OUTPATIENT APPOINTMENTS**

R.W. Costello reports founding Phyxiam: grants from GSK, Therevance and Aeragen: patents on acoustics to assess inhaler use; patents on assessing adherence; and patents on detecting and predicting exacerbations. All other authors have nothing to disclose.

P75 **DEVELOPMENT OF THE UK SEVERE ASTHMA REGISTRY AS A PERFORMANCE-MONITORING QUALITY IMPROVEMENT PLATFORM**

No specific funding was received for this work. The lead author (PEP) has attended advisory boards for AstraZeneca, GlaxoSmithKline and Sanofi; has given lectures at meetings/webinars, with/without honoraria, supported by AstraZeneca, Chiesi and GlaxoSmithKline; has attended international conferences with AstraZeneca; has taken part in clinical trials sponsored by AstraZeneca, GlaxoSmithKline, Regeneron and Sanofi; is conducting research funded by GlaxoSmithKline for which his institution receives remuneration and quality improvement activity at his institution supported by AstraZeneca.

P86 **THE ACCURACY OF ICD-10 DIAGNOSTIC CODES FOR THE IDENTIFICATION OF PNEUMOTHORAX AND PULMONARY EMBOLISM: A RETROSPECTIVE STUDY IN TWO UK CENTRES.**

This work was submitted to the 2025 ERS Congress.

P89 **A 'CALL TO ACTION' IN DETERMINING AND ESTIMATING A MINIMUM IMPORTANT DIFFERENCE (MID) IN 6-MINUTE WALKING DISTANCE (6MWD) IN PH-ILD**

This study was supported by Ferrer Internacional, S.A

This work was presented at ISPOR - The Professional Society for Health Economics and Outcomes Research.

P90 **SEEING WHAT'S IMPORTANT: ELICITING EXPERT JUDGEMENTS TO ESTIMATE THE MINIMALLY CLINICALLY IMPORTANT DIFFERENCE FOR SIX-MINUTE WALKING DISTANCE IN PH-ILD'**

This study was supported by Ferrer Internacional, S.A

This work was presented at ISPOR - The Professional Society for Health Economics and Outcomes Research.

P91 **REAL-WORLD HEALTHCARE RESOURCE UTILIZATION IN PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS INITIATING ANTIFIBROTIC THERAPY IN THE MEDICARE FEE-FOR-SERVICE POPULATION**

United Therapeutics Corporation sponsored the study

This abstract was presented at ISPOR 2025, Montreal, Canada

## P95 EVALUATION OF CHATGPT AND GEMINI IN GENERATING DIFFERENTIAL DIAGNOSES IN DIFFUSE INTERSTITIAL LUNG DISEASES (DILDS) FROM TRANSCRIBED RADIOLOGICAL REPORTS

Imperial College School of Medicine

## P100 PLACE OF DEATH IN PEOPLE WITH PULMONARY FIBROSIS: A COHORT STUDY USING ELECTRONIC HEALTHCARE RECORDS IN ENGLAND

VN is funded by The Laurent Fellowship for Respiratory Health

## P105 AN AUDIO TRIMMING ALGORITHM FOR COUGH ANALYSIS IN AMBULATORY RECORDINGS: A PROOF-OF-CONCEPT STUDY.

: TT, HV, DD, ET, YZ & FK are employees of Vitalograph. JS consults for Vitalograph Ltd and her hospital receives royalties from Vitalograph for use of an audio compression algorithm.

## P112 DIFFERENCES IN AX-8 AND PLACEBO EFFECTS ON COUGH BOUTS IN REFRACTORY OR UNEXPLAINED CHRONIC COUGH: A NEW COUNT- AND DURATION-BASED ANALYSIS

Study funded by Axalbion. JAS is a paid consultancy fees for providing advice to Axalbion. Vitalograph pays royalties to the hospital in which JAS works.

## P113 EFFICACY AND SAFETY OF NALBUPHINE EXTENDED-RELEASE IN REFRACTORY CHRONIC COUGH: RESULTS FROM THE PHASE 2A RIVER TRIAL

This study was funded by Vertex Pharmaceuticals Incorporated.

This abstract was previously presented at the 2025 European Respiratory Society Congress.

## P115 ASSOCIATION BETWEEN BASELINE COUGH BURDEN AND GEFAPIXANT DISCONTINUATIONS DUE TO TASTE-RELATED ADVERSE EVENTS

Funding for this research was provided by Merck Sharp & Dohme LLC, a subsidiary of Merck & Co., Inc., Rahway, NJ, USA.

Data included in this abstract have previously been presented in full at European Respiratory Society; September 27-October 1, 2025; Amsterdam, Netherlands.

## P116 CLINICALLY RELEVANT ATTENUATION OF COUGH WITH GEFAPIXANT: A POST HOC ANALYSIS

Funding for this research was provided by Merck Sharp & Dohme LLC, a subsidiary of Merck & Co., Inc., Rahway, NJ, USA.

Data included in this abstract have previously been presented in full at European Respiratory Society; September 27-October 1, 2025; Amsterdam, Netherlands.

## P117 PATIENT GLOBAL IMPRESSION OF CHANGE: CONTEXTUALIZING PATIENT-REPORTED OUTCOMES IN GEFAPIXANT PHASE 3 TRIALS

Funding for this research was provided by Merck Sharp & Dohme LLC, a subsidiary of Merck & Co., Inc., Rahway, NJ, USA.

Data included in this abstract have previously been presented in full at European Respiratory Society; September 27-October 1, 2025; Amsterdam, Netherlands.

## P118 UNDERSTANDING THE EXPERIENCE AND UNMET NEEDS OF PATIENTS LIVING WITH SEVERE ASTHMA ON BIOLOGIC THERAPY; INCLUDING VIEWS ON CLINICAL REMISSION

Vishal Patel and Mandeep Moore are employees of GSK. Liam Heaney is the Academic Lead for the UK MRC Consortium for Stratified Medicine in Severe Asthma; received speaker/lecture fees from AZ, Sanofi, Circassa, GSK and Teva; has received grant funding from GSK and AZ; been involved in clinical trials with GSK, AZ and Roche/Genentech; has received travel funding from AZ, Sanofi, Teva and GSK; participated in advisory boards for GSK, AZ and Celltrion. Dr Llinos Jones participated in lectures and received honoraria from Astra Zeneca, Sanofi, GSK, Guys and St Thomas' Hospital. Pujan H Patel participated in lectures, presentations and attended meetings and received honoraria from GlaxoSmithKline (GSK), AstraZeneca, Regeneron / Sanofi Chiesi. Hitasha Rupani participated in lectures and received honoraria from GSK, AZ, Sanofi; received Grants from GSK and Consulting fees from Areteia. Andrew Whittamore is the clinical lead for Asthma and Lung UK; worked with Sanofi and AZ as part of Asthma and Lung UK joint working agreement; received speaker/lecture fees from GSK, NIOX (formely Circassa), Sanofi and Chiesi; received conference travel funding from NAPP and Chiesi; study advice and manuscript writing services for Chiesi. Alex James Fynney, Daniele Montalto, Gareth Turley have no conflicts of interest to declare.

## P119 IMPLEMENTING INHALER RECYCLING SCHEMES - EXPERIENCE WITHIN A LARGE UK NHS TRUST

This project was supported by a project grant from Chiesi Limited, who had no input to the design or running of the project.

P120 **HOW CAN WE EMBED SUSTAINABILITY IN THE INHALER PRESCRIPTION-USE-DISPOSAL PATHWAY?**

The study was funded by GSK

P123 **DOES ETHNICITY INFLUENCE THE INVESTIGATIVE PATHWAY AFTER PRESENTATION WITH BREATHLESSNESS? – AN EPIDEMIOLOGICAL STUDY USING CPRD RECORDS**

This work is supported by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme [223512/Z/21/Z] and the National Institute for Health and Care Research (NIHR) Leicester Biomedical Research Centre (BRC). The views expressed are those of the authors and not necessarily those of the NIHR or the Department of Health and Social Care.

P124 **INFLUENCE OF DEPRIVATION ON COPD PATIENTS WITH RESPIRATORY ACIDAEMIA REQUIRING NON-INVASIVE VENTILATION.**

National Institute for Health Research (RfPB)

P125 **DEDICATED RESPIRATORY SERVICE FOR ROUGH SLEEPERS**

GSK have provided a small amount of funds to cover food and taxi vouchers for patients to support the service.

P126 **UNDERSTANDING THE ACCEPTABILITY AND PROCESS MECHANISMS OF A PATHWAY NAVIGATION INTERVENTION TO ENGAGE NON-RESPONDERS IN LUNG CANCER SCREENING.**

This work is funded by Yorkshire Cancer Research (Ref L403). No conflicts of interest declared.

P128 **PATIENT- AND PHYSICIAN-REPORTED SYMPTOM BURDEN AMONG PATIENTS WITH BRONCHIECTASIS IN EUROPE**

Data collection was undertaken by Adelphi Real World as part of an independent survey, the Adelphi Real World Bronchiectasis DSP. The DSP is wholly owned by Adelphi Real World. Insmed are one of multiple subscribers to the DSP. This study was funded by Insmed Incorporated. Urte Sommerwerck has received honoraria paid to their institution from Insmed, DGIM, Pneumology Refresher Cologne, and honoraria from IGES, and has participated in advisory boards for Insmed and AMNOG. Mark Small and Denise Baldock are employees of Adelphi Real World. Prina Donga, Melanie Lauterio, and John Fastenau are employees and shareholders of Insmed Incorporated. Medical writing support was provided by Rosie Morley, PhD, of Envision Value & Access, a part of Envision Medical Communications, and funded by Insmed Incorporated.

P133 **IMPACT OF ELEXACAFTOR/TEZACAFTOR/IVACAFTOR ON CLINICAL OUTCOMES AND QUALITY OF LIFE IN CHILDREN WITH CYSTIC FIBROSIS AGED 6-11 YEARS IN THE REAL-WORLD SETTING**

This study was funded by Vertex Pharmaceuticals Incorporated.

This abstract was accepted for presentation at the North American Cystic Fibrosis Conference (NACFC) taking place in Seattle, WA from Oct 22-25, 2025.

P140 **HOME SPIROMETRY TO OPTIMISE RESPIRATORY SECONDARY CARE NEW PATIENT APPOINTMENTS**

Clinical Team (respiratory physiologists) - Employees of NuvoAir

P144 **WHAT INFORMATION SHOULD A PAEDIATRIC ASTHMA DISCHARGE LETTER CONTAIN - A DELPHI STUDY.**

Aleksandra Gawlik-Lipinski: paid honoraria from Chiesi and AstraZeneca

P145 **IMPACT OF BREATHING PATTERN INTERVENTIONS ON BREATHING PATTERN DISORDERS IN TEENAGERS WITH POST-COVID SYNDROME**

Funded by Action Medical Research & RBH Charity

P148 **AI-DRIVEN IDENTIFICATION OF HIGH-RISK COPD PATIENTS FOR BIOLOGIC THERAPY: PATHWAY DEVELOPMENT OPPORTUNITIES**

This abstract was previously presented at the 2025 European Respiratory Society Congress.

P149 **THE LANDSCAPE OF MARKETED AND PIPELINE DIAGNOSTIC TESTS FOR CHRONIC LUNG CONDITIONS**

LifeArc funded

P150 **DIAGNOSTIC CLASSIFICATION OF THE EMPHYSEMA SUBTYPE OF COPD USING N-TIDAL® CAPNOGRAPHY**

The studies which provided the data for this report were funded by F. Hoffmann-La Roche AG.

This abstract was presented at ATS 2025.

P151 **HOME HIGH-FLOW THERAPY: INSIGHTS FROM REMOTE-MONITORING AND LINKED ROUTINE CLINICAL DATA**

Supported by an unrestricted ResMed investigator initiated research award.

This abstract was previously presented at the 2025 European Respiratory Society Congress.

## P154 BLOOD EOSINOPHIL COUNTS AND ESCALATION TO HIGH DOSE ICS, IN COPD PATIENTS FROM A COMMUNITY SERVICE.

T.N. has received speaker and travel grants from AstraZeneca, Chiesi and Sanofi, and a research grant from AstraZeneca; D. S. has received personal fees from Adovate, Aerogen, Almirall, Apogee, Arrowhead, AstraZeneca, Bial, Boehringer Ingelheim, Chiesi, Cipla, CONNECT Biopharm, Covis, CSL Behring, DevPro Biopharma LCC, Elpen, Empirico, EpiEndo, Genentech, Generate Biomedicines, GlaxoSmithKline, Glenmark, Kamada, Kinaset Therapeutics, Kymera, Menarini, MicroA, OM Pharma, Orion, Pieris Pharmaceuticals, Pulmatrix, Revolo, Roivant Sciences, Sanofi, Synairgen, Tetherex, Teva, Theravance Biopharma, Upstream and Verona Pharma. A.G.M has received honoraria from GlaxoSmithKline and Sanofi and has received non-financial support from Verona pharma, all not related to this work. A.B, T.J, Z.P and R.D report no COI.

## P156 PREVALENCE OF CARDIOPULMONARY EVENTS AND RISK FACTORS BEFORE COPD DIAGNOSIS: ABACOS RISKS STUDY

The study was funded by AstraZeneca. Richard Russell, the presenting author, has no relevant conflict of interest to declare

The abstract is submitted as a late breaker at ERS 2025, September 27- October 1, 2025, in Amsterdam, Netherlands

## P157 EFFECT OF BENRALIZUMAB ON AIRWAY FUNCTION MEASURED BY IMPULSE OSCILLOMETRY DURING EOSINOPHILIC EXACERBATIONS: RESULTS FROM THE ABRA STUDY

The ABRA study was funded by AstraZeneca through an unrestricted investigator-initiated research grant awarded to the senior investigator (MB)

## P169 COMPARATIVE COST-EFFECTIVENESS OF SCREENING FOR LUNG CANCER USING DIFFERENT RISK SCORES AND THRESHOLDS: THE YORKSHIRE LUNG SCREENING TRIAL

Disclosure Prof. Callister declares receiving grants from Yorkshire Cancer Research and NIHR. Prof. Crosbie declares receiving grants from Yorkshire Cancer Research; receiving payment or honoraria from Bayer; and having stock options in Everest Detection. The remaining authors declare no conflict of interest. Acknowledgments We acknowledge the contribution of the whole YLST clinical team in delivering this study. The study was funded by Yorkshire Cancer Research (award reference L403). The funders had no input in the design and conduct of the study. Prof. Crosbie is supported by the Manchester National Institute for Health Research Manchester Biomedical Research Centre (IS-BRC-1215-20007).

## P170 DIAGNOSES OF NEVER-SMOKERS REFERRED TO THE LUNG CANCER MULTI-DISCIPLINARY TEAM: LUNG CANCER AND BEYOND

SBN is funded by the National Institute of Health and Care Research (NIHR) (Doctoral Research Fellow, NIHR303686) and Ruth Strauss Foundation. The views expressed in this publication are those of the author(s) and not necessarily those of the NIHR, NHS or the UK or the Department of Health and Social Care.

## P183 ENHANCING ANTIBIOTIC EFFECTIVENESS USING THE REPURPOSED MULTIPLE SCLEROSIS DRUG, GLATIRAMER ACETATE, TO COMPROMISE MEMBRANE INTEGRITY OF CF-RELEVANT PATHOGENIC BACTERIA.

Funding: LifeArc and Cystic Fibrosis Trust

This abstract was previously presented at ECFS Milan 2025

## P185 INCIDENCE OF DIABETES MELLITUS FOLLOWING HOSPITALISATION WITH INFLUENZA

Funding received from NIHR Nottingham BRC

## P187 DIVERSITY OF PSEUDOMONAS AERUGINOSA IN NON-CYSTIC FIBROSIS BRONCHIECTASIS: INSIGHTS FROM A NORFOLK PATIENT POPULATION

Local QIB Clinical Seedcorn Funded Work

## P191 INVESTIGATING THE RELATIONSHIP BETWEEN INTERLEUKIN-6 (IL6) AND HEALTH OUTCOMES ONE YEAR AFTER HOSPITAL DISCHARGE FOR COVID-19

Medical Research Council and National Institute for Health and Care Research

## P193 DEVELOPMENT OF A MULTI-MODAL MODEL TO PREDICT ASTHMA OUTCOMES USING DIGITAL MONITORING TOOLS

R.W. Costello reports founding Phyxiam: grants from GSK, Therevance and Aeragen: patents on acoustics to assess inhaler use; patents on assessing adherence; and patents on detecting and predicting exacerbations. All other authors have nothing to disclose.

