November 2026 Volume 81 Supplement 1

Thorax

AN INTERNATIONAL JOURNAL OF RESPIRATORY MEDICINE

British Thoracic Society Winter Meeting 2026

QEII Centre
Broad Sanctuary
Westminster
London SW1P 3EE

25 to 27 November 2026

Programme and Abstracts

thorax.bmj.com

BMJ Group

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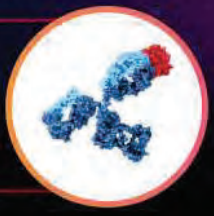
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TEZSPIRE:
THE FIRST AND ONLY
BIOLOGIC TO TARGET TSLP TO REDUCE ALL KEY BIOMARKERS OF AIRWAY INFLAMMATION[1–3]

TSLP acts as an upstream epithelial 'alarm bell' driving multiple downstream inflammatory pathways[2–5]



Blocking TSLP with TEZSPIRE helps provide broad control over the inflammatory pathways that drive the clinical manifestations of severe asthma and CRSwNP[1–3,5–9]



Explore how targeting TSLP may support treatment decisions beyond individual biomarkers in patients with severe asthma, including those with comorbid CRSwNP.

Scan to learn more about the **epithelial science behind TEZSPIRE.**

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APPROVED INDICATIONS:[1]

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.

As an add-on therapy with intranasal corticosteroids for the treatment of adult patients with severe CRSwNP for whom therapy with systemic corticosteroids, and/or surgery do not provide adequate disease control.

CRSwNP, chronic rhinosinusitis with nasal polyps; HCP, healthcare professional; MoA, mechanism of action; TSLP, thymic stromal lymphopoietin.

1. TEZSPIRE (tezepelumab). Summary of Product Characteristics; **2.** Gauvreau GM, *et al. Expert Opin Ther Targets.* 2020;24(8):777–792; **3.** Menzies-Gow A, *et al. Respir Res.* 2020;21(1):268; **4.** Heijink IH, *et al. Clin Exp Allergy.* 2014;44(5):620–630; **5.** Panettieri R Jr, *et al. J Asthma Allergy.* 2024;17:219–236; **6.** Diver S, *et al. Lancet Respir Med.* 2021; 9(11):1299–1312; **7.** Corren J, *et al. Eur Respir J.* 2023;61(3):2202202; **8.** Kaur D, *et al. Chest.* 2012;142(1):76–85; **9.** Luk HG, *et al. Ear Nose Throat J.* 2025:1455613251363018.

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Scan the QR code to access TEZSPIRE (tezepelumab) Prescribing Information

GB-79157 | September 2026
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AstraZeneca

PROGRAMME

AND

ABSTRACTS

Thorax

British Thoracic Society
Winter Meeting 2026

QEII Centre
Broad Sanctuary
Westminster
London SW1P 3EE

25 to 27 November 2026
Programme and Abstracts

Approved by the Federation of the
Royal Colleges of Physicians of the UK
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(6 credits per day).
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Pharmaceutical Sponsors and Other Sponsors

The British Thoracic Society gratefully acknowledges sponsorship from all companies who purchase an exhibition stand at the Winter Meeting. These organisations include pharmaceutical companies.

Sponsorship fees have been used directly to support the delivery of the event including the cost of venue hire, audio-visual, catering, and general conference administration. No sponsorship fees are used towards prizes or social events. None of them have any input into the programme content or the planning of the conference. Furthermore, the Society does not allow any sponsored symposia at this event, within the programme, or associated in any way with it.

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BTS is grateful to AstraZeneca for providing a grant to part-fund the Conference Grants. This organisation has not been involved in establishing the arrangements or the selection criteria for these grants and was not involved in selection.