## P197 DUPILUMAB CAUSES IMPROVEMENTS IN SPIROMETRY RATIOS IN PATIENTS WITH MODERATE-TO-SEVERE ASTHMA, WHICH CORRELATE WITH CHANGES IN FRACTIONAL EXHALED NITRIC OXIDE AND ASTHMA CONTROL

Research sponsored by Sanofi and Regeneron Pharmaceuticals Inc. ClinicalTrials.gov Identifier: NCT04400318. Medical writing/editorial assistance was provided by Jo Mooij, MSc, of

Excerpta Medica, and was funded by Sanofi and Regeneron Pharmaceuticals Inc., according to the Good Publication Practice guidelines.

## P198 TEZEPELUMAB REDUCES OCS USE AND IMPROVES SINO-NASAL SYMPTOMS IN OCS-DEPENDENT PATIENTS WITH SEVERE ASTHMA AND COMORBID CHRONIC RHINOSINUSITIS (OVERALL AND WITH NASAL POLYPS): RESULTS FROM THE PHASE 3B WAYFINDER STUDY

David J Jackson has received advisory board and speaker fees from AstraZeneca, GSK, Novartis, Sanofi and Teva Pharmaceuticals; and has received research grant support from AstraZeneca. Njira L Lugogo has received consultancy fees for participation in advisory boards from Amgen, AstraZeneca, Genentech, GSK, NIOX, Novartis, Regeneron Pharmaceuticals, Sanofi and Teva Pharmaceuticals; has received fees for non-speaker bureau presentations from AstraZeneca and GSK; has received speaker fees (own content) from NIOX; has received travel support from AstraZeneca; and her institution has received research support from Amgen, AstraZeneca, Avillion, Genentech, Gossamer Bio, GSK, Janssen, Regeneron Pharmaceuticals, Sanofi and Teva Pharmaceuticals. Mark Gurnell has attended advisory board meetings for AstraZeneca and has received speaker fees from AstraZeneca, HRA Pharma and Novartis. Liam G Heaney has received grant funding from, participated in advisory boards for and given lectures at meetings supported by Amgen, AstraZeneca, Boehringer Ingelheim, Circassia Pharmaceuticals, Evelo Biosciences, GSK, Novartis, Roche, Sanofi, Teva Pharmaceuticals and Theravance Biopharma; has received grants from Aerocrine AB, Amgen, AstraZeneca, Genentech, GSK, MedImmune, Novartis and Vitalograph; has received sponsorship for attending international scientific meetings from AstraZeneca, Boehringer Ingelheim, Chiesi, GSK and Napp Pharmaceuticals; has taken part in asthma clinical trials sponsored by Boehringer Ingelheim, GSK and Roche, for which his institution has been remunerated; and is the Academic Lead for the Medical Research Council Stratified Medicine UK Consortium in Severe Asthma, which involves industrial partnerships with several pharmaceutical companies, including Amgen, AstraZeneca, Boehringer Ingelheim, GSK, Janssen Pharmaceuticals and Roche. Stephanie Korn has received fees for lectures and/or advisory board meetings from AstraZeneca, GSK, Novartis, Roche, Sanofi Aventis and Teva Pharmaceuticals. Guy Brusselle has received fees for advisory boards and/or speaker fees from Amgen, AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, MSD, Novartis and Sanofi. Pascal Chanez has provided consultancy services for AstraZeneca, Boehringer Ingelheim, Chiesi, Johnson & Johnson, GSK, MSD, Novartis, Sanofi, SNCF and Teva Pharmaceuticals; has served on advisory boards for Almirall, AstraZeneca, Boehringer Ingelheim, Chiesi, Johnson & Johnson, GSK, Novartis, Sanofi and Teva Pharmaceuticals; has received lecture fees from ALK-Abelló, AstraZeneca, Boehringer Ingelheim, Boston Scientific, Centocor, Chiesi, GSK, Novartis and Teva Pharmaceuticals; has received industry-sponsored grants from AstraZeneca, ALK-Abelló, Boehringer Ingelheim, Boston Scientific, Centocor, Chiesi, GSK, Novartis, Roche and Teva Pharmaceuticals; and is the president of the scientific committee « Fondation du souffle » 2021–2025. Ricardo del Olmo has received research, advisory board and speaker fees from AstraZeneca, Boehringer Ingelheim, Gador, GSK, Mundipharma, Novartis and Sanofi Genzyme. Jean-Pierre Llanos is an employee of Amgen and owns stock in Amgen. Nanna Keeling, Javier Moreno Sapena, Bill Cook, Amit Parulekar, Neil Martin and Shradha Chandarana are employees of AstraZeneca and may own stock or stock options in AstraZeneca. This study was funded by AstraZeneca and Amgen, Inc. We thank Benjamin Emmanuel and Christopher S Ambrose, both of AstraZeneca, Gaithersburg, MD, USA, for their input towards the analysis design. Medical writing support was provided by Richard Claes, PhD, of PharmaGenesis London, London, UK, with funding from AstraZeneca and Amgen, Inc.

This abstract was previously presented at European Academy of Allergy and Clinical Immunology Annual Congress, 13–16 June 2025, Glasgow, UK.

## P199 MEPOLIZUMAB IMPROVES AND SUSTAINS ASTHMA CONTROL AND LUNG FUNCTION IN PATIENTS WITH SEVERE ASTHMA INDEPENDENT OF BASELINE BLOOD EOSINOPHIL COUNT IN THE REAL WORLD: RESULTS FROM REALITI-A AT 2 YEARS

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This is an encore abstract whose data was accepted and earlier presented at the European Academy of Allergy and Clinical Immunology (EAACI) Congress 2025; 13-16 June 2025; Glasgow, Scotland.

## P200 REAL WORLD EXPERIENCE; AN INTERIM ANALYSIS OF EFFECTIVENESS OF TEZEPELUMAB IN PATIENTS WITH SEVERE UNCONTROLLED ASTHMA ACROSS THE UK

This study was sponsored and funded by AstraZeneca, with medical writing support from Adelphi. COIs have been provided over email.

## P201 TWICE-YEARLY DEPEMOKIMAB EFFICACY IS SUSTAINED ACROSS SEASONS IN PATIENTS WITH ASTHMA: ANALYSES OF POOLED PHASE III SWIFT-1/2 STUDIES

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Previously presented at European Academy of Allergy and Clinical Immunology - 44th Annual Congress; Glasgow, United Kingdom; 13 - 16 June 2025

## P202 ON-TREATMENT CLINICAL REMISSION WITH TEZEPELUMAB IN OCS-DEPENDENT PATIENTS WITH SEVERE ASTHMA IN WAYFINDER

David J Jackson has received advisory board and speaker fees from AstraZeneca, GSK, Novartis, Sanofi and Teva Pharmaceuticals; and has received research grant support from AstraZeneca. Njira L Lugogo has received consultancy fees for participation in advisory boards from Amgen, AstraZeneca, Genentech, GSK, NIOX, Novartis, Regeneron Pharmaceuticals, Sanofi and Teva Pharmaceuticals; has received fees for non-speaker bureau presentations from AstraZeneca and GSK; has received speaker fees (own content) from NIOX; has received travel support from AstraZeneca; and her institution has received research support from Amgen, AstraZeneca, Avillion, Genentech, Gossamer Bio, GSK, Janssen, Regeneron Pharmaceuticals, Sanofi and Teva Pharmaceuticals. Mark Gurnell has attended advisory board meetings for AstraZeneca and has received speaker fees from AstraZeneca, HRA Pharma and Novartis. Liam G Heaney has received grant funding from, participated in advisory boards for and given lectures at meetings supported by Amgen, AstraZeneca, Boehringer Ingelheim, Circassia Pharmaceuticals, Evelo Biosciences, GSK, Novartis, Roche, Sanofi, Teva Pharmaceuticals and Theravance Biopharma; has received grants from Aerocrine AB, Amgen, AstraZeneca, Genentech, GSK, MedImmune, Novartis and Vitalograph; has received sponsorship for attending international scientific meetings from AstraZeneca, Boehringer Ingelheim, Chiesi, GSK and Napp Pharmaceuticals; has taken part in asthma clinical trials sponsored by Boehringer Ingelheim, GSK and Roche, for which his institution has been remunerated; and is the Academic Lead for the Medical Research Council Stratified Medicine UK Consortium in Severe Asthma, which involves industrial partnerships with several pharmaceutical companies, including Amgen, AstraZeneca, Boehringer Ingelheim, GSK, Janssen Pharmaceuticals and Roche. Stephanie Korn has received fees for lectures and/or advisory board meetings from AstraZeneca, GSK, Novartis, Roche, Sanofi Aventis and Teva Pharmaceuticals. Guy Brusselle has received fees for advisory boards and/or speaker fees from Amgen, AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, MSD, Novartis and Sanofi. Pascal Chanez has provided consultancy services for AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Johnson & Johnson, MSD, Novartis, Sanofi, SNCF and Teva Pharmaceuticals; has served on advisory boards for Almirall, AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Johnson & Johnson, Novartis, Sanofi and Teva Pharmaceuticals; has received lecture fees from ALK-Abelló, AstraZeneca, Boehringer Ingelheim, Boston Scientific, Centocor, Chiesi, GSK, Novartis and Teva Pharmaceuticals; has received industry-sponsored grants from AstraZeneca, ALK-Abelló, Boehringer Ingelheim, Boston Scientific, Centocor, Chiesi, GSK, Novartis, Roche and Teva Pharmaceuticals; and is the President of the scientific committee of La Fondation du souffle (2021–2025). Ricardo del Olmo has received research, advisory board and speaker fees from AstraZeneca, Boehringer Ingelheim, Gador, GSK, Mundipharma, Novartis and Sanofi Genzyme. Jean-Pierre Llanos is an employee of Amgen and owns stock in Amgen. Javier Moreno Sepena, Claudio Marchese, Bill Cook, Neil Martin, Shradha Chandarana and Amit Parulekar are employees of AstraZeneca and may own stock or stock options in AstraZeneca. This study was funded by AstraZeneca and Amgen, Inc. Medical writing support was provided by Joe Jones, PhD, of PharmaGenesis London, London, UK, with funding from AstraZeneca and Amgen, Inc.

This abstract will be presented at the European Respiratory Society (ERS) International Congress, 27 September–1 October 2025, Amsterdam, Netherlands.

## P204 SUPPRESSION OF FENO BUT NOT EOSINOPHILS WITH TEZEPELUMAB IS ASSOCIATED WITH IMPROVEMENTS IN SMALL AIRWAYS IN UNCONTROLLED SEVERE ASTHMA.

No conflicts regarding abstract. Previous speaking fees from AstraZeneca

## P207 PATH TO CONSENSUS: PATIENT AND PHYSICIANS PERSPECTIVES ON REDUCING BACKGROUND MEDICATION IN PATIENTS WITH SEVERE ASTHMA CONTROLLED ON BIOLOGIC THERAPY IN THE UK

Writing of abstract funded by AstraZeneca

P210 **THE IMPACT OF HETEROGENEITY AND FISSURE INTEGRITY ON THE TREATMENT RESPONSE OF AIRWAY SCAFFOLDS FOR EMPHYSEMA-RELATED HYPERINFLATION**

This abstract has been accepted as an oral presentation for ERS Congress 2025 in Amsterdam due to take place from 27 September 2025 to 01 October 2025.

P218 **HIGH FREQUENCY BRONCHOSCOPIC MICROWAVE ABLATION FOR PATIENTS WITH LUNG CANCER: INTERIM RESULTS FROM THE UNITED KINGDOM**

Creo Medical Ltd. was the sponsor for the clinical research trial from which the data for the abstract is derived. I have not received any funding from the sponsor.

This abstract has been accepted to ERS to be presented on 29th September 2025 in Amsterdam.

P220 **A SERVICE EVALUATION OF THE ENDOBRONCHIAL ULTRASOUND SERVICE (EBUS), INCLUDING MOLECULAR DIAGNOSES AND PATIENT SATISFACTION AT UNIVERSITY HOSPITAL SERVICE (UHS)**

This work was previously presented at the 2025 European Respiratory Society Congress.

P238 **SPATIAL TRANSCRIPTOMIC PROFILING AT SINGLE-CELL RESOLUTION REVEALS LUNG RESPONSES TO STERILE INJURY**

Funded by grants from the Wellcome Trust (104553/z/14/z, 211050/Z/18/z to T.S.C.H. and 222426/Z/21/Z to P.K.).

P240 **PULMONARY EXTRACELLULAR VESICLE MICRORNAS INDUCE ALVEOLAR MACROPHAGE DYSFUNCTION IN ARDS**

Medical Research Council

P243 **CHARACTERISATION OF SRC-FAMILY KINASE INHIBITORS IN LUNG FIBROBLASTS SHOWS DISTINCT SIGNALLING COMPLEXES REQUIRED FOR MATRIX SYNTHESIS AND DIFFERENTIATION**

Funded by a collaborative framework agreement between University College London (UCL) and Chiesi Farmaceutici SpA. The data produced/disclosed in this work/abstract confirm/reproduce what emerges from the references cited (any further reasoning or deduction will require additional confirmation).

This abstract will be presented at the ERS Congress in Amsterdam in September/October 2025.

P251 **INVESTIGATION OF MYELOID-DERIVED SUPPRESSOR CELLS AS A BIOMARKER FOR THE DIFFERENTIAL DIAGNOSIS OF LUNG DISEASES**

APB & AMM equally contributing first authors
NB & KMA equally contributing last authors

P261 **QUANTIFYING OXYGEN REQUIREMENT IN EARLY WARNING SCORES IMPROVES RECOGNITION OF DETERIORATION COMPARED TO THE NATIONAL EARLY WARNING SCORE 2 (NEWS2) IN PATIENTS ON OXYGEN.**

Competing interests: PW holds grants from the National Institute for Health Research (NIHR) and has held grants from Wellcome and Sensyne Health (now Arcturis Data). He is supported by the NIHR Biomedical Research Centre, Oxford. He was previously Chief Medical Officer for Sensyne Health (now Arcturis Data), holds shares in the company and provides consultancy. He sits on the NIHR HTA General Funding Committee. There are no other competing interests reported. Funding: This study has been delivered through the National Institute for Health and Care Research (NIHR) Oxford Biomedical Research Centre (BRC). The views expressed are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care.

P263 **ASSESSMENT OF DIAPHRAGM MOTOR UNIT ACTIVITY USING HIGH-DENSITY SURFACE ELECTROMYOGRAPHY**

This research study is funded by Asthma + Lung UK

P266 **TRANSCUTANEOUS ELECTRICAL STIMULATION OF THE UPPER AIRWAY DILATOR MUSCLES IN PATIENTS WITH MOTOR NEURONE DISEASE AND HEALTHY VOLUNTEERS.**

It will be presented at the ERS Congress 2025 in Amsterdam

P267 **ASSESSMENT OF THE IMPACT OF NEBULISER TYPE ON FUGITIVE EMISSIONS IN A TREATMENT ROOM WHEN DELIVERING INHALED MEDICATIONS**

Authors are employed by a medical device company.

P268 **EXTRA-FINE PARTICLE DEPOSITION OF TRIPLE INHALER THERAPIES USING THE NEXT GENERATOR IMPACTOR**

Enrico Zambelli, Alessandro Cavecchi, Laura Franzini and Alessio Piraino are employees of Chiesi Farmaceutici S.p.A. Brian Lipworth reports grants and support from Chiesi Farmaceutici S.p.A.

## P275 DO SELF-DRAINERS HAVE HIGHER INDWELLING PLEURAL CATHETER INFECTION RATES THAN NON-SELF-DRAINERS?

This work was presented at UK Pleural Society June 26th 2025.

## P283 PULMONARY HYPERTENSION ASSOCIATED WITH COVID-19-INTERSTITIAL LUNG DISEASE

Ferrer Pharmaceuticals have provided funding indirectly to support development of a Research Registry in patients with ILD-PH.

## P284 INTERIM RESULTS FROM PHINDER: PULMONARY HYPERTENSION SCREENING IN PATIENTS WITH INTERSTITIAL LUNG DISEASE

The PHINDER Study is sponsored by United Therapeutics Corporation, Research Triangle Park, NC, USA

Abstract is to be presented at ERS in October 2025.

## P286 IS THERE DIURNAL OR SEASONAL VARIATION IN THE 6-MINUTE WALK TEST DISTANCE IN PATIENTS WITH PULMONARY VASCULAR DISEASE?

JN Newman and BK Knox-Brown are joint last authors.