Directions to the QEII Centre

Location:

QEII Centre
Broad Sanctuary
Westminster
London
SW1P 3EE

Nearest Tube Stations

St Jame's Park (4 mins walk), Westminster (5 mins walk), Victoria Station (14 mins walk)

Car Parking

There are several car parks within walking distance of the Centre, but parking spaces are limited.
Van and Car parking is available at the Q-Park Victoria, which is an 8-minute walk from the Centre.

Thorax 2026;**81**(Suppl 1):Ai–Axcii

Prescribing information and adverse event reporting can be accessed by scanning or clicking on the QR code at the bottom of this page
Promotional content organised and sponsored by Sanofi/Regeneron
For UK healthcare professionals only

DUPIXENT® (dupilumab) Injection 300mg

JOIN SANOFI AND REGENERON AT WINTER BTS MEETING 2026, 25-27 NOVEMBER 2026, QEII CENTRE, LONDON

Visit stand 31-34 to meet the Sanofi and Regeneron team to discover what it takes to build a stronger foundation in COPD



ONLY DUPIXENT ACHIEVED STATISTICAL SIGNIFICANCE ON THE FOLLOWING MEASURES OF TREATMENT SUCCESS FOR PATIENTS WITH UNCONTROLLED TYPE 2-DRIVEN COPD[1-3]



1 REDUCES MODERATE OR SEVERE EXACERBATIONS

In BOREAS, patients receiving DUPIXENT + SOC[a] achieved up to 30% risk reduction vs placebo + SOC (rate ratio 0.70, 95% CI 0.58–0.86; P<0.001) in annualised rate of moderate or severe exacerbations[b,c,3]

2 IMPROVES HOW PATIENTS BREATHE

Lung function
In BOREAS, patients receiving DUPIXENT + SOC achieved up to 160 mL improvement in pre-BD FEV_1 at Week 12 which was sustained to Week 52 vs placebo + SOC (LSM difference 83 mL, 95% CI 42–125; P<0.001)[d,3]

3 IMPROVES HOW PATIENTS FEEL

Symptoms
In BOREAS, DUPIXENT + SOC achieved a −2.7-point improvement in E-RS:COPD score at Week 52 vs −1.6 with placebo + SOC (LSM difference −1.1, 95% CI −1.8 to −0.4; P=0.001)[e,3]

Quality of life
The BOREAS trial demonstrated a −3.4-point difference in SGRQ total score between DUPIXENT + SOC vs placebo + SOC at Week 52 (LSM difference −3.36 [95% CI: −5.46 to −1.27]: P=0.0017)[f,g,3]

DUPIXENT has a generally well tolerated safety profile[1]

The most common adverse reactions in atopic dermatitis, asthma, and CRSwNP are injection-site reactions (including erythema, oedema, pruritus, pain, and swelling), conjunctivitis, conjunctivitis allergic, arthralgia, oral herpes, and eosinophilia. An additional adverse reaction of injection-site bruising has been reported in EoE and COPD. Additional adverse reactions of injection-site induration, injection-site rash, and injection-site dermatitis have been reported in COPD. Rare cases of serum sickness, serum sickness-like reaction, anaphylactic reaction, and ulcerative keratitis have been reported[1]

For additional safety information, please see Summary of Product Characteristics.

INDICATION

DUPIXENT is indicated in adults as add-on maintenance treatment for uncontrolled chronic obstructive pulmonary disease (COPD) characterised by raised blood eosinophils on a combination of an inhaled corticosteroid (ICS), a long-acting $beta_2$-agonist (LABA), and a long-acting muscarinic antagonist (LAMA), or on a combination of a LABA and a LAMA if ICS is not appropriate.[1]

STUDY DESIGN

BOREAS was a Phase 3, multicentre, international, double-blind, randomised, placebo-controlled trial that evaluated DUPIXENT as add-on treatment in patients with uncontrolled COPD. Eligible patients (aged 40–80 years) had physician-diagnosed COPD (≥12 months) with moderate-to-severe airflow limitation; received triple therapy (ICS, LABA, and LAMA, or LAMA and LABA if ICS contraindicated) at baseline; were current/former smokers (≥10 pack-years); had post-BD percent predicted FEV_1 >30% to ≤70%; had type 2 inflammation at screening (blood EOS count ≥300 cells/μL); and had ≥1 moderate or severe acute exacerbation of COPD in the previous year. Eligible patients (n=939) were randomised 1:1 to receive add-on subcutaneous DUPIXENT 300 mg (n=468) or matching placebo (n=471) q2w for 52 weeks while on established background therapy (SOC) with ICS, LABA, and LAMA, or LAMA and LABA if ICS was inappropriate. The primary endpoint was the annualised rate of moderate or severe exacerbations of COPD at 52 weeks (met).[1,3]

AE, adverse event; **BD**, bronchodilator; **CI**, confidence interval; **COPD**, chronic obstructive pulmonary disease; **CRSwNP**, chronic rhinosinusitis with nasal polyps; **EoE**, eosinophilic oesophagitis; **E-RS:COPD**, Evaluating Respiratory Symptoms in Chronic Obstructive Pulmonary Disease; **FEV_1**, forced expiratory volume in 1 second; **ICS**, inhaled corticosteroid; **IL**, interleukin; **LABA**, long-acting $beta_2$-agonist; **LAMA**, long-acting muscarinic antagonist; **LSM**, least-squares mean; **q2w**, once every 2 weeks; **SAE**, serious adverse event; SGRQ, St George's Respiratory Questionnaire; **SOC**, standard of care; **UK**, United Kingdom.

[a]Triple inhaled therapy, or double inhaled therapy if ICS is inappropriate.[1] [b]Moderate exacerbations were defined as exacerbations that resulted in treatment with systemic corticosteroids, antibiotics, or both. Severe exacerbations were defined as exacerbations that resulted in hospitalisation or observation for >24 hours in an emergency department or urgent care facility.[1] [c]Primary endpoint: In BOREAS, the rate was 0.78 (95% CI 0.64–0.93) vs 1.10 (95% CI 0.93–1.30) with placebo + SOC.[3] [d]At Week 12 in the BOREAS trial, patients in the DUPIXENT + SOC arm achieved a 160 mL improvement from BL in pre-BD FEV_1 vs a 77 mL improvement in the placebo + SOC arm (LSM difference vs placebo: 83 mL; 95% CI 42–125; P<0.001). At Week 52 in the BOREAS trial, patients in the DUPIXENT + SOC arm achieved a 153 mL improvement in pre-BD FEV_1 vs a 70 mL improvement in the placebo + SOC arm (LSM difference vs placebo: 83 mL; 95% CI 38–128; secondary endpoint).[3] [e]E-RS:COPD scores range from 0 to 40, with higher scores indicating greater disease severity.[3] [f]BOREAS LSM change in SGRQ total score from BL to Week 52: DUPIXENT + SOC −9.73 vs placebo + SOC −6.37 (LSM difference −3.36; 95% CI −5.46 to −1.27, P=0.0017).[1,3] [g]The SGRQ is a 50-item questionnaire designed to measure and quantify health status in adult patients with chronic airflow limitation. Scores range from 0 to 100, with lower scores indicating a better quality of life; minimum clinically important difference, 4 points.[3]

1. DUPIXENT Summary of Product Characteristics. **2.** National Institute for Health and Care Excellence (NICE). TA1142. Dupilumab for maintenance treatment of uncontrolled chronic obstructive pulmonary disease with raised blood eosinophils. Available at: https://www.nice.org.uk/guidance/TA1142. Accessed October 2026. **3.** Bhatt SP, et al. N Engl J Med. 2023;389:205–214.