## P289 PATIENT AND CLINICIAN EXPERIENCES OF A FENO-GUIDED ASTHMA MANAGEMENT INTERVENTION IN PRIMARY CARE

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## P291 CLINICAL EFFECTIVENESS OF ANTI-INFLAMMATORY RELIEVER THERAPIES (AIR) FOR EXACERBATION PREVENTION: A NON-INFERIORITY STUDY

Richard Beasley has received institutional research funding from AstraZeneca and Teva, and personal fees from AstraZeneca and Teva. Ian D Pavord has received personal fees from AstraZeneca, GSK, Sanofi-Regeneron, Amgen, Areteia , Circassia. Michael Crooks has received grants from the National Institute for Health and Care Research, Asthma + Lung UK, AstraZeneca, Boehringer Ingelheim, Chiesi, Phillips, and Pfizer; honoraria, fees and/or non- financial support from AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Orion, Novartis, Pfizer, Synairgen and Sanofi. Brian Lipworth reports grants and personal fees from Chiesi, personal fees from Boerhingher Ingelheim, grants and personal fees from Meda, grants and personal fees from Teva, grants from Janssen, grants from AstraZeneca, grants from Roche, outside the submitted work. Peter Wark report no conflicts of interests. Rachel Malone and Arthur Jackson are employees of Chiesi Limited Victoria Carter, Adam Marsh, Neva Eleangovan and Freya Tyrer are employees of Observational and Pragmatic Research Institute, United Kingdom. David B. Price has advisory board membership with AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Novartis, Viatris, Teva Pharmaceuticals; consultancy agreements with AstraZeneca, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Novartis, Viatris, Teva Pharmaceuticals; grants and unrestricted funding for investigator-initiated studies (conducted through Observational and Pragmatic Research Institute Pte Ltd) from AstraZeneca, Chiesi, Viatris, Novartis, Regeneron Pharmaceuticals, Sanofi Genzyme, and UK National Health Service; payment for lectures/speaking engagements from AstraZeneca, Boehringer Ingelheim, Chiesi, Cipla, Inside Practice, GlaxoSmithKline, Medscape, Viatris, Novartis, Regeneron Pharmaceuticals and Sanofi Genzyme, Teva Pharmaceuticals; payment for travel/accommodation/meeting expenses from AstraZeneca, Boehringer Ingelheim, Novartis, Medscape, Teva Pharmaceuticals.; owns 74% of the social enterprise Optimum Patient Care Ltd (Australia and UK) and 92.61% of Observational and Pragmatic Research Institute Pte Ltd (Singapore); is peer reviewer for grant committees of the UK Efficacy and Mechanism Evaluation Programme, and Health Technology Assessment; and he was an expert witness for GlaxoSmithKline.

## P292 EFFECTIVENESS OF EXTRAFINE SINGLE-INHALER TRIPLE THERAPY ON ASTHMA CONTROL AND QUALITY OF LIFE ONE YEAR POST TREATMENT ACCORDING TO PATIENTS' SMOKING STATUS: THE TRIANGLE STUDY

This study was funded by Chiesi Hellas. P Katsaounou has received grants/research support from GSK and Viatris and honoraria/consultation fees from GSK, Pfizer, Kenvue, AstraZeneca, Chiesi, Win Medica. A Korkontzelou is a former employee of Chiesi Hellas and AstraZeneca Greece. E Papapostolou is an employee of Chiesi Hellas. P Katerelos reports financial support as statistical analyst of the present study. P Efstathopoulos is an employee of Chiesi Hellas. K Kostikas has received honoraria for presentations and/or consultancy fees from AstraZeneca, Berlin-Chemie, Boehringer Ingelheim, Chiesi, ELPEN, GSK, Guidotti, Menarini, Pfizer, Sanofi, and Specialty Therapeutics, his department has received funding and/or grants from AstraZeneca, Boehringer Ingelheim, Chiesi, ELPEN, GSK, Menarini, he worked with AstraZeneca as Global Medical Head Respiratory Biologics – 02.09.2024 to 29.11.2024, he is the Editor-in-Chief of Pneumon (pneumon.org), official journal of the Hellenic Thoracic Society and is a member of the GOLD Assembly

P293 **EXACERBATION AND RESCUE MEDICATION REDUCTION WITH EXTRAFINE FORMULATION SINGLE-INHALER TRIPLE THERAPY IN ADULT ASTHMA PATIENTS: OUTCOMES FROM THE TRIMAXIMIZE UK STUDY**

RR has received fees for conducting the study. JR and AR are employees of Chiesi Ltd.

P294 **THE BETRI STUDY: PRELIMINARY INFORMATION ON CHARACTERISTICS OF PATIENTS WITH ASTHMA OR ASTHMA-COPD OVERLAP TREATED WITH MEDIUM-STRENGTH INHALED CORTICOSTEROID / LONG-ACTING BETA-AGONISTS (ICS/LABA) AND SWITCHING TO HIGH-STRENGTH ICS/LABA OR MEDIUM-STRENGTH BECLOMETHASONE /FORMOTEROL /GLYCOPYRRONIUM (BDP/FF/G)**

This study was conducted by the Observational and Pragmatic Research Institute (OPRI) Pte Ltd and was funded by Chiesi Farmaceutici S.p.A.

This work was previously presented at ATS.

P295 **TREATMENT WITH EXTRAFINE FORMULATION SINGLE-INHALER TRIPLE THERAPY IMPROVES HEALTH-RELATED QUALITY OF LIFE AND ASTHMA CONTROL – RESULTS FROM THE TRIMAXIMIZE UK STUDY**

RR has received fees for conducting the study. JR and AR are employees of Chiesi Ltd.

P297 **BUDESONIDE/GLYCOPYRRONIUM/FORMOTEROL FUMARATE DIHYDRATE TRIPLE THERAPY FOR INADEQUATELY-CONTROLLED ASTHMA: KALOS AND LOGOS STUDY RESULTS**

Mehul Patel is an employee of AstraZeneca and may hold stock/stock options in the company. These studies were funded by AstraZeneca.

This is an encore presentation of an abstract submitted to the ERS 2025 congress, which will take place from 27 September to 1 October 2025.

P298 **TIME COURSE OF EXACERBATION REDUCTION WITH EXTRAFINE BECLOMETHASONE DIPROPIONATE, FORMOTEROL FUMARATE, AND GLYCOPYRRONIUM BROMIDE (BDP/FF/GB) PMDI IN ADULTS WITH MODERATE TO SEVERE ASTHMA: A POST-HOC ANALYSIS OF THE TRIMARAN AND TRIGGER STUDIES.**

Chiesi Ltd.

This work was previously presented at AAAAI.

P300 **EFFECT OF BASELINE ALVEOLAR GAS TRANSFER MEASUREMENT (KCO) ON RESPONSE TO BIOLOGICS IN SEVERE ASTHMA IN THE UK SEVERE ASTHMA REGISTRY**

No specific funding was received for this analysis. The lead author (PEP) has attended advisory boards for AstraZeneca, GlaxoSmithKline and Sanofi; has given lectures at meetings/webinars, with/without honoraria, supported by AstraZeneca, Chiesi and GlaxoSmithKline; has attended international conferences with AstraZeneca; has taken part in clinical trials sponsored by AstraZeneca, GlaxoSmithKline, Regeneron and Sanofi; is conducting research funded by GlaxoSmithKline for which his institution receives remuneration and quality improvement activity at his institution supported by AstraZeneca.

P302 **THE VIRTUAL PATIENT STRATIFIES PATIENT-SPECIFIC RESPONSE TO BRONCHIAL THERMOPLASTY WITH HIGH ACCURACY**

AirPROM 7th EU Framework (grant 270194), Royal Academy of Engineering (grant RF\201920\19\275), Dutch Lung Foundation (grant 5.2.13.064JO), the Netherlands Organization for Health Research and Development (grant 90713477), and Boston Scientific

P303 **REAL WORLD ASSESSMENT OF IMPACT OF DIGITAL HOME MONITORING IN DIFFICULT-TO-TREAT ASTHMA**

RW Costello is founder of Phyxiom

M3 **SUPPORTING 'SWAP TO STOP' SMOKING CESSATION: A PILOT TRIAL OF VAPE INFORMATION PROVISION WITHIN LUNG CANCER SCREENING**

University of Bristol Cancer Research Fund, the CRUK-funded Integrative Epidemiology Programme at the University of Bristol (C18281/A29019) and the Policy Support Fund at the University of Bristol.

M10 **EVALUATION OF PARTICIPANT EXPERIENCE IN THE NIHR HTA OPACE (OPTIMISING AZITHROMYCIN PREVENTION TREATMENT IN COPD TO REDUCE EXACERBATIONS) TRIAL - A PRAGMATIC TRIAL OF CONTINUED VS DISCONTINUED AZITHROMYCIN PROPHYLAXIS IN COPD.**

NIHR HTA award 134464

M11 **REAL-LIFE OUTCOMES OF PATIENTS WITH COPD INITIATING BUDESONIDE/GLYCOPYRRONIUM/ FORMOTEROL AFTER A PREVIOUS DUAL THERAPY IN SPAIN. ORESTES STUDY.**

The ORESTES study was funded by AstraZeneca.

M12 **REAL-WORLD DISEASE CONTROL AND STABILITY IN PATIENTS WITH COPD TREATED WITH EXTRAFINE TRIPLE THERAPY WITH BECLOMETASONE DIPROPIONATE, FORMOTEROL, AND GLYCOPYRRONIUM**

Luca Di Palma, Roberto Pizzoccheri, Ulrica Scaffidi Argentina, Laura Franzini and Alessio Piraino are employees of Chiesi Farmaceutici S.p.A. Guy Brusselle, Petros Bakakos, Daiana Stolz and Paola Rogliani report grants and support from Chiesi Farmaceutici S.p.A.

M13 **ANTIBIOTIC AND SYSTEMIC CORTICOSTEROID USE IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND TYPE 2 INFLAMMATION RECEIVING DUPILUMAB**

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M14 **ASSOCIATION BETWEEN EARLY LUNG FUNCTION IMPROVEMENT WITH DUPILUMAB AND SUSTAINED CLINICAL OUTCOMES IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CLINICAL PERSPECTIVE**

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M15 **DUPILUMAB EFFICACY ACROSS BASELINE EOSINOPHIL COUNTS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION**

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## M16 TREATMENT FAILURES DURING AN EOSINOPHILIC EXACERBATION FOLLOWING TREATMENT WITH BENRALIZUMAB OR PREDNISOLONE: DATA FROM ABRA

The ABRA study was funded by AstraZeneca as an unrestricted investigator initiated research grant to the senior investigator

## M17 SEX DIFFERENCES IN THE RELATIONSHIP BETWEEN SHIFT WORK STATUS AND PREVALENT ASTHMA IN THE OUR FUTURE HEALTH COHORT

Funding from North West Lung Centre Charity

## M19 REAL-LIFE EVALUATION OF FORCED EXPIRATORY RATIOS WITH ASTHMA EXACERBATIONS AND SYMPTOMS.

Jing Yuan On reports no conflicts of interest. Dania Ahmed reports no conflicts of interest. Fraser Paterson reports no conflicts of interest. Remo Poto reports personal fees (talks) from AstraZeneca and GSK. Atanu Bhattacharjee reports no conflicts of interest. Jake Brown reports no conflicts of interest. Frans De Jongh reports no conflicts of interest. Robert Greig reports personal fees (talks) from AstraZeneca. Tom Fardon reports honoraria, assistance with travel, conference fees and other funding from Novartis, Chiesi, GSK, AstraZeneca, Boehringer Ingelheim, Pfizer, Teva and Insmed; advisory boards in the previous three years for AstraZeneca and GSK; and is Chair of the Scotland Precision Asthma Group and sat on the UK Precision Asthma Group (AstraZeneca). Zuzama Diamant received speaker or consultant honoraria and/or served on advisory boards at: ALK, Aclaris, Arcede, Arrowheadpharma Pharmaceuticals, Airsonett, Biosion, Curovir, Foresee Pharmaceuticals, Galenushealth, GlaxoSmithKline, HippoDx, Pleuran, QPS-Netherlands, Sanofi-Genzyme-Regeneron, TPG Capital, all outside the submitted work. Travel grant received from ALK. During the last 3 years of her assignment as Research Director Respiratory and Allergy, QPS-Netherlands received an European grant from ERA4TB (IMI) and funding from Foresee Pharmaceuticals for early clinical studies. She serves/served as associate editor for Respiratory Medicine and Allergy and acted as Chair of the Asthma Expert Panel at EUFOREA (2020-2024). Brian Lipworth reports grants, personal fees (consulting, talks and advisory board), other support (attending ATS and ERS) from AstraZeneca; grants, personal fees (talks and consulting) from Sanofi/Regeneron, personal fees (consulting, talks and advisory board) from NIOX in relation to the submitted work; grants, personal fees (consulting, talks, advisory board), other support (attending ERS) from Teva, personal fees (talks and consulting), grants and other support (attending ERS and BTS) from Chiesi, personal fees (consulting and talks) from Lupin, personal fees (consulting) from Glenmark, personal fees (consulting, talks, advisory board), other support (attending BTS) from Boehringer Ingelheim; and the son of BJL is presently an employee of AstraZeneca. Rory Chan reports institutional grants from Chiesi, AstraZeneca and GlaxoSmithKline for setting up and chairing the Scottish Airways Research Network; serving on an advisory board for AstraZeneca; personal fees (talks and drafting educational material) from AstraZeneca, personal fees (talks) from Chiesi, personal fees (talks) from Thorasys and personal fees (drafting educational material) from Vitalograph; support attending meetings from AstraZeneca, Chiesi, NIOX, Sanofi-Regeneron and Vitalograph.

## M21 IMPACT OF THE SENTINEL PROGRAMME ON ASTHMA CARE ACROSS SIX PRIMARY CARE NETWORKS IN ENGLAND.

Analysis sponsored and funded by AstraZeneca UK

## M22 KEY INDICATORS OF CARE FOR PATIENTS HOSPITALISED WITH ASTHMA AND COPD WITH COMORBID MENTAL HEALTH OR COGNITIVE IMPAIRMENT: PRELIMINARY FINDINGS FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME (NRAP).

This work was supported by the National Respiratory Audit Programme (NRAP) which is hosted by the Royal College of Physicians (RCP) and commissioned by the Healthcare Quality Improvement Partnership (HQIP) as part of the National Clinical Audit Patient Outcomes Programme (NCAPOP).

M24 **THE PREVALENCE OF PSYCHOLOGICAL SYMPTOMS IN SEVERE AND DIFFICULT ASTHMA**

This abstract will be presented at the 2025 ERS Congress.

M27 **A PAIN IN THE NECK? HEALTHY VOLUNTEER ACCEPTABILITY OF TRANS LARYNGEAL ULTRSASOUND TO ASSESS VOCAL FOLD MOVEMENT IN MIMICKED INDUCIBLE LARYNGEAL OBSTRUCTION**

NIHR funding for research time

M31 **STRINGENT AMERICAN (ATS/ACAAI/AAAAI) ENDORSED 6-COMPONENT DEFINITION OF ASTHMA REMISSION IS NOT ACHIEVABLE IN A REAL-WORLD BENRALIZUMAB COHORT**

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This abstract will be presented as a poster at the 2025 ERS Congress.