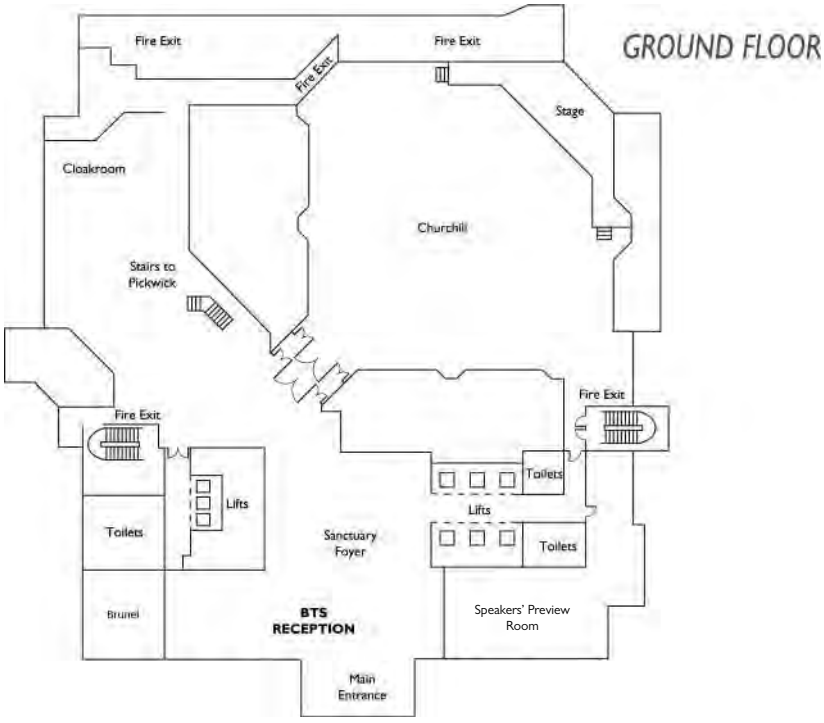
To view the prescribing information for DUPIXENT (dupilumab) please scan or click this QR code

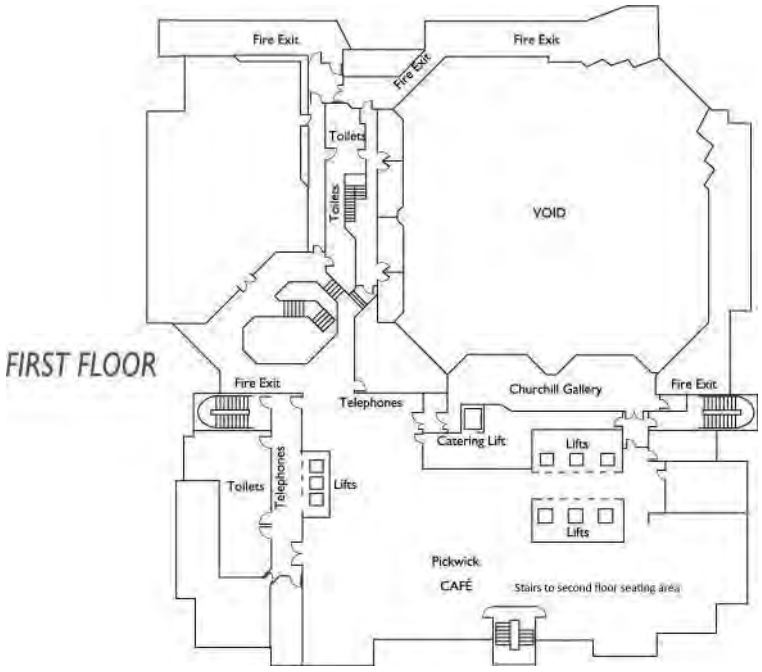
Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store. Adverse events should also be reported to the Sanofi drug safety department on Tel: 0800 0902 314. Alternatively, send via email to UK-drugsafety@sanofi.com

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MAT-XU-2603759 (v1.0) | October 2026

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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 25 and Thursday 26 November and from 8.00am to 2.30pm on Friday 27 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.