M34 **SEMI-AUTOMATED QUANTIFICATION OF PET-CT IN TB CONTACTS AND ITS ASSOCIATION WITH PROSPECTIVE OUTCOMES**

This study was funded by an MRC Confidence in Concept award

This abstract is to be presented at ERS Congress 2025 in September 2025

M36 **PERIPHERAL BLOOD INFLAMMATORY AND IMMUNE BIOMARKERS AS CORRELATES OF TB INFECTION PHENOTYPES CHARACTERISED BY PET-CT: A PROSPECTIVE COHORT STUDY IN HOUSEHOLD CONTACTS**

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*The number next to the author indicates the abstract number, not the page number.*


Aasvang EK, P262
Abbas H, P285
Abdo M, P4
Abed El-Khaleq Y, P254
Abernethy K, S78, P227, P235
Abkir M, S70
Abrams S, S5
Abugassa E, P60, P92, P171
Abujamel T, P192
Acosta-Mena D, S41
Adamali HI, S13, S15
Adams W, P254
Adamson A, S44, S138, P68, M22
Adamson AJ, S7
Aday C, P60
Adcock I, T4
Addala D, P81, P269
Addala DN, S36, P77
Addy C, S53
Adegbenro E, S5
Adejumo I, P64
Adesoye G, M10
Adhikari I, P28
Adil Irshad M, P29
Adlakha S, P55
Adriaenssens EM, P187
Afzal B, P231
Aganin I, P279
Agaoglu E, P214
Agaoglu ES, P213, P216, P218
Agarwal A, P94
Agnew S, P25
Agrawal S, P82
Agusti A, S1
Ahmad HM, S73
Ahmad I, P256
Ahmad N, S173, P37
Ahmad T, P170
Ahmed DA, M19
Ahmed M, P234
Ahmed MI, S68, P133
Ahmed S, P188
Ahmed W, S33, S162
Ahn-Jarvis J, P187
Ainsworth B, P289
Ajayi C, P205
Akin C, P158
Akram MJ, P234, P236
Akwa LG, P126
Al Arfi K, P227, P235
Al-Ameri M, P2
Al-Harbawi B, P92
Al-saadi M, P177
Alabed S, S82, P281, P285
Alajmi R, P122
Alasfour A, S124
Alaws H, P158
Alcada J, S154
Alcázar B, S102
Alcázar-Navarrete B, M11
Alex A, P246
Alexander H, P215
Alexander L, S66, P163
Alfonso-Cristancho R, S164, P199
Ali A, S73, S98, P177, P233
Ali F, P171
Ali S, S116
Aliberti S, T6
Alkhathami M, S77, P266
Alkhdour A, P28
Allahyar A, S161
Allan L, P163
Allen A, P178
Allen AP, P182
Allen RJ, S88
Allinson JP, S99, P162, P253, P257
Allsopp LP, P183
Allsopp R, S34
Almaghrabi A, S77, P266
Almahmoudi JA, S128
Almeida H, S45
Almeida M, P6
Almeshari M, M20
Almond H, P98
Alnasser TN, P281
Alsahab M, P279
Alshahrani MI, M20
Alsharifi A, S77, S95
Alshehri A, M20
Alsulayyim A, P30
Altincatal A, P197
Alvarez-Nishio A, S5
Alves Moreira L, S99
Alzahrani A, P30
Alzaid M, P209
Alzetani A, P165
Amaral A, S113, M37
Amcha K, P28
Amin A, P160
Anagnostopoulou C, P264, P265
Anand TS, M42
Ananth S, P79, P257
Anderson D, S2, P148, M10
Andrews C, S62
Angwin W, P132
Anigboro K, P12
Aniscenko J, T2
Annaw A, S6
Annema JT, P302
Anpalagan H, S130
Antoniou KM, P251, P252
Antonogiannaki EM, P212
Antoun E, S147
Anwar M, P188, M10
Anwar S, S154
Anzueto A, M13
Apostolidou A, P251
Arachchillage DJ, S165
Ardesi F, S54
Arefi M, M35
Arif KA, S78
Armstrong-James D, S124, S163
Arnold D, P184
Arnold DT, S35, S123
Arora J, S109
Arries R, S3
Arron J, S105
Ashe H, P87
Ashfaq M, S16
Ashley P, S172, P159
Ashraf A, P163
Aslam H, P226
Aslam Z, P62
Asqalan A, P211
Assaduzzaman M, S75
Astell H, M25
Astin R, P255
Aston S, S5, P188
Atefeh K, S32
Athanasiou N, M8
Athanasopoulou V, P161, P212
Atherton P, P175
Atkinson J, M3
Attwood A, M3
Aujayeb A, S150, P78, P163, P222
Aul R, S2, P254
Aung H, S172, P4, P159
Aung HWW, S176, P13
Austin M, S74
Aveyard N, P301
Avoseh MD, S153
Aw TC, S154
Awad H, P197
Awan ZA, P141
Axmann J, P150
Ayis S, P33
Ayling-Smith J, P188, S53
Azarce K, P131
Azeez NF, P119
Azevedo P, S68, P133
Azouaghe MO, M48

Babikir E, P303
Babu S, P29
Bacchini Jeanneret G, S81
Badri H, S116
Bafadhel M, T3, S9, S12, S100, S122, S175, P4, P8, P10, P155, P157, M13, M16
Baguneid A, S34, P275, P278
Bahra A, S159, M25
Bailey M, S4
Bailey P, P188
Baird DM, P98
Bairwa J, P94
Bajwah S, P16
Bakakos P, P264, P265, M12
Baker D, S156
Baker J, S59, S100, S104, S122, P155
Baker JR, T3
Baker L, P188
Bakerly ND, P262
Balasubramanian S, P125
Balasundaram K, S172, P159
Balata H, P209, P215
Baldock D, P128
Baldwin D, S31
Baldwin M, S140
Ball M, P29
Ballance K, S116
Bamashmous S, P192
Band M, T6
Bandara H, T6
Bandi S, M43
Banerjee A, P222
Bange RK, P234
Banks J, S6
Banner K, M42
Bansal K, S51, P184
Banya W, S11, P20
Barathan S, S114
Barbaressou Z, M8

# Author index


Barber CM, S110
Barber E, P64
Barbonchielli D, P102
Barker B, P205, P206
Barker N, P146
Barksby B, S86
Barnes A, P98
Barnes PJ, S120, S121
Barnett J, P25, P167
Barningham TJ, P130
Baron R, P9
Barr K, M35
Barrable F, S162
Barraclough R, S116
Barradell A, P31
Barratt SL, S13, S15, P254
Barrett G, P132, M10
Barth SA, P254
Bartlett-Pestell S, S99, P162
Bartlett-Pestell SWJ, S60
Barton T, S31
Bartziokas K, P161
Basit Kayani A, S98
Basra R, P63
Bateman NA, P31
Bates J, P47
Batool A, S63
Battenfield ML, S153
Bauer D, S174
Bawuagh A, M7
Bax S, P20, P22, P283
Baxter J, S36
Baxter S, P259
Bayes H, S2
Baynes G, S159
Bazaz R, S5
Bean O, S52
Beasley R, S52, P291
Beasy G, P187
Beaton M, P136
Beattie A, S32
Beaumont-Kellner H, P66
Bech M, S57
Bechtold D, P245
Beckett P, P172
Bedawi E, S36, P269
Bedford JP, S30, P261
Bedi P, S2
Beech A, S136, P154, P257
Beersaerts A, P4
Begley N, P245
Beirne P, P27, P88
Bell AJ, S37
Bell D, M43
Bellamy V, S8
Belloli E, P284
Belton M, M41
Belvisi MG, T6, S37
Benger JR, S6
Bennett J, P82
Bennett M, S46, S47, S49, P260, M23
Bensabai R, S64
Benson J, P95
Bentley A, S97, P41, P223, P225
Benza RL, S146
Berair R, P205, P206
Beranova E, M10
Bermejo-Sanchez L, S175, M16
Bernardino de la Serna JBS, M1
Berrar-Torre I, P131
Bessell E, S109
Best C, S74
Bevan A, S114
Beverstock J, P46
Bewick T, P181
Beyatli S, S109
Bhalwel I, P21
Bhamani A, S67, P170
Bhamji NB, S80
Bharkhada A, P65
Bhat J, S103
Bhatnagar M, P81, P269
Bhatnagar R, S150
Bhatt N, S35
Bhatt SP, S101, M13
Bhattacharjee AB, M19
Bhatti K, P242
Bhutra P, P86
Bibby A, P163, P168, M3
Bicknell S, S105
Bidari K, P181
Bie EMDDDe, S143, S146
Bikmalla S, S111
Bikov A, S97, P41, P223, P225
Bilancia R, P163
Bingham Y, S45
Binmahfooz SK, S143
Birch R, P76
Birchall J, P172
Bird M, P16
Bird N, S135
Birring S, P115, P116, P117
Birring SS, P103, P104, P111, P113
Bishop S, S15
Bizymi N, P251, P252
Bizymis AP, P251, P252
Blacker K, P76
Bladon H, S159
Blake A, S62
Bloom B, P67
Bloom C, S130
Blowers E, M2
Blume C, T5, S108
Blyth K, S32, S35, S149, P163
Blyth KG, S66
Blythe N, S6
Boast LA, P15
Boddy C, S54
Bogaard HJ, S146
Bokhari S, S78, P227, P235
Boland A, P16, P27, P88
Bolton C, M10
Bolton CE, S28, P185, P257
Bondoc M, P297
Bongers T, P52
Bonini M, S135
Bonner K, P138
Bonta P, P302
Boorsma R, P18, P102
Boreland S, P22, P23
Borthwick L, S86
Borton R, P254
Bothwell H, S111
Bou-Zeid W, S13
Boucly A, S143
Bourdin A, P201
Bourke S, S3, S93
Bourke SC, P124, P153
Bourne M, S172, S176, P13, P159
Bowden AR, P96
Bowen K, P297
Bowers R, P119
Boyd J, S127
Boyle A, S169
Boyle N, S63
Bracchi M, P188
Bradding P, S54, S89, S105
Braddy-Green A, S60, S99, P162, P253, P257
Bradford M, S156
Bradley ER, P9
Bradley J, S39, S42, P27, P88
Bradley JM, S4, P38
Bradshaw E, S124
Brady J, P273
Brailsford W, T6, S37
Brame A, S154
Bramley K, S154
Branchett W, M36
Brassanyi B, S106
Breen R, P188
Bremner P, S1
Brennan A, S20, P57
Brennan V, S50, P193
Brewin K, S3, P124
Bridges E, S106, P238
Brightling CE, S1, S54, S56, S133, S172, S176, P4, P13, P159, P191, P302, M31
Brignall K, P18
Brij SO, P9
Brill SE, P300
British Early COPD Network (BEACON) Study, P257
Broad M, M28
Broderick J, P40
Broderick M, P284
Brooks S, P25
Brown A, S147
Brown H, P160
Brown J, S39, S42, S109, S133, P188, M31, M39
Brown JB, M19
Brown JP, M41
Brown M, M41
Brown R, S21, S22
Brown S, P135
Brown SA, P290
Brown T, S133, P75, P300, M31
Brown TP, S134, P203
Brown V, S105
Browne P, P211
Bruselle G, S132, P294
Brusselle G, P198, P202, M12
Bryan N, S75
Bucataru A-M, P131
Bucataru AM, S163
Buchanan H, S37
Buckley E, P168, M3
Buhl R, S174
Bunce R, P188
Bunclark KB, P286
Burge G, S111
Burge PS, S111
Burgel P-R, S38
Burgel PR, P4
Burgess A, P39
Burgoyne RA, S86
Burhan H, S133, P75, P200, P207, M30, M31
Burke H, S7
Burke L, P87
Burman M, P188
Burn P, P218
Burns S, P148

Burrell T, P296
Burrough J, P102
Burton R, M30
Busby J, S129, P75, P300
Bush A, S45, P138
Bussell E, S15
Butcher S, M5
Butler C, P62
Butler J, P109, P114
Butler JM, P110
Butt H, P299
Buxton M, P34, P35
Byrne AJ, S63
Byrne L, S110, S115
Byrne N, M18

Cain J, P245
Callet A, S41
Callister M, S31, P169
Callister MEJ, P126, M5
Callow B, P80
Calverley PM, P257
Cameron S, M25
Caneja C, P44
Cannon JC, P286
Cano-Gamez K, S147
Canonica GW, S132, S164, P199
Capocci S, P188, M41
Cappellini G, P6
Capstick T, S126, P27, P88
Capstick TGD, P119
Carballido-Romero B, P17
Carder M, S110, S115
Cardner M, S161
Carlin C, P3, P148, P151
Carlisle V, M3
Carlton E, S6, S152
Carr S, S4
Carroll D, P3
Carroll M, S39, S42
Carter R, M45
Carter S, S72
Carter V, S102, P291, P294
Cartledge J, P67
Cartwright J, S161
Cartwright K, T6, S37
Caruso P, S145
Cass S, S100, S104, S175, P155, M16
Cass SP, T3, P4
Cassella J, P113
Castellanos-Uribe M, S86
Castelli G, P20
Castro M, P197, P302
Cattini A, S4
Cavecchi A, P268
Cefaloni F, P195
Celli B, S1
Cenerini G, S25
Cerrone E, S82, P285
Chai A, P41, P225
Chakir J, P302
Chakrabarti B, S24
Chakrabati B, P223
Chakraborty A, P245, P246
Chakraverty S, S29, S141
Chalitsios CV, P64
Chalmers J, S39, S42, P168, P184
Chalmers JD, T6, S37, S38, P178, P182
Chambers R, P100
Chambers RC, P243
Chan J, S116, S146
Chan LT, P213, P214, P216, P218
Chan ML, P52
Chan R, S131, P200, P204
Chan RC, M19
Chandak V, P177
Chandarana S, P198, P202
Chandra R, P142
Chandrasekharan S, P64
Chanez P, S135, P198, P202, P302
Chang E, P247, P249
Chaplin E, P31
Chapman KR, P113
Charalampopoulos A, S82, P285
Charters P, P282
Charters PFP, T1
Chatfield K, S79
Chaudhary N, P139
Chaudhry A, P274
Chaudhry ZJ, S73
Chaudhuri E, S152
Chaudhuri R, S105, S133, S164, P75, P199, P302, M31
Chauhan A, S33
Chauhan AJ, S134, P203
Chavali R, P227
Chawla M, P119
Chawla R, P5
Cheadle M, S116
Chen C, T2, S126, S167, P121
Chen H, P8, P156
Chen N, P18
Chen R, P297, M14
Cheng L, P238
Cheng M, P8
Cheng Y, P172
Chetty M, P163
Cheung DS, S1
Cheung M, P233
Chew WM, P269
Chi S, P247, P248
Chien N, P102
Chishimba L, M30
Cho P, P111
Cho PSP, P103, P104
Chong B, S99
Choo-Kang B, P188
Chotirmall S, S58
Choudhury G, P257
Chown H, P192
Choy D, S105
Choy J, P287
Christenson SA, M15
Christie JJ, P210
Christopher D, P5
Chu J, S147
Chu JY, S148, S170
Chua F, S18, S154, P20, P22
Church C, S146
Chynkiamis N, P264, P265
Cicognini F, P74
Cipolli M, S68, P133
Clark J, P282
Clark KEN, P24
Clark TW, P178, P182
Clarke A, S2
Clarke C, S37
Clarke L, P43
Clarke TB, T2
Clarkson F, S26
Clarkson-Sheahan N, M5
Clay H, P223
Clement C, S6
Clewes C, P200
Clifford R, S86, S88, S89
Clifford RL, S107
Clifton I, P74, P75
Clifton IJ, S159, P119
Clive G, P188
Clutterbuck L, P267
Coates K, S6
Coats T, S156
Cobham V, S51, M28
Coelho S, S111
Coghlan JG, S146
Cohen J, M21
Çolak Y, S174
Coleman M, P188, M48
Coll R, S169
Collins A, S5
Condliffe R, P285
Condliffe RA, S82
Conlon J, S4
Connell D, S124
Connett GJ, P142
Connolly B, P38
Connor TMO, P40
Connors J, S152
Conroy K, S150, P163
Contoli M, P4
Cook B, P198, P202
Cook H, S152
Cook K, S5
Cookson A, P282
Cookson WO, P247, P248, P249, P250
Cooney MT, S72
Cooper CB, S26
Corcoran J, S150, P80, P219
Corish C, S72
Cornwall O, P79
Corradi M, P6
Correa-Jaque P, S146
Corregidor C, M11
Coss P, S20, P57
Costello RW, S50, P69, P193, P303, M23
Cottini M, S131
Couillard S, S174
Cowburn A, P239
Cowburn AS, S144, S165
Cowell G, S32
Cowie S, P188
Cowley A, P188
Cowman S, P188
Crabtree R, S8, S10
Craig C, S150, P163, P215
Craig S, S24
Craven AL, P47
Crawshaw A, S111, P16
Creamer A, S67
Creanor S, S152
Creber PW, P70
Creer D, M41
Creese J, P31
Croft E, P41, P225
Crook C, S17, S156, P17, P98
Crookes E, S92
Crooks M, S2, P291, P296
Crooks MG, S159, P61, P178, P182, P194, M21
Crooks R, P163
Crosbie P, S62, P169