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

Inspire

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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]



Effective.[5]	Evidence-based.	Proven.	Credible.
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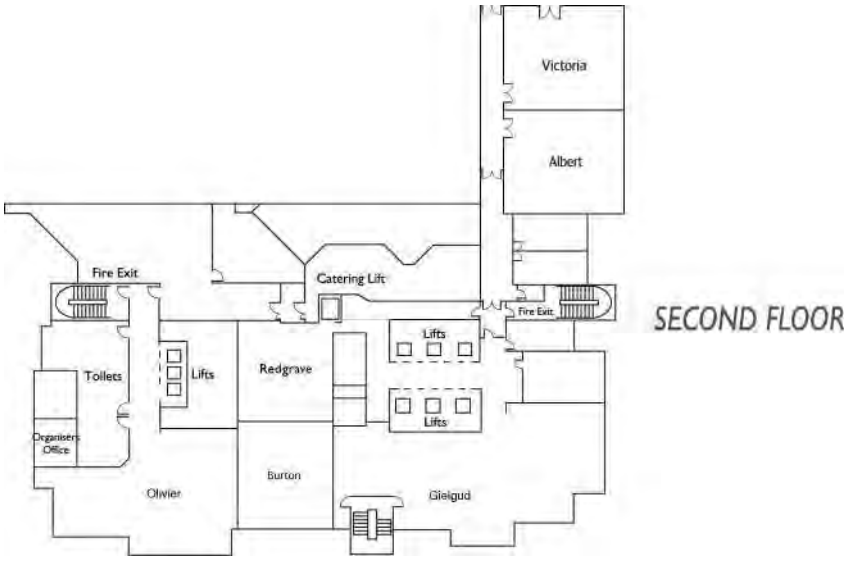
1 Database analysis; Pépin JL, Bailly S, Rinder P et al. J Clin Med. 2021;10(5):936. **2** Strollo et al. NEJM 2015. **3** Woodson BT et al, Otolaryngol Head Neck Surg. 2016. **4** Woodson et al, OtoHNS 2018. **5** Woodson, BT, Strohl, K P, Soose, R J et al. Upper Airway Stimulation for Obstructive Sleep 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.

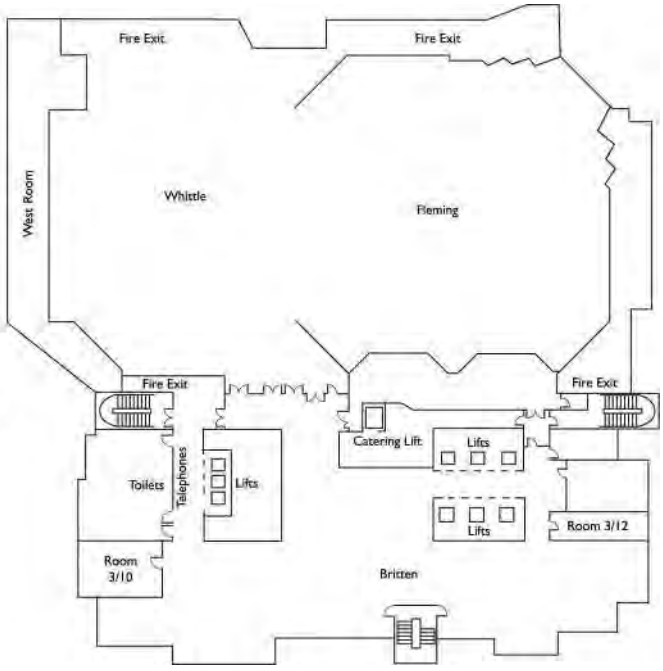
Inspire therapy is not suitable for all patients. Talk to your patients about the risks, benefits and expectations associated with Inspire therapy. Risks associated with the surgical implantation procedure include infections and temporary tongue weakness. In rare cases, tongue paresis and atrophy may occur. After inserting the implant, some patients may need to readjust the settings to improve effectiveness and make it easier to become accustomed to it. Important safety information and product manuals can be found at www.InspireSleep.co.uk/important-safety-information or call 0800 031 80 81.

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The QEII Centre - Second and Third Floors



SECOND FLOOR

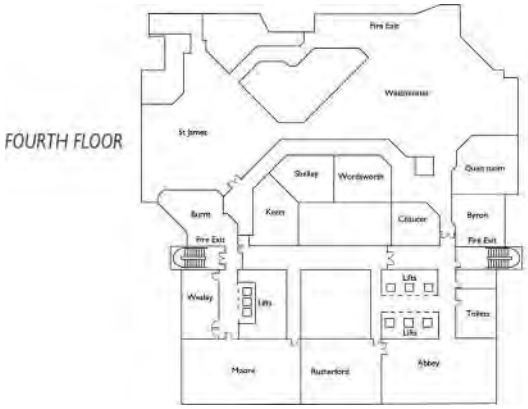


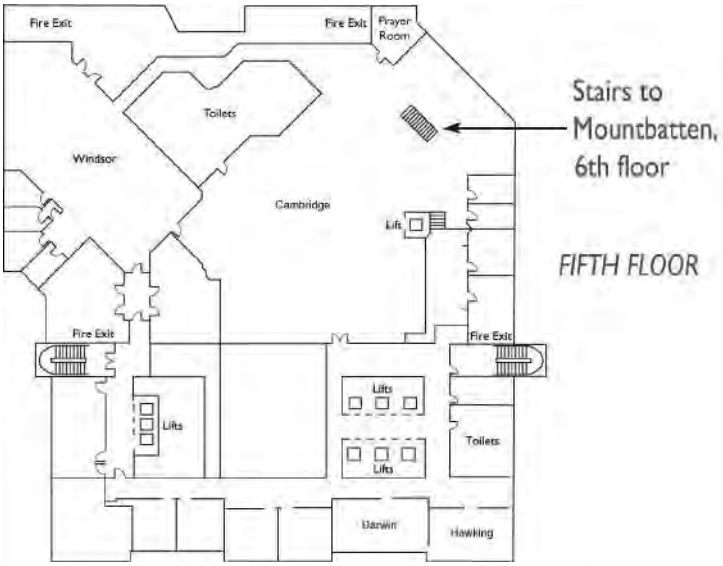
THIRD FLOOR

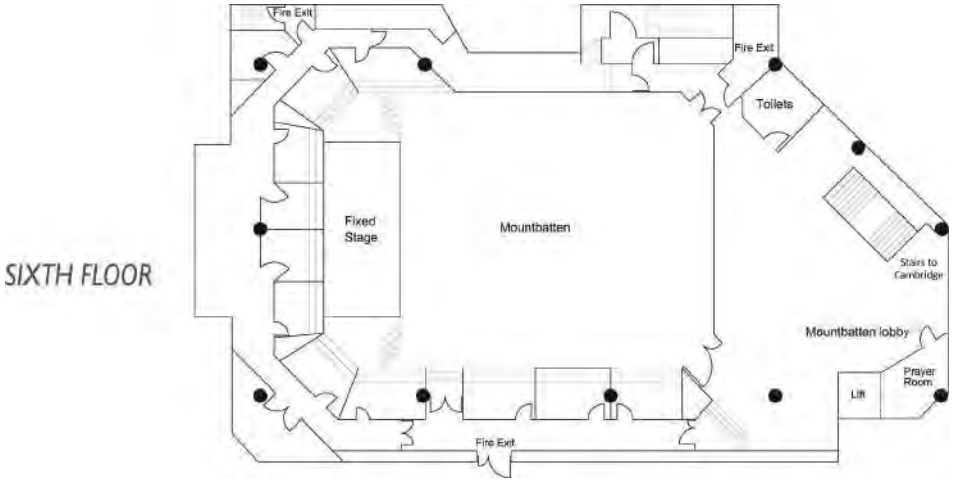
Full café facilities will be open in the Pickwick on the 1st floor from 8.00am to 4.00pm on Wednesday 25 and Thursday 26 November and from 8.00am to 2.30pm on Friday 27 November. A snack bar, serving hot and cold drinks, sandwiches and confectionery only, will be open at the same times in the Whittle & Fleming on the 3rd floor.