Crosbie PAJ, P126
Cross A, S5
Crowe J, S2
Crowley L, S90
Crowley LE, S167, S168, P16
Croxford P, S13, S15
Cruz-Utrilla A, S142
Csoma B, P41
Cucato G, S29, S141
Cuddihy M, S171
Cullen R, S52
Cumella A, P7, P122
Cummings H, S159, P61, P194, P207, M21
Cunnington M, P8
Cushen B, S50, S171
Cushing A, P148
Cuthbert K, P259
Cuthbertson I, S145

d'Ancona G, P7, P195, M24, M29
D'Cruz R, S78, P227
Daccò V, S68, P133
Dadej M, P46
Dahabreh M, P135
Dahlen S-E, T4
Daigneault P, P133
Dakin R, S37
Daley CL, S38
Dallaway A, S23
Dalton J, S154
Daly E, S36
Daly L, P249, P250
Daly R, P72
Daly-Gourdialsing A, P183
Danby R, P154
Daneshvar C, S36, P163, P176, P178, P182, P219
Danzo F, P20, P23
Darwish M, P178, P182
Daryanani R, P36
Das I, M38
Das V, P5
Davies E, P50, P63
Davies G, M10
Davies H, P173
Davies J, S32
Davies JC, S69, S70, P183
Davies M, S93, P50
Davies R, P17
Davies S, S159
Dawson S, M25
Daynes E, S29, S137, S139, S140, S141, P31, P32, P34, P35
DCruz L, S33
DCruz R, P235
De A, P64
De Baets G, S161
de Batisda-Casero L, S142
de Bie E, P50
De Bolfo J, P147
de Braal VJF, P24
De Corso E, S160
de Fonseka D, S35, S36
de Lima Headley D Alferes, S37
De Looper A, M24
De Oliveira J, M3
De Ridder T, P6
De Sousa P, M6
De Souza T, S116
De Soyza A, S2, S38, S39, S40, S42, S125
De Soyza J, S103, S119
De Vos M, S11
de-Soyza A, M10
Dean C, P242
Dean-Jalloh A, M29
Deas C, S124
Decout A, T2
Dede K, M8
Dedicoat M, M42
DeFoneska D, P163
Deka H, S59
del Olmo R, P198, P202
del Rosario A, S104
Deliu N, S143
Dennison P, T5, S108
Denniston P, P78
Denton CP, P23
Dervisevic S, P180
Desai S, P22, P95
Desbordes P, S155
Deslée G, S174, M13, M14
Deugi V, P245, P246
Deus P, P174
Dev Vellore A, S2
Devani N, P46
Devaraj A, S13, S112, P22, P283
Devenny A, P136
Devulder J, S60
Devulder JV, S120, S121
Dexter JP, M4
Dexter K, P137
Dey P, P227
Dhadda S, P228
Dhar R, P5
Dhara A, P67
Dhariwal J, S104, P195, P200, M24, M29
Dhary Y, P222, P274
Dhasmana D, P188
Dhasmana DJ, P129
Dhillon R, T5
Dhiman P, S31
Dhooria S, P94
Dhrampal A, P211
Dhungana A, S23
Di Palma L, P294, M12
Diable E, S71
Diamant Z, M19
Diar-Bakerly L, S62
Diar-Bakerly N, S33, S62
Dickens JA, P96
Dickson J, S67
Dicpinigaitis P, P115, P116, P117
DiGiandomenico A, T6, S37
Dimopoulos K, P283
Diver S, S54
Dixon G, S13, S15
Djukanovic R, M31
Djukanovich R, S133
Do QN, S68, P133
Dobra R, S48
Dobson P, S100, P155
Dockry R, P106
Doctor SS, P92
Dodd JW, S7, S44, S51, S117, P68, P70, P300, M22, M28, M32
Dods S, P8, P156
Doe G, S11, P123
Doe S, P75, P300
Doffman S, S67
Doherty H, S50, P193, P303
Doherty M, P75, P205, P206, P300
Doherty R, P21
Domi V, S18
Dominic C, P180, P187
Donaldson A, P29
Donaldson S, S2
Dong T, S65, S147, S148, S170
Donga P, P128
Donnelly L, S60
Donnelly LE, S120, S121
Dorn J, P150
Dorrian D, S4, S169
Dosanjh D, S2
Dounavi A, P212
Dowding R, P38
Downes A, S71
Doyle J, S154
Drake S, S49
Drazich-Taylor S, P180
Drennan D, P105, P107
Drennan DP, P112
Drew M, P205, P206
Drover H, S137, S138, S139, P31, P32
Drummond I, T5
Du H, P249, P250
Du Q, S37
Du Rand I, P224
Duckers J, S39, S42, S68, P133
Duckworth A, S17, S156, P17, P98
Duffin T, S111
Duffy SM, S89
Duffy SW, P164
Duignan N, M18
Duncan A, P20
Duncan E, P143
Dunlop A, M46
Dunlop J, P174
Dunmore B, S145
Dunmore BJ, S143
Dunning J, P255
Dunning M, M7
Dupont L, P116, P117
Durie A, S111
Durkan H, M48
Durmus Y, P74
Durrington H, S49, P245, P246
Durrington HJ, P299, M17
Dushianthan A, S4
Duvall K, S63
Dwarakanath A, P49, P226
Dwivedi K, S82, P281, P285
Dwyer R, P69
Dyson J, P61

Eaden J, P254
Early F, M10
Echevarria C, S29, S141
Edefo J, M7
Edey A, P168
Edgecomb A, S162
Edward D, P279
Edwards A, S6
Edwards C, P188, P254
Edwards G, S11
Edwards SA, S172, P159
Ee W, S100, P155
Efstathiou C, S56
Efstathopoulos P, P292
Egan S, P255
Ehsan N, S84
Eke Z, T6, S37

Ekezie W, P64
Ekkelenkamp M, S127, P186
El Jadid S, S118
El-Emir E, S11
El-Nayal A, P60
Elakhdar Y, P171
Elborn S, S39, S42
Eleangovan N, P291
Elek CKA, P187
Elliot CA, S82, P285
Elliott MW, P49
Ellis P, P234
Elmahy A, S11
Elneima O, S176, P13
Elsheikh A, S36, S147, S148, S170, P81, P269
Elston P, P121
Elton-Walters L, P28
Elworthy V, S156, P17
Emanuel P, S37
Eminton A, T5, S108
Emms H, S13, S15, P98, P301
Eng C, P232
Enriquez R, P235
Erpenbeck V, P150
Erwin P, S118
Evans H, S172, P159
Evans R, S29, S137, S139, S141, P32, P184
Evans RA, S11, S56, S172, S176, P13, P123, P159, P191

Fabri L, P102
Facer C, M5
Fahim A, S111, P53, P102, P254
Fallon J, P163
Fama F, M41
Fan C, S38
Farahi N, S166
Fardon T, P200, P204, P207, M19
Farrell R, P273
Farrell S, M25
Farrelly L, S67
Faruqi S, S133, S159, P75, P194, P300, M21, M31
Fasham J, S156
Fastenau J, P128
Fearby S, P109
Fearns A, M33
Feary J, S112, S113, S114, P73, M37, M46
Feghali-Bostwick C, S89
Felgate H, S149
Feliciano J, S41
Felton I, S71
Feng Y, S147, S148, S170
Fennel D, S32
Fennell D, S34
Fenwick P, S60
Fenwick PS, S120, S121
Ferey B, M14
Ferguson K, P163
Fernandes M, P195, M24, M29
Fernandez C, S38
Fernández Delgado M, S81
Ferreira L, P20
Fewings C, S159
Fiassam O, S140
Figa K, P222
Figueira-Gonçalves JM, M11
Filby K, S79
Finch S, S2
Finlay C, P270
Finn E, P222, P274
Finney LJ, S60, S99, P162
Fisher A, S86
Fisher M, P279
Fishwick D, S110
Fisk A, S27
Fisk M, M10
Fitas E, M11
Fitzgerald A, P167
Fleming L, S45, S48, P138
Fleming LF, P141
Fletcher SV, S157, P28
Flower L, S166
Floyd SV, P111
Flynn C, P4
Flynn CA, S176, P13
Foden J, P279
Fogarty A, P125
Forde SH, S63
Forrest A, P230
Forster JL, P134
Forster MD, P170
Fowler S, S47, S105, P72
Fowler SJ, S46, S49, S133, P260, M23, M31
Fowles A, S138
Fox-Rushby J, P33
Franciosi A, P1
Francis A, P7
Frangulyan R, S2
Frankl MR, P56
Franssen FM, P4
Franssen FME, M14
Franzini L, S132, P268, P298, M12
Fraser V, P181
Frater A, P166
Freeman A, P11, P178, P179, P182
Freeman D, S97
Freydina D, S108
Frost F, P186, M10
Frost J, P168, M3
Fuchs T, S69
Fuld J, P76, M10
Fuld JP, S26
Funes D, S81
Fyles F, P25, P223, M30
Fynney A, P118

Gabe R, P126, P169
Gadoud A, S14
Gadsby J, S126
Gadwal V, P233
Gaik WT, P150
Gajanan A, S82
Gale CP, P156
Gall R, P197
Gallagher C, S72
Gallant K, P8
Gallego-Moll C, S42
Galloway R, T6, S37
Gamboa F, S68
Ganaie MB, P171
Ganesan D, P87
Gangi S, S87
García García M, P258
Garcia Mingo A, M41
Garden EJ, P59
Gardiner N, S140
Garfield B, S154, P283
Garin M, P113
Garner JL, P210, P213, P214, P216, P218
Gaston B, P246
Gates J, S104, P195, M24, M29
Gatheral T, S14, S39, S42, P163
Gaunt D, S6
Gaur A, S84, S146
Gavin O, S20, P57
Gawlik-Lipinski A, S43, S44, P144
Gay N, P254
Gayle A, P294
Gbajie NP, P188
Geddes L, S94
Gennatas S, S64
George P, S154, P20, P22, P30, P283
George PM, S13, P192
Georgiadis T, M8
Gerrard G, S64
Gerry S, S30, P261
Gervasutti A, P160
Ghani H, S83, S85
Ghauri M, P233
Ghio S, S146
Ghofrani HA, S146
Ghorpade P, S152
Ghosh D, S3, S19, S93, P49, P58, P232
Ghout I, S41
Gibbons L, S2
Gibbons M, S2, P254
Gibbons MA, S13, S15, S17, S156, P17, P98
Gibbs J, P245, P246
Giffen E, P3
Gilbert HR, S176
Gilbey S, P205, P206
Gill A, S32
Gill C, P69, P193, M23
Gill CM, S50, P303
Gill JJ, P152
Gilles S, P183
Gillespie L, S133, M31
Gillies CL, S43
Gilmour A, T6
Gilmour-Caunt A, M38
Gilpen R, P241
Gilworth G, P33, P36
Girardello L, P6
Gleeson F, S32
Gleeson FV, S31
Gleeson L, P270, P273
Glenn M, S4, S118
Godfrey E, P33, P36
Goeminne P, S127
Goh K, P189
Goh KY, M5
Goh ZM, S82, S146, P285
Gokal D, P290
Gold S, P215
Goldman NR, P23
Goldring J, S150, M41
Gonem S, P75
González J, M11
González-Barcala FJ, P197
Goodman A, S125
Goodrum SE, S17
Goodwin C, S152
Gor D, S41
Gordon D, S37
Gore M, S45, P138
Gore R, P75, P196
Gorman P, S16
Gorsuch T, P188
Gotera C, M11
Gothard P, M41

Gould S, S164, P199
Gourlay J, P163
Govender R, M27
Govoni M, P257
Gowing F, S76, S94
Gracie CJ, P24
Gracie K, M5
Gräf S, S143, S146
Graham LM, P45
Grainge C, P294
Gramegna A, P186
Grant A, P44
Graves J, P173
Gray J, S3, P124
Gray S, P140
Graziadio S, P89, P90
Greco M, P238
Green B, P188
Green CG, P192
Green E, P109
Green K, P149
Green L, P195, M24, M29
Green M, P89, P90
Green RH, S54
Greene C, S118
Greene G, S50, P193, P303
Greening N, S1
Greening NJ, S172, S176, P4, P13, P159, P178, P182
Greig R, P204, M19
Grenville J, S51, S117, M28, M32
Grenville JS, P70
Gribbon M, S154
Griffin O, S72
Griffiths A, P163
Grimbaldeston MA, S1
Grolmusova N, M35, M38, M43
Grundy S, S33, S62
Guck J, S165
Gudur S, P84
Guignabert C, S143
Gulati A, P53, P228, P229
Gunarwardena H, S15
Gunning SGS, T1
Guo H, P249, P250
Guo Y, S60
Guo-Parke H, S118
Gupta A, S28
Gupta BK, P139
Gupta M, P94
Gupta N, P94
Gupta RK, M41
Gupta SN, P5
Gurnell M, P198, P202
Gutierrez MG, M33
Gutierrez PV, P238
Guvensen G, P170
Gyertson K, S67
Gyselinck I, P4

Hacker A, S67
Hackett T, S86
Hackshaw A, S67
Hadda V, P94
Hagan G, S111
Haider R, M45
Hakim A, P121
Halayko AJ, S86
Haldar P, M34, M35, M36, M38, M42, M43, M47
Hale M, P200
Hale MJ, P14
Hall E, M18
Hall H, S67
Hall IP, S28
Hall P, S48
Hall R, P224
Hallifax R, S36, P269
Hallifax RJ, P81, P85
Halpin D, P2
Hamakarim T, P86
Hameed A, S82, P285
Hamilton FW, S123
Hamilton L, S12
Hamrouni M, S28
Han JK, S161
Han Liu W, S18
Hanania NA, S1, M14
Hancock N, P126, P169
Hand A, P275, P278
Hand S, P211
Handoo V, P50
Haney S, P132
Hanrahan C, P40
Hansel NN, S101, M14
Hanson H, S156
Haq A, S162
Haq N, S19, P58
Harding CJ, S26
Hardy A, P188
Hardy E, S14
Hardy J, S13, S15
Hargreaves L-M, S10
Haris F, P195, M24, M29
Haris M, S150, P92, P163, P208, P222, P274
Harries C, P283
Harries TH, S12
Harriman A, S79
Harrington J, P83
Harris F, S2
Harris K, P33, P36
Harris MM, P68
Harrison CH, S30, P261
Harrison D, P175
Harriss E, P269
Hart N, S78, P227
Hart S, S2, P101
Hartley T, S3, S13, S15
Harvey-Dunstan T, S28
Hashim M, P163
Hassan H, P211
Hassan MM, P171
Hassani A, P175
Hatton PA, S153
Hawkins NM, S101
Haworth C, T6, S37, S39, S42
Haworth CS, S38
Hay S, S45
Hayes G, P200
Hayhoe A, P187
Hayton C, S14, P97
Hazra NC, P8
Healy L, S46, S47, S49
Healy O, M18
Heaney L, S105, P75, P192, P207, P300
Heaney LG, S129, S133, P62, P118, P198, P202, M31
Heaps A, S15
Heatley H, S102
Heble J, S101, M13, M15
Hegab H, P226
Hegazi A, P127
Heightman M, P283
Helgeson SA, P158
Helman J, P297
Hemida MF, S73, S98, P177
Hemnes A, S143
Henderson J, S7, M22
Henderson N, M5
Henderson R, P132
Henley W, P291
Hennayake C, T6, S37
Henshall D, P86
Hepworth C, P146
Herbert S, P283
Herrejón A, M11
Heslin GC, P96
Hettiarachchi M, P217
Hewitt R, S154, P20, P22
Hickes W, S31
Hickey F, S171
Hickey L, P151
Higbee D, S51, M28, M32
Higbee DH, P70
Hill A, S39, S42
Hill AT, S2
Hill M, P287
Hillson K, S45, P138
Hilton ST, S76, S94
Hinchcliffe F, P275, P278
Hinde S, P169
Hingorani A, S67
Hinks T, T4, S105
Hinks TSC, S61, S106, P238
Hitchcock F, S82
Ho OSH, S22
Hockley J, S126
Hodgetts R, S62
Hodkinson P, S2
Hodson J, S79
Hoffman EA, P257
Hogarth DK, P302
Hogben C, P22
Hogg AEJ, P129
Hokmabadi A, P281
Holden E, P188
Holehouse R, P43
Holland CW, P175
Hollins S, S148, S170
Holmes R, S37, P149
Holt K, P106
Hong A, P31
Hooi Tee A, P12
Hoole S, S83, S85
Hopewell H, M38
Hopkinson N, P30
Hopkinson NS, S11, P33, P122
Horgen FD, S89
Horst C, S67
Horton K, S108
Houchen-Wolloff L, P31
Houghton L, P215
Housley D, P237
Hovel M, S63
Howard AL, S5
Howard LS, S146
Howarth P, S105, S160, S164, P199, P201, P302
Howell T, P219
Howkins K, P43
Howlett P, S112, S113, P73, M37, M46
Hoxha A, M48

Hoyle JL, S116
Htwe T, P12
Huajian L, P241
Huang C, M21
Huang R, S14
Huang Y, P185
Hudson B, T1, S156
Huerta A, P4
Hughes A, P152
Hughes DA, P183
Hughes G, S14
Hughes K, P174
Hughes R, T6, S37
Hughes S, M10
Hulance B, P188
Hull JH, S25, P103, P104, P253
Hull R, S37
Hull RC, T6
Humbert M, S143, S146
Hume E, S29, S141
Hunter C, P109, P110, P114
Hunter E, M32
Huntley CC, S111
Hurdman JA, S82, P285
Hurst J, S39, S42, P188
Hurst JR, S67, S101, M15
Hurt K, P188
Husband A, S125
Hussain A, P167
Hussain I, P167
Hussain M, P171
Hussain S, P51, P54
Hussein M, S98
Hussell T, S62
Hutchinson A, S11
Hutchinson N, M9
Hutton G, P126
Hutton GK, P164
Hwee J, S162

Ibrahim AA, S73, P177
Ibrahim E, P160
Ibrahim K, S2
Ibrahim W, S176, P13
Idris E, P75, P205, P206
Idris O, P226
Idzik A, M45
Ifzaal M, P171
Ignatowicz AM, P120
Iheozor-Ejiofor Z, S115
Ilyas M, P277
Imaan S, S19, P58
Imtiaz Z, P151
Ing A, P210
Injety M, P41, P225
Insall R, S32
Ionescu A, S35, S150, P163
Ionita A, M10
Ip H, M41
Iqbal B, S36, S147, P77, P81, P269, P276
Iqbal M, S172, P159
Iqbal MM, P191
Iqbal N, P82
Ireland PJ, P153
Irojah K, P228, P229
Irshad S, P233
Irving S, S45
Ishaque G, S84
Ishaque M, S84
Ishtiaq S, S98
Ismail N, S14
Ito K, P247, P248, P249, P250
Iyer A, P219
Iyer S, S74

Jackman C, S48
Jackson A, S132, P291, P294, P298
Jackson D, P200, P207
Jackson DJ, S104, S133, P75, P195, P198, P201, P202, P297, M24, M29, M31
Jackson MM, T2, P192
Jacob J, S67
Jacobs E, M10
Jacques L, P201
Jafri S, P256
Jaggi T, S58
Jahangir M, P51, P54
Jain J, P236
Jajbhay D, P78
Jakobi T, S58
James J, S118
James LK, S109
James T, S136, P154
Janes SM, S67, P170
Janssens W, S1, P4
Jarreta D, S135
Jarrold I, S11
Jarvis H, M41
Jawad H, P92
Jeerakathil T, P287
Jenkins E, S90, S167, S168
Jenkins G, P22, P242, P254, P283
Jenkins R, P20
Jenkins RG, S87, S91, P100
Jennings M, S68, P133
Jerrard K, P38
Harris J, P184
Jha A, P196
Johal D, P143
Johannson K, P94
Johansson M, P164
Johar A, S124
Johl N, P188
John A, S165, P242
John AE, S87, S91
John J, P45
Johnson E, T6, P136
Johnson J, P37
Johnson M, S110
Johnson RS, P239
Johnson S, S116
Johnston SL, T2, S58
Johnstone S, P275, P278
Jolley CJ, P111, P263
Jones A, S5, P11, P179
Jones ABP, P178, P182
Jones DJL, S88
Jones J, P55
Jones K, P188
Jones L, S159, P118
Jones MG, S157, P28
Jones P, P257
Jones R, S146
Jones RJ, S143
Jones TL, S134, P203
Jongh F, M19
Joplin H, M30
Joseph A, P89, P90
Joseph J, P215
Joseph S, P125
Joshi D, S12
Joshi L, S63
Joshi M, P94
Judge S, P147
Jung SH, M42
Juniper M, P78

Kabalan L, M10
Kabir T, S112, S114, P73
Kadam R, P5
Kadwani O, S150, P78
Kaenmuang P, P23
Kahai R, P20
Kakouri E, P180
Kalam S, P82
Kalapurakal K, S5
Kalkat M, P208
Kalli S, P151
Kaltsakas G, S77, S78, S95, P227, P235, P264, P265, P266
Kamal F, P192
Kamath A, P180
Kamavuako EN, P263
Kamil A, M34, M36
Kamil HG, P208, P222, P274
Kan A, S103
Kanabar V, S104
Kanabar-Raivadera V, S108
Kandiyali R, S6
Kanellakis N, S65, S147
Kanellakis NI, S148, S170, P77
Kangulec S, P143
Kannampuzha L, P196
Kaplan A, S102
Kardasopoulos D, S96
Kareem T, P92
Karir A, P210, P213, P214, P216, P218
Karlsen APH, P262
Karpetis E, S116
Karsanji U, P123
Katerelos P, P292
Katsampouris E, P164
Katsaounou P, P292, M8
Katsoulis O, T2
Kaul H, P302
Kaur J, P64
Kaushal N, P81
Kawalek J, P33, P36
Keane F, P105, P107
Keegan R, S15
Keeling N, P198
Kelleher P, P192
Kelly AJ, S89
Kelly C, S66
Kelly D, P215
Kelly M, S39, S42
Kelly R, M18
Kelting F, S74
Kemp B, P71
Kennedy B, S20, P57
Kennedy MPT, M5
Kennedy N, S13, S15, P74
Kennington E, S11
Kennington EJ, P149
Kenny P, P241
Kent BD, S20, P7, P57, P303
Kenward A, P45
Kerr P, P193
Kerr PJ, S50
Kette S, P81

Khan AA, S84
Khan F, P19, P26, P254
Khan I, S84
Khan M, P173
Khan S, S35, S36, P171, P205, P206, P221, P274
Khan T, P177
Khan WA, P9
Khan Z, P26
Khanijoun N, S14
Khanom F, P246
Kharodia T, S111
Khaw CR, S67
Khooshemehri P, S104, P195, P244
Khunti K, P123
Kidd AC, S32
Kidney JC, S4, S118
Kidson CRT, P280
Kiely D, S81, P284
Kiely DG, S82, S146, P281, P285
Kilbride C, S76, S94
Kim D, S32
Kim JW, M34, M36
King JA, S69
King L, P143
King N, P33, P36
Kinsey J, S136
Kirkham S, S62
Kirkwood J, P98
Kite C, S23
Kitt M, P107
Kitt MM, P112
Kleem T, P210
Klein L, S4
Klenerman P, S61, P238
Klooster K, P210
Knaier R, S25
Knappenberger K, P297
Knight J, S147
Knight JC, S148, S170
Knight SB, S33, S62
Knolle M, P196
Knox AJ, S86
Knox-Brown BK, S26, P286
Kobor MS, S86, S107
Kogh K, P269
Koh MS, S102
Kok-Ting Wan P, S65
Kokosi M, S154, P20, P22
Kolosz B, P296
Kolvekar S, P255
Kon OM, S113, M37, M48
Kon SS, S11
Kon SSC, S155
Kontou K, P31
Kontra I, S109
Kooner-Evans S, S157
Korff G, P20
Korkontzelou A, P292, M8
Korn S, S1, P198, P202, M13
Kosmidis C, S124
Koster D, P210
Kostikas K, P292
Koukaki E, P264
Koul P, P5
Koulopoulou M, P102
Koulouris N, P264, P265
Koulousousa E, S148, S170
Kouranos V, S154, P20, P22
Kouritas V, P211
Kovacs G, S146
Kramer A, P75
Krassowska K, S175, M16
Kricker J, S57
Krishnamurthi R, P187
Kulasekararaj A, S156
Kulkarni T, P5
Kumar A, S24
Kunst H, P188, P190
Kunzemann Martinez A, P243
Kurmi OP, P139
Kurukulaaratchy R, S133, M31
Kusumoto M, T2, S58, P192
Kwok A, P25
Kwok H, S138
Kyi NM, S14
Kyrou I, S23

La Rosa C, P115, P116, P117
Labaki WW, M14
Lacbay S, S45, P138
Lagan AL, S86
Lagergren P, S124
Lagojda L, S23
Lai LYH, M25
Lakhani N, P65
Lam CSP, P8
Lam E, S5
Lam J, P195, M24, M29
Lam MT, P51, P54
Lambert C, P135
Lambert G, P150
Lampadakis S, P212
Landers C, S72
Lane A, S6
Lane N, S3, S96
Langley R, P136
Latham M, S19, P58
Lau A, S96
Lau L, T5, S108
Lauterio M, P128
Laverty A, P122
Laverty S, P192
Laviolette M, P302
Lawrence H, P188
Lawrie A, S143, S146
Laws A, S32
Lawson J, P66
Lea S, S59
Leary RA, P69
Lecky F, S6
Lee B, S130
Lee D, P284
Lee J, M34, M35, M36
Lee JK, S164, P199
Lee K, P54
Lee KK, P48, P51
Lee KL, P56
Lee PH, P178, P182
Lee S, M41
Lee TT, S102
Lee WTN, S133, M31
Legg R, S151
Lehmann M, S161
Lei M, P250
Leslie K, P176
Leung N, M32
Lever H, P108, M26
Lewin S, P33
Lewin SA, P36
Lewis D, P89, P90
Lewis G, P289
Lewis KE, M7
Lewis M, S109
Lewis S, S2
Lewtas A, S138
Li D, P275, P278
Li O, S83
Li S, P238
Li T, S161
Liang J, P78
Liapikou A, P251, P252
Liew F, S56
Liew MF, S54
Lim CH, S96
Lim CJ, P288
Lim J, S72, P1
Lim RH, P150
Limbrey RM, S157, P28
Lin D, S86
Lin M, P91
Lin WY, S146
Linden D, P241
Linden DA, S4, S118
Lindop J, P126
Lindsay M, P240
Lines S, S17, P98
Lines SJ, P17
Ling K, P224
Linkson L, S2
Lino-Alvarado A, P157
Lipman M, S126, P188, M33, M39
Lipman MCI, M41
Lipworth B, S131, P268, P291
Lipworth BJ, S161, P197, P204
Lipworth B, M19
Lipworth J, P200
Lithgow A, P188
Liu B, S87, S91
Liu MC, S164, P199
Llaneza Lago S, P269
Llanos J-P, P198, P202
Lloyd CM, S18
Lloyd V, P188
Lo DKH, S43
Lodge KM, S165, P239
Loebinger M, S41, S42
Loebinger MR, T6, S39
Logan J, S133, M31
Logue P, P241
Long MB, T6, S37
Long Y, S61
Longhurst H, S156
Loo Y, P215
Lopez J, P181
Lopez Jimenez EL, S87
Lopez R, S60, S99, P162
Lordan J, S133, S146, M31
Lorent N, P190
Loukides S, P161, P212
Loutsios C, P196
Lowe DJ, P3, P148
Loy M, P237
Lozano-Rojas D, S56
Lozewicz S, M39
Lucas J, P137
Ludlow S, P72
Luengo-Fernandez R, S152
Lugogo N, P297
Lugogo NL, P198, P202
Lupton C, P10, P200

Lusardi N, P143
Lutchegadoo S, P12
Lutchman Singh KLS, M1
Lyen S, T1
Lynch GA, S35
Lyng E, P205, P206

Ma D, P247, P248, P249
Ma J, P250
Ma X, P263
Ma Z, P247
Macdougall M, P163
Machado A, S127
MacHale E, S50, P193, P303
MacIsaac JL, S86, S107
Mackay A, P150
Mackay EC, P103, P104
Mackay L, P132
Mackenzie R, S12, P33
Mackenzie Ross R, P282
Mackenzie-ross R, T1
Maclay J, P163
Macleod KA, P134
MacLeod MA, P162
MacPherson A, S32, P163
Macpherson L, S16
Madabhushi H, S125
Madas S, P5
Madkhali A, S77, P266
Mafham C, S167, S168
Mah J, S60, S99, P162
Maher K, P284
Mahida R, S167, P240
Mahida RY, S168
Mahmood A, P21, P65
Mai Y, S61
Maidstone R, P245
Maidstone RJ, M17
Main E, S76, S94
Majeed M, P171
Majrshi M, P30
Majumdar A, P2
Makhecha S, S48
Malachira A, P188
Malhotra A, P188
Malhotra AM, P190
Mallea JM, P158
Mallia P, T2, S58
Malm Tillgren S, S57
Malone R, P291
Mamo S, P131
Man Kwong G Ng, S14
Man WD-C, S11, S155
Manal F, P93
Mandal S, S76, S94, P46
Mandizha J, P17, P98
Manga A, P19
Mange K, S38
Mangera Z, M41
Manifield J, S140
Mankad V, S161
Mannan S, P228, P229
Manoharan N, S65, S147, S148, S170
Mansell SK, S76, S94
Mansoor H, M43
Mansur A, S105, P200
Mansur AH, S129, S133, P75, P205, P206, P207, P300, M31
Manuel A, S24, P152, P223
Marchese C, P202
Marchi E, T4, S105, S106
Marchini G, P243
Marciniak S, P83
Marciniak SJ, S142, S151, P178, P182
Marghlani D, S124, S127
Marguerie S, P28
Marguerie SE, S157
Marion A, S99, P162
Marion ANJM, P257
Marquette M, P211
Marray CS, S49
Marsden P, P108, P113
Marsh C, S10
Marsh J, P205, P206
Marshall B, P220
Marshall BG, S157, P28
Marshall C, P126, P169
Marshall J, S99
Marshall S, P43
Martin C, M47
Martin G, T6
Martin L, P188, M48
Martin N, P198, P202
Martin Nguyen A, P115, P116, P117
Martinelli AW, P85
Martinez FJ, M15
Martínez Molina J, S158
Martinez Nunez RT, S104
Martinez-Nunez RT, T5, S108, P244
Masani V, P168
Maskell N, S32, S150, P184
Maskell NA, S6, S152
Masoli J, P301
Masoli M, P300, P301
Mason C, P223
Mathews KS, P6
Matonhodze M, P205, P206
Matthaiou AM, P251, P252
Mattock L, S74
Mauger D, S38
Mawson P, S39, S42
Maxwell CB, S88
May J, S87, S91
May ST, S86
Maynard O, M3
Maynou L, S42
Mayoral K, P138
Mayoral Oritz K, S45
Mayse ML, P210
McAndrew A, S152
McArthur S, P259
McAuley DF, S4, S169
Mcauley HJ, P4
McAuley HJC, S176, P13
McCabe C, P283
McCabe J, S67
McCabe K, P62
McCallion P, P39
McCallum H, P175
McCallum J, S126
McCann C, S18
McCarthy C, S63
McCarthy CM, P1
McCarthy JM, P1
McCarthy S, S3, P124
McCollough N, P62
McConnachie A, S133, M31
McCormack J, S63
McCormick L, P237
McDonald FE, P175
McDowell PJ, S133, P62, M31
McElroy L, P136
McGarry J, P163
McGarvey L, S105, P113, P115, P116, P117, P257
McGarvie S, P25
McGoldrick M, M18
McGouldrick MA, S124
McGowan A, P256
McGown AD, M40
McGuire A, S42
McHugh E, P131
McInnerney D, P126
McIntosh E, T6, S37
McKean M, S63
McKeever J, S131
McKeever TM, S28, P64, P185
McKelvey MC, S4
McKenzie J, P192
McKinnon A, P25
McKone EM, S72
McLaren J, S161
Mclaughlan D, S5
Mclean C, P141
McLeavy A, P143
McMullan CE, S88
McNally E, S20, P270
McNally R, S39, S42
McNeil L, S127
McVeigh JG, P40
Megally A, P297
Megaritis D, S29, S141
Meghani NJ, S116
Meghji J, M47, M48
Mehdi SB, P217
Mehmood A, P226
Mehmood H, S98
Mehmood T, P205, P206
Mehta H, P48
Mehta S, P283
Meireles I, P213, P214, P216, P218
Melhorn J, S106
Melville M, P43
Menzel M, S57
Mercer W, P181
Merriman R, P188, P190
Message S, P75, P205, P206, P300
Messer B, S93
Metcalfe C, S6
Metersky ML, S38
Meyhoff CS, P262
Micallef ACL, P183
Micallef R, P143
Middleton S, S28, P185
Millaire É, P113
Miller C, S32
Miller D, S2, P134
Miller J, S2
Miller R, S36, S150
Millington T, S10
Milne A, S150
Milroy R, S2
Mincham KT, T2
Minnis P, S16, P21, P241
Mir A, P282
Mirabelli F, P20
Miravitlles M, M15
Mishra E, S35, S149, P269
Mishra EK, P187
Mistry V, S176
Mitchell B, P17