Thorax 2026;**81**(Suppl 1):Ai–Axcii

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 25 and Thursday 26 November and from 8.00am to 2.30pm on Friday 27 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 25 NOVEMBER 2026

Time	Details			Location/Floor
8.00am-9.00am	COFFEE/TEA and visit the exhibition			Whittle & Fleming/3rd
8.45am-4.00pm	Poster viewing	P1-P15	Advancing lung cancer diagnostics and treatment	Whittle & Fleming/3rd
10.00am-11.00am	Authors present	P16-P29	Cough clinical profiles and novel interventions	
		P30-P41	Pulmonary vascular disease-from novel therapies to clinical service improvements	
		P42-P56	Breaking barriers-improving lives, transforming access to healthcare	
		P57-P70	TB: from diagnosis to care	
8.45am-4.00pm	Moderated poster viewing	M1-M15	ILD: biomarkers and beyond	Cambridge/5th
8.30am-9.00am	BTS Journal Club		Cough	Albert/2nd
9.00am-10.30am	Joint BTS/BALR Symposium (part I)		Pioneering new frontiers in respiratory research: The Big Data Revolution	Windsor/5th
9.00am-10.30am	Symposium		The changing bronchiectasis landscape: Infection, Biologics and Anti-inflammatories	Churchill/Ground
9.00am-10.30am	Symposium		Asthma going viral	Mountbatten/6th
9.00am-10.30am	Symposium		The environment and workplace exposures: uncovering pathways for chronic respiratory diseases	St James/4th
9.00am-10.35am	Spoken session	S1-S6	Empowering the patient to improve self-management	Westminster/4th
9.00am-10.35am	Spoken session	S7-S12	From RSV prevention to asthma care in childhood	Moore/4th
9.00am-10.35am	Spoken session	S13-S18	Transforming pleural disease: from prediction to patient centred care	Abbey/4th
9.00am-10.35am	Spoken session	S19-S24	COPD in primary care	Rutherford/4th
10.30am-10.45am	Coffee & Tea		Whittle & Fleming and Britten/3rd	
10.45am-12.15pm	Symposium		Targeting COPD: from mechanistics to therapeutics	Churchill/Ground
10.45am-12.15pm	Symposium		UK Transformation Plans	Mountbatten/6th
10.45am-12.15pm	Symposium		Critical Care: what's new in acute respiratory failure?	Windsor/5th
10.50am-12.25pm	Spoken Session	S25-S30	Lung cancer – AI & risk	St James/4th
10.50am-12.25pm	Spoken Session	S31-S36	Rewriting the immune landscape of the lung	Moore/4th
10.50am-12.25pm	Spoken Session	S37-S42	Beyond Type 2 asthma	Abbey/4th
10.50am-12.25pm	Spoken Session	S43-S48	Occupational lung disease – focus on silicosis	Rutherford/4th
11.00am-12.00pm	SAG open meeting		Pulmonary Embolism and other Pulmonary Vascular Diseases	Victoria/2nd
12.00pm-2.15pm	LUNCH	*Not included in the delegate fee. Card payments only*		Pickwick / 1st and Whittle & Fleming/3rd
12.30pm-1.30pm	SAG open meeting		Cough	Albert/2nd
12.45pm-1.45pm	SAG open meeting		TB and Non-Tuberculous Mycobacteria	Victoria/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).