Mitchell J, P106
Mitchell JA, S91
Mitchelmore P, P178, P182
Mitchinson L, P126
Mobus S, S26
Modafferi G, P243
Moffat K, P134
Moffatt MF, P247, P248, P249, P250
Mohammad S, P82, P275, P278
Mohammed A, P178, P179, P182
Mohammed IS, S97
Mohan A, P5
Mohan D, S1, P150
Mohan T, P2
Moitra S, P5
Molgaard J, P262
Molyneaux PL, S18, S154, P22, P254
Molyneux AW, S92, P237
Molyneux P, P20
Mondal A, P94
Montalto D, P118
Monteiro W, S172, P159, M35
Moore D, S4
Moore E, P181
Moore F, M18
Moore M, P118
Moore S, S145, M40
Moore VC, S27
Moore-Crouch R, S48
Moores R, M41
Morais C, M35
Moran E, S16
Moran N, P215
Moreira LA, P162, P257
Moreland JA, S31
Morelli T, P11, P178, P179, P182
Moreno Sapena J, P198
Moreno Sepena J, P202
Morgan AD, P100
Morgan C, S51, P156, M22, M28, M32
Morgan CR, P70
Morgan D, P188
Morgan H, S111
Morgan M, S11
Morgan S, P230
Morgan SB, S61
Morgan T, P33, P36
Morice A, P113, P115, P116, P117
Morland K, P91
Morozow A, P219
Morrell N, S145
Morrell NW, S142
Morris CE, P290
Morris T, P61, M21
Morris-Rosendahl D, S156
Morrison M, P163
Morviducci M, P20
Moss A, S75
Moss J, P17
Mostapha N, P41, P225
Mounsey C, P81, P269
Mounsey CA, P77
Mountjoy E, S37
Movitz C, P297
Mowat A, P53, P228, P229
Mpagama S, S113, M37
Mplizou M, P212
Mudassar R, P93
Mughal A, P188
Muhammad A, P93
Muhammad M, P82
Mukherjee B, P283
Mukherjee R, P234, P236
Mulholland S, S13, S15, P102, M32
Muller E, S68, P133
Mullerova H, S99, P8, P156, P257
Mullin ML, S67
Mullol J, S160
Mullvihill J, P69
Munavvar M, P217
Munday JG, P14
Murphy A, S54, S172, P14, P159
Murphy AC, P26, P65, P290
Murphy P, S78, P235
Murphy PB, P227
Murphy RA, P183
Murphy S, P43, P188
Murray C, M23
Murray CS, S46, S47, P260
Murray J, M33, M41
Murray L, S2
Murray N, P188
Murray R, S20
Murray T, M25
Murtagh E, S16
Murthy N, S103
Mushtaq I, S73, S98, P177
Mussell G, P153
Mustafa R, S2
Muthusami A, P101
Mwanga E, S113, M37
Mwasuku C, S122, S175, P157, M16
Myall K, P18, P283
Myall KJ, P103, P104
Myers J, P188
Myerson J, P178, P182

Nabayego P, M10
Naeem M, P221, P271
Naftel J, T5, S108
Nagarajan T, S2, S136, S150, P154
Nagel M, P267
Nagra S, P125
Naidu B, P240
Naidu SB, S67, P170
Nair A, S67
Nair P, S1
Najafi K, S121
Nakanishi S, S165, P239
Nanda V, P180
Nanzer A, P200
Nanzer AM, S104, P195, M24, M29
Naqvi M, P102
Naran P, M33
Naran R, M39
Narayan S, S89
Nasa P, P229
Naseer MH, S3, P124, P222
Nashat H, P283
Nasim MZ, P55
Nasser S, P196
Naureen S, P101
Nava GW, S51, P68, P70, M22, M28, M32
Navani N, P170, M41
Navaratnam V, P100
Navarro M, P196
Naveed S, S54, P75
Nayak A, P5
Nayyar M, P231
Neal MA, P175
Needham P, M18
Neelam-Naganathan D, S146
Neilly M, S32
Neilly MDJ, P163
Nelson A, S18
Neto E, S29, S141
Nettleton K, S111
Neupane S, P139
Neville D, S134, P203
New JM, T6
Newing A, P270
Newman J, S143, S146, P50, P196
Newman JN, P286
Newman L, P29
Newnham N, P275, P278
Newport D, M42
Ng AYKC, S143
Ng J, S1
Ng KL, S31
Ngeh J, P138
Nguyen DTH, S166
Nguyen-Phuoc K, S18
Nicholson T, S72, P219
Nickol AH, M6
Nilsen T, M35
Nissim A, S109
Nixon N, P74
Nobes E, S53
Noble JH, S52
Norman J, S106
Norris K, P98
Norris V, S57
Norwell J, S127
Novotny P, S172, P159
Novsarka I, M34, M35, M36
Ntotsis K, S54, S56, S172, P159, P191
Ntoumou V, M8
Numbere NK, P188
Nuttall M, M2
Nyikadzino K, M40

O'Beirne R, S44, P68, P144, M22
O'Brien H, S63
O'Brien HO, P1
O'Connell B, P114
O'Connor J, M28
O'Connor-Power F, P273
O'Driscoll BR, P262
O'Garra A, M36
O'Hara K, M25
O'Hickey S, S111
O'Kane C, S169
O'Kane CM, S4
O'Mahony A, P87
O'Malley D, S63
O'Neill B, P38
O'Regan E, M18
O'Reilly A, M47
O'Reilly-Smyth A, P66
O'Riordan SJ, P273
O'Shea MK, M42
Oakden V, P171, P208, P222, P274
Oakes A, P234
Oatey K, S2
Obasi C, P297
Obisi N, P228
Odin B, P33
Odubanjo T, M39
Offen D, P127, P165
Ogden M, S99

Ola M, P302
Oldham N, P79
Oliver C, P140, P141
Oliver E, S142
Olsen MH, P262
Olsen O, S76
Omar M, M33
on behalf of East of England Severe Asthma Network, P196
on behalf of the CoMiTED Investigators, S6
On JY, M19
Onasanya AA, P120
Ong VH, P23
Ong W, P132
Openshaw PJM, S56
Orme MW, S137, S139, P32
Orton CM, P210, P213, P214, P216, P218
Osarogue A, M8
Osborne K, P18
Osman L, M24
Ostridge K, P257
Ottewill C, S50, P69, P193, P303
Overton K, P194
Owadally Y, P162
Owen C, P196, P207
Owusu-Afriyie J, S95, P266
Ozsancak Ugurlu A, P234

Padley S, S32, S70
Page J, P280, P282
Page JW, T1
Page K, S34
Page R, M35
Paiva AR, P17
Paixão C, S127
Pakpahan E, S3, P124, P153
Palazon A, P239
Pallarés A, M11
Palmer-Walsh A, S152
Panchal R, S34, S150, P163, P275, P278
Pandya H, P297
Pang YL, S107
Panou V, S32
Pant S, S23
Pantin T, S116, P75
Pao C, P135
Papadopoluou E, S127
Papapostolou E, P292
Papasarantou A, P264, P265
Papavasileiou V, P161, P212
Papi A, S132, P2, P297, P298, M13
Papineni P, P188
Paramasivam E, M5
Paraskakis E, S45
Pareek M, M43, M47
Parekh D, S90, S167, S168
Parfrey H, P96
Park JE, M6
Park M, M48
Parker GJM, S70
Parker R, P143, P223
Parker S, P109, P110, P114
Parker SE, S172, P159
Parker SM, P113
Parkes ED, S27
Parkinson S, P174
Parnham MJ, S57
Parrott H, P140
Parsons L, P63
Parsons M, P242
Parulekar A, P198, P202
Pascual S, M11
Passariello M, S154
Pasteur MC, P187
Patel A, M33, M41
Patel AX, P150
Patel B, S154
Patel D, S33
Patel H, S12, M38
Patel I, S7, M22
Patel J, S86, S107
Patel K, S73, S98, P15
Patel M, S2, S129, P75, P297, P300
Patel P, S133, S163, P75, P118, P131, P200, P207, P300, M31
Patel PH, S129
Patel R, S67
Patel S, S11, P143
Patel V, P118
Paterson F, M19
Pates K, P186
Patoole S, S150
Patrick J, P156
Patrick T, S67
Patterson Z, P154
Pauling J, S15
Pavitt M, P178, P182
Pavlou B, S45
Pavord I, S105, S174, P291
Pavord ID, S52, S106, S133, S135, M31
Paxton K, P284
Payne E, P132
Peace L, P109
Pearson J, S32
Pearson RA, P175
Peel T, S165
Pepke-Zaba J, S83
Pepke-Zaba JPZ, P286
Perera MI, P47
Periwal P, S157
Perucha E, P244
Peto R, P138
Petrica N, S81
Petrou I, T6
Petta V, M8
Pfeffer P, P71, P121, P200
Pfeffer PE, S109, S129, P14, P67, P75, P300
Philip G, P115, P116, P117
Philip K, P30
Philip KEJ, P122
Phillips A, S111
Phillips G, S24
Phillips J, T5, P175
Phoo Thwe KM, P87
Pickles R, P175
Piggott L, S20, P7, P57
Piggott LM, P303
Pike J, P200
Pilania K, P94
Pilette C, S164, P199
Pillai A, P205, P206
Pillay TD, M33
Pilling L, S5
Pindolia K, P63
Pini M, S81
Pink K, P200
Pioselli B, P243
Piper R, P173
Piraino A, P268, P294, P298, M12
Pirzada O, P188
Pisacano N, P239
Pitel L, M21
Pitsidianakis G, P251
Pitts O, S58
Pizzo E, P254
Pizzoccheri R, M12
Plant P, P223
Platé M, P243
Platt A, S161
Plebanski M, P246
Poirot O, P107, P112
Poku I, S5
Poli G, P6
Polkey M, P30
Pollack M, P8, P156
Polverino E, T6
Polwarth G, S143
Poole HW, M9
Poole S, P24
Poole-Cowley J, P136
Poozhikunnath B, P42
Popoola C, S131
Porte J, S28
Porter JC, P100
Poto R, M19
Pott H, P4
Pott J, P67
Potter J, M39
Potter JL, M41
Potts J, S35
Poupouzas G, M8
Powell B, S14
Powell I, S5
Powell M, P119
Pradeep V, P156
Praet N, P6
Prague JK, P98
Prakash Kurmi O, S23
Prasad A, P153
Prashntkumar S, S84
Premuda C, P186
Prendecki R, S67
Preston M, P66
Preston R, P270
Prevost AT, S11
Price D, S102, P291, P294, M10
Price L, P30
Price LC, P283
Prior K, P75, P300
Proenca C, S81
Prosper S, S28
Prudon B, S3
Pryer E, P196
Puaar S, P143
Punja P, S125
Punton S, P137
Purcell M, P178, P179, P182
Purdon B, P163
Pye A, S119, S128
Pye HV, P187
Pyman E, S87

Quaife SL, P126, P164
Quarton S, P16
Quinn C, P273
Quinn P, P224
Quint J, S41
Quint JK, S7, S43, S44, S137, S138, S139, P4, P32, P68, P100, M22
Quon BS, S68

Qureshi V, S84

Rabbani F, P173
Rabe KF, S101
Radcliffe G, S95, P266
Rafferty G, S77, S95
Rafferty GF, P263
Rahim Y, P221, P271
Rahman L, S91
Rahman M, P82
Rahman N, S31, S35, S147, S149, S150, P163, P269, P276
Rahman NM, S36, S65, S148, S170, P77, P81, P85, P272
Raj T, S14, P97
Raja Gopal V, P61, M21
Rajasekar P, S86, S88, S89, S107
Rajeev Y, S69
Rajhan A, P233
Rajini S, P163
Raju S, P42
Rakić M, S155
Ralph L, S157
Ramachandraiah S, P29
Ramachandran P, P79, P277
Ramadore S, P127, P165
Ramakrishnan S, S100, S174, S175, P4, P155, P157, M16
Ramalingam S, S2
Ramaswamy M, P91
Ramsay M, S78, S95, P227, P235
Randazzo E, S4
Ransley D, S154
Rao R, S103
Raote P, P253
Raptakis T, P212
Rashid B, P236
Rashid MT, S92
Rashid S, P188
Rashid Z, P26
Rashkovetsky R, P287
Rathnapala A, P94
Ratjen F, S68, P133
Ratnasingham M, S89
Ratnayake P, P233
Rattigan E, S126, P188
Raveendran K, P22
Rawal B, P283
Rayment JH, P133
Raywood E, P140, P141
Read AT, M41
Reader R, P285
Reddy R, P163, P222
Reddy RV, P221, P271
Redfern OC, S30, P261
Redmond C, S129
Redmond KL, S118
Reed A, P192
Rees S, S6
Rehill A, P270
Rehman S, S98
Reid P, P163
Reilly C, S111
Reilly CC, P111
Reinders LG, P150
Renaudat C, S41
Rennison-Jones C, S13
Renzoni E, S154, S156, P20, P22, P283
Renzoni EA, P23
Rhatigan KL, P103, P104
Rhodes CJ, S143, S146
Rhodes J, S149
Riaz R, S84
Richards J, P293, P295
Richardson C, P145
Richardson H, T6
Richardson M, S56, S137, S139, S176, P32, P302
Riches C, P194
Richmond RC, S117
Ricketts W, P255
Ridge C, P257
Ridout D, S76
Ritchie AI, P257
Ritchie L, P147
Rivera-Ortega P, S13, S15, S17, S156, P17, P98
Rivers P, P65
Rizvi S, P26, P82
Roach KM, S88, S89
Robbins HA, P164
Roberts C, P20, P253
Roberts J, P215
Roberts NJ, P34, M2
Roberts-McCarthy E, P91
Robertson LR, P286
Robinson GV, P59
Robinson M, P194
Robinson P, P138
Robinson R, S71
Robinson S, P91
Robinson-Barella A, S125
Robson J, M5
Roche J, P163
Roche JJ, S32
Roche N, P8, P294, M14
Rochell E, S138
Roddy E, M45
Rodger K, M5
Rodrigues F, P215
Rodrigues JCL, T1, S13, S15, P280, P282
Rodrigues P, P178, P179, P182
Rodríguez Gallego Y, S55, P258
Rogerson S, P126
Rogliani P, S101, P197, M12
Rollo RF, P183
Rooney E, M42
Rooney M, P66
Rosario J, M25
Rose L, S122, S126, P71
Rose V, P71
Rosenblatt J, P242
Rosenthal M, S70
Ross C, P111
Ross E, P136
Rossanese M, P243
Rossdale J, T1, P282
Rosser A, P188
Rostron A, P293, P295
Rothman AMK, S82, S143, S146
Rothman ARK, P285
Rowe C, P215
Rowland L, P17
Rowland MJ, S4
Rowlands B, P80
Roxas C, P195, M24, M29
Roy K, P63
Rudman S, M28
Ruffino G, S13, S15, P173
Rupani H, T5, S108, S129, P75, P76, P118, P200, P207, P300
Russell A-M, P129
Russell AM, P98
Russell R, S11, S12, P152, P156, M25
Russell REK, T3, S9, S100, S175, P10, P155, P157, M16
Russell RJ, P302
Russell R, P293, P295
Rutter MK, M17
Ryan C, S51, M28
Ryan D, S50
Ryan P, P205, P206
Ryder S, P63
Rynne J, P244

Sabale A, P5
Sabtu N, P188
Sadler-Ladell J, S36
Sadoun A, S21
Sagar D, P12, P60
Sage E, P163
Saghir M, S73
Saglani S, S45, P138
Saha S, M25
Saha-Chaudhuri P, M15
Sahay S, P284
Said B, S113, M37
Sakota H, P70, M32
Salcedo Lobera E, S55, S158, P258
Saleem Ullah A, P76
Salehi M, P281
Salehian SS, P141
Saliu E, P173
Salman A, P93, P171
Salman R, P74
Salter P, P297
Salvadori M, P6
Salvi S, P5
Sammut D, S159, M25
Sampsonas F, S148, S170
Sandri F, P6
Sánchez-Covisa J, M11
Sanghavi K, T2
Sann K, P173
Santillo M, P289
Sapey E, M20
Saqib H, P26
Saralaya D, S2, S133, M31
Sarfraz MR, S73, S98, P177
Sathanapally H, P123
Sathyamoorthy T, P277
Satia I, P113, M13
Sattar N, S102
Saumtally H, P189
Saunders G, P143
Saunders P, P24
Saura A, M11
Sava IA, S166
Savale L, S143
Savill A, P98
Sawant AP, P189
Sayao J, P228
Sayers I, S107
Sayers R, S52, P98
Sayo M, P53
Scadding A, S3
Scaffidi Argentina U, M12
Scaffidi-Argentina U, S132, P294, P298
Schievano S, S76, S94
Schiza S, P252
Schleich F, P201
Schlotterbeck J, S4

Schmeck B, P4
Schmid CM, S151
Schmidt-Trucksäss A, S25
Schofield S, S114
Scholand MB, P284
Schumann DM, P302
Schwarz C, S68
Schwendinger F, S25
Schwiening M, S142
Sciurba FC, M15
Scott A, S90, S167, S168, P240
Scott D, S11
Scott J, P153
Scott M, P153
Scott R, S71, P184
Scott SE, P164
Scotton CJ, S13, S15, S17, S156, P98
Screaton N, S32
Searle L, S74
Seatter A, T1
Seecharran T, S149
Segal T, P145
Sehajpal R, S163
Sehdev S, S11, S155
Sehgal N, S116
Sein Z, P80
Selby A, P142
Selby B, S2
Selby M, M35
Selfe L, S125
Selvakumar L, P287
Semple T, S70
Senior N, S5
Senkathir M, S82
Sereno M, S56
Serjeant H, P17
Sestini P, P20, P23
Sethi DK, S149
Sfika M, P212
Shabnam S, S43
Shafiq F, S84
Shafiqa N, S5
Shah A, S58, S124, S127, S163, P186, P192
Shah K, P14, M21
Shah M, P297
Shah N, S78, P227, P235, P265
Shah PA, S54
Shah PL, P210, P213, P214, P216, P218
Shah S, P143
Shahbakhti H, S99, P162
Shahidi M, P188
Shahin Y, P288
Shahridan Faiez T, S58, P192
Shaikh A, S97
Shakeel A, P93
Shakespeare J, S27
Sharhiar Z, S73, P177
Sharkey M, P281
Sharma A, P94
Sharma AK, P94
Shaw C, P147
Shaw D, S54, S107, P125
Shaw DE, P64
Shaw J, S14, S34, P97
Sheares K, S146
Sheares KS, P286
Sheego M, P205, P206
Shelley S, P57, P303
Shemald T, S159
Shen J, T4, S105
Sherlock C, M18
Sherratt F, S5
Sherrill JD, S161
Shield T, S39, S42
Shiner E, P111
Shirai T, P197
Shoemark A, S127
Short C, S70
Short P, P163
Shotton H, P78
Shrikrishna D, P75
Shrimanker R, S51, S105, P70, P75, M28, M32
Shrosbery F, S60
Sibila O, T6
Siddika A, M24
Siddiqui S, S133, P178, P182, M31
Simmonds I, P126, P169
Simmons A, P238
Simons A, P25
Simons SO, P150
Simpson A, S46, S47, S49, P246, P260, P296, M23
Simpson AJ, S46, S49, P153
Simpson D, S4
Simpson DA, S118
Sindelar M, S4
Singanayagam A, T2, S58, S124, P192
Singapuri ASDD, P302
Singh A, P5, P123
Singh C, P5, P125
Singh D, S59, S132, S136, P154, P156, P257, P294, P298
Singh H, S5, P48
Singh N, P94
Singh P, P94
Singh R, P55, M10
Singh Randeva H, S23
Singh S, S29, S141, S154, P5, P94
Singh SJ, S137, S138, S139, S140, P32
Singh V, P94
Singham SS, P141
Sinha I, S44, P144
Sinnett T, S126
Sitbon O, S146
Sivaramakrishnan A, P233
Skeikh A, S2
Skene I, P67
Skinner D, S102
Slattery K, P270
Slebos DJ, P210
Slinger C, P108, M26, M27
Slough J, P27, P88
Small M, P128
Smallcombe N, S175, M16
Smalley DL, P130
Smets E, S11, S155, P3
Smith A, S2, P89, P90, P200
Smith DJF, S18
Smith DL, S18
Smith DP, S92, P237
Smith E, P82, P175
Smith J, P105, P106, P107, P115, P116, P117
Smith JA, P112, P113
Smith LJ, P48
Smith M, M7
Smith O, S50, P193, P303
Smith S, P188
Smith T, S167
Smith TS, S168
Smith WE, P260
Snelgrove RJ, T2, S60
Soares M, P169
Sobala R, P153
Sobczynska E, P215
Soe W, T5, S108, S157, P29
Sohn H, P280
Solé A, S68, P133
Soliman M, S101, S174, M13, M14, M15
Sommerwerck U, P128
Song CY, M18
Song WJ, P103
Sonnappa S, S45, P138, P145
Soo I, S154
Soon E, S142, S145
Soto Hurtado EJ, S55, S158, P258
Soto Soto JS, P158
Sou A, S14
Soukup T, S124
Sousa AR, S160
Sovani M, S3, P5
Sovani MP, P55
Spain N, S11
Sparkes P, M48
Spencer K, S90, S167, P240
Spencer KL, S168
Spencer LG, P25
Sperrin M, M25
Spinks KM, S157, P28
Spinou A, P33, P36
Spittle DA, S119
Spohr H, P211
Springer K, P297
Srinivasan D, P55
Srirangan A, P20, P23
Srivastava S, S78, P227, P235
Stacey R, P299
Stadon L, S150
Stamp A, P175
Stanel S, S13, S15, P98
Stanel SC, S156, P17
Stanton A, P163
Steed L, P67
Steer H, S35
Steer J, S39, S42, P153
Steier J, S21, S22, S77, S78, S95, P227, P266
Steiner M, S172, P123, P159
Steiner MC, S176, P13
Steinfort DP, P210
Steward M, P98
Stewart I, P242, P254
Stewart-Knight K, M24
Stimson S, P125
Stobo J, T6, S37
Stock C, P20
Stock CJW, P23
Stockley A, P17
Stockley R, S167
Stockley RA, M20
Stockton-Powdrell C, M25
Stoddart A, S2
Stolz D, P302, M12
Stone H, S111
Stone R, P62
Stone T, M3
Stoodley L, M6
Stratakos G, P264, P265
Strawbridge E, P173
Strickland J, S169
Stubbs H, S32
Stubbs HD, S66
Sturman J, M42

Stylianou P, S88
Suarez-Cuartin G, T6
Subbe C, S30, P261
Subramaniam A, P256
Subramanian D, P205, P206
Succony L, P163
Sudhir R, S34, P275, P278
Sudhir RC, P82
Suganthavel K, P47
Suggett JA, P267
Suh E, P235
Suh ES, S78, P227
Sulaiman N, P188
Sullivan A, S39, S42
Sullivan J, P205, P206
Sumbal A, P93
Summers C, S166
Sundaralingam A, S36, P81
Sunjaya AP, S11
Suntharalingam J, T1, S2, P282
Suran S, P137
Sutanto J, P109, P110, P132
Sutcliffe F, P96
Sutcliffe J, P25
Sutcliffe M, S8, S10
Sutcliffe R, S145
Suter P, P204
Sutharsan S, S68, P133
Sutherland T, P254
Sutherland TJT, P27, P88
Sutton B, P205, P206
Sutton D, S161
Swaffield B, P47
Swaminathan A, P284
Swarnakar R, P5
Sweetser T, P45
Swietlik E, S142
Swift AJ, S82, P281, P285
Swindle E, S108
Swindle EJ, T5
Syed R, P51, P54
Sykes DL, S127, S159, P194
Sylvester KP, S11, S26
Sylvester KPS, P286
Symonds J, S35
Symvoulaki K, P251, P252
Szkonter ML, S31
Szram J, S112, P73

Tabasum P, S98
Taboada DT, P286
Taggart CC, S4, S118
Taki A, P23
Tal-Singer R, S1
Talker L, P150
Tan GM, P166
Tan R, S54
Tan S, P162
Tan T, S130
Tana A, P210, P213, P214, P216, P218
Tandel S, S4, S169
Tanro M, P247
Tao A, S5
Tate M, S66
Tate P, P188
Taube C, P197
Tavernier G, P72
Tawalbeh R, P211
Taylor A, P3, P148, P151
Taylor E, S152
Taylor K, P17
Taylor L, S96, P219
Taylor S, S106
Taylor SJC, S11, P33
Taylor T, P105, P107
Taylor TE, P112
Tedd H, S96
Telisinghe L, P176
Templin H, S125
Teng NMY, S18
Teper A, S38
Testa I, S45
Thakrar R, M41
Thee S, S127, P186
Thelwall P, P175
Thenet D, S109
Thet P, S157
Thickett D, S90, S167
Thickett DR, S168, P240
Thickett K, M45
Thillai M, S15
Thomas A, S75, P233
Thomas C, P10
Thomas K, P181, M8
Thomas R, M13
Thomas S, S9
Thompson AAR, S82, P285
Thompson E, M33
Thompson F, P29
Thompson J, S6
Thomson L, P195, M24, M29
Thorne K, P178
Thorne KI, P182
Thornton T, S176, P13
Thould H, P98
Thrasher CM, P284
Thu A, S32
Thumbe A, S173
Thwaites RS, S56
Tibble H, S45
Tibiletti M, S70
Tickle M, P125
Tidmarsh B, P108, M26
Tilakaratne D, S14
Tinnion P, P152
Tisi S, S67
Titmuss V, P29
TNS the TRAJECTORY Study Team, S68
Tomas J, P16
Tomo U, S111
Tomos I, P251, P252
Tonkin J, P213, P214, P216
Tonkin-Crine S, P289
Topalović M, S155
Topalovic M, S11, P3
Torre IB, S163
Tortorella M, P44
Toshner M, P50
Toshner MR, S143, S146
Toshner MT, P286
Toumpanakis D, P161
Townend J, S102
Townson KM, P59
Trainer H, P213, P214, P216
Trasieva T, P297
Travassos E, P105
Treves J, P83
Trewick S, S5
Trindade APS, S163
Trivedi D, S33
Troosters T, P40
Truong V, S14
Tsaknis G, P221, P271
Tsaneva-Atanasova K, S13, S15
Tseng M, P149
Tsim S, S32, S66, S149, P163
TST the TRAJECTORY Study Team, P133
Tu L, S143
Tuck S, S2
Tuck SA, P210
Tucker E, S150
Tuli RT, P141
Tunney M, T6, S37
Turgoose J, M21
Turley G, P118
Turnbull C, M6
Turnbull H, P175
Turner A, S2, P113
Turner AM, S111, S119, S128, P15, P120
Turner L, S162
Turner RD, P103, P104
Turner-Frick E, S85
Turtle L, S5
Tweed C, M33, M39, M41
Tyas A, M25
Tyrer F, P291
Tzanakis N, P251, P252
Tzionis NK, S120
Tzouvelekis A, S148, S170

Ugurlu AO, P236
Uller L, S57
Unstead MP, P47, P59
Upadhyay H, S39, S125
Upton PD, S143
Urquhart DS, P134
Usher A, S2
Ussaid A, P82
uthas D, S161
Uttewill C, M23
Uwagboe I, P242
Uys S, P166

Vairelles C, S155
Vairon A, S87
Valand A, S86
Valerio C, M41
Valipour A, P210
Van Dijk M, P210
Van Geffen P, S44
Van Orshoven K, S11, P3
Van Tongeren M, S115
Van-Geffen P, P68
Van-Tongeren MJ, S110
Vandorou KT, P161
Vanfleteren LE, P4
VanGeffen P, M22
Vapiwala A, P19
Varma K, P15
Varoli G, P6
Vasarmidi E, P252
Veale N, P85
Vele A, S132, P298
Vele Witharamalage M, P84, P217
Vendrell M, T6
Venn H, P105, P107
Venn HE, P112
Verghese D, P156
Verghese P, S67
Verma R, M35, M38, M43

Vermeersch K, P4
Vermeulen I, P111
Verrier E, P205, P206
Veryard L, S157
Vestbo J, P257
Vethanayagam D, P287
Viaud I, P6
Vichiendilokkul A, S135, P201
Viganò LV, P6
Vijayakumar B, P97
Viligorska K, T6
Villar SS, S143
Villetti G, P243
Virchow JC, S132, P298
Virk H, P271
Vitri B, P22, P23
Vogelmeier CF, S1, S101, P156, M15
Vogiatzis I, S29, S141
Volhina O, P213, P214, P216
Vontetsianos A, P264, P265
Vorup-Jensen T, P183
Voulgareli I, P161, P212
Vozza E, S166
Vulkan D, P126, P169

Wagenmann M, S160
Wagner J, S25
Wain LV, S56, S86, S88, P191
Walas K, M24
Walker E, P3, P148, P151
Walker N, P163
Walker NF, M42
Walker P, S39, S42
Walker S, S6, S35, S150, S152, P7, P184
Walker SA, S18
Walker SP, P85
Wallace C, S146
Wallace G, P255
Wallbanks S, S111
Wallis A, P29
Wallis T, S157, P28
Walrave L, S160, S162
Walsh J, S75
Walsh O, P269
Walsh OR, P272
Walsh S, S15, P69
Walsh SM, M18
Walters GI, S111, S115
Walters R, P243
Wang K, P289
Wang L, S137, S139, P32
Wang M, P247, P249, P250
Wang R, S46, S47, S49, P260, M23
Wang S, S87
Warburton SM, P149
Ward C, S118
Ward E, S2
Ward J, T5, S108
Ward M, P129
Ward TJ, P4
Ward TJC, S176, P13
Wardman H, S49
Wark P, P291
Warner K, P276
Warren S, P175
Warrier A, P83
Waschki B, P4
Wat D, P188
Waters R, P143
Watkinson PJ, S30, P261
Watson A, P234, M10
Watson M, P241
Watson R, S39
Watt M, P10, P14, P61, P200, M21
Watz H, P4
Weatherall M, S52
Webber M, S149
Wedzicha JA, S60, S99, P162, P257
Weinberg SE, P225
Weinman J, P248
Weitnauer L, S71
Welch H, S150
Wells A, P20, P283
Wells AU, S154, P22, P23
Wells C, P145, P146
Wells M, S13, S15
Welz K, P210
Welzenbach K, S4
Wen F, P197, M15
Wensley D, P65
West A, P18
West D, S6
West J, S143
Westley CJ, S79
Westphal B, P135
Wetherall K, S133, M31
Whelan P, M25
Whipday L, S46
Whipday LS, P260
Whitby K, P235
White A, P188
White L, S14, P205, P206
White P, S35, S150, P36
White PT, S12, P33
Whitehouse A, P135, P143
Whiting A, P140
Whittaker L, P142
Whittamore A, P118
Whyte B, P235
Whyte TB, M48
Wickham G, S149
Wickremasinghe M, P254
Wiffen L, S134, P203
Wiggans R, S116
Wiggans RE, S110, S115
Wight A, P174
Wijayaratne T, P82
Wilcock H, S8, S10
Wilcox M, S59
Wildgoose J, S2
Wildman M, M25
Wilkins MR, S143
Wilkinson IB, M10
Wilkinson T, S44, S138, P11, P144, P178, P179, P182
Wilkinson TM, P257
Wilkinson TMA, S7, P68, M22
Wilkinson W, P220
Williams G, P61
Williams N, P254
Williams S, P149
Williams Z, S25
Williamson JP, P210
Willis N, P175
Willis TW, S146
Willmore L, S46, P260
Willshaw S, S5
Wilson M, P50
Wilson T, M5
Wise RA, P297
Wiseman D, S154, P162
Wolf A, P8
Woltmann G, M38, M43
Wong C, S87
Wong E, S130
Wong H, P282
Wong J, P249
Wong T, S99, P215
Woo E, P165
Wood C, P48
Wood J, P223
Wood S, P173
Woodhead T, P231
Woods D, S154
Wootton D, S5
Worrell JC, S63
Wort SJ, S81, S146, P283
Wragg C, S35
Wright A, S12, S172, P4, P159
Wright AKA, S176, P13
Wright B, P131
Wright L, P62
Wu B, P91
Wu Z, S18
Wysoczanski R, M14

Xia C, S101, M13, M14
Xie CY, S144
Xu C, P8
Xu J, P115, P116, P117
Xu X, P192

Yadavilli R, P160
Yamani RY, S106
Yang M, S4
Yang X, S1
Yangannagari SS, P234
Yao H, S91
Yap HY, S78, P56
Yaqoob F, P48
Yardley L, P289
Yasin FF, S151
Yasmeen A, P82
Yasser M, M39
Yates JC, P257
Yazbeck L, S18
Yee B, P165
Yilmaz Z, M10
Yim C, P34, P35
Yioe V, P25, P254
Yip W-H, P23
Young B, S5
Young H, P125
Yousefi P, S117
Yu A, P91
Yuan R, P243
Yung J, P209

Zafar H, P285
Zahid A, P53
Zaidi S, M30
Zambelli E, P268
Zanin O, S108, P244
Zar Mon K, P196
Zarcone M, S87
Zarroug M, P175
Zawieja M, P122
Zeitouni L, S131
Zenner D, M47
Zhang C, S77

# Author index


Zhang E, P250
Zhang J, P78
Zhang L, S160, S164, P199
Zhang X, S38, P238
Zhang Y, P169, P247, P248, P249, P250
Zhang Z, S166
Zhao H, P247, P248
Zhao M, P183
Zheng M, S175, P157, M16
Zhifang Ni M, S124
Zhong M, P246
Zhou ZZ, M1
Zhu C-Q, P201
Zigel Y, P105, P107, P112
Zounemat-Kermani N, T4
Zubair M, P271

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**Professor Mona Bafadhel, Chair, BTS Science and Research Committee**

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