Time	Details			Location/Floor
1.15pm-2.00pm	BTS Scientific Guest Lecture		Mediating best immune responses: which is greatest the nose or the lung?	Churchill/Ground
2.00pm-3.00pm	SAG open meeting		Occupational and Environmental Lung Disease	Albert/2nd
2.00pm-3.00pm	SAG open meeting		Bronchiectasis	Victoria/2nd
2.15pm-4.15pm	Moderated Poster discussion	M1-M16	ILD: biomarkers and beyond	Cambridge/5th
2.15pm-3.45pm	Symposium		The Mike Morgan Legacy - Prioritising Respiratory Care & Delivering the NHS Long Term Plan	Churchill/Ground
2.15pm-3.45pm	Joint BTS / BPRS Symposium		Breathing from the Start: The Impact of Air Pollution on Respiratory Health from Birth to Adolescence	Mountbatten/6th
2.15pm-3.45pm	Award 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	Advancing lung cancer diagnostics and treatment	St James/4th
2.15pm-4.00pm	Poster discussion	P16-P29	Cough clinical profiles and novel interventions	Westminster/4th
2.15pm-4.00pm	Poster discussion	P30-P41	Pulmonary vascular disease-from novel therapies to clinical service improvements	Moore/4th
2.15pm-4.00pm	Poster discussion	P42-P56	Breaking barriers-improving lives, transforming access to healthcare	Abbey/4th
2.15pm-3.50pm	Poster discussion	P57-P70	TB: from diagnosis to care	Rutherford/4th
3.00pm-4.30pm	Coffee & Tea	Whittle & Fleming and Britten/3rd		
3.15pm-4.15pm	SAG open meeting		Tobacco Dependency	Gielgud/2nd
3.15pm-4.15pm	SAG open meeting		Critical Care, Respiratory Failure and Mechanical Ventilation	Albert/2nd
4.15pm-4.45pm	Award presentations			Churchill/Ground
4.45pm-5.30pm	The BTS President's Address		Education and Networking: The Power to Shape the Future	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 26 NOVEMBER 2026

Time	Details			Location/Floor
8.00am-9.00am	COFFEE/TEA			Whittle & Fleming/3rd
8.00am-9.00am	SAG open meeting		Pharmacist	Gielgud/2nd
8.45am-4.00pm	Poster viewing	P71-P85	Biologics galore!	Whittle & Fleming/3rd
10.00am-11.00am	Authors present	P86-P100	Respiratory infections: from disease burden to outcomes	
		P101-P109	Global and public health: future aspects with impact	
		P110-P122	The breath of childhood: science, innovation and care	
		P123-P137	Asthma or something else?	
		P138-P152	Novel approaches to improving COPD care: imaging, AI and acute intervention	
		P153-P167	ILD: cohort studies and service improvement	
		P168-P180	Bronchiectasis unconfined	
8.45am-4.00pm	Moderated poster viewing	M16-M31	Real world pleural medicine: improving care outcomes, training and delivery	Cambridge/5th
8.30am-9.00am	BTS Journal Club		Pulmonary Embolism	Victoria/2nd
9.00am -10.30am	Symposium		Lung cancer in never smokers; how can we identify those at highest risk?	Churchill/Ground
9.00am -10.30am	Symposium		Evolving practice in the delivery of pulmonary rehabilitation: where next?	Mountbatten/6th
9.00am -10.30am	Joint BTS/BALR Symposium (part 2)		Pioneering new frontiers in respiratory research: The Big Data Revolution	Windsor/5th
9.00am-10.35am	Spoken session	S49-S54	Next generation asthma care	St James/4th
9.00am-10.35am	Spoken session	S55-S60	Bronchiectasis: biology, biomarkers and better care	Westminster/4th
9.00am-10.35am	Spoken session	S61-S66	ILD: risk prediction	Moore/4th
9.00am-10.35am	Spoken session	S67-S72	Paediatric respiratory medicine: vaccines, asthma and innovation	Abbey/4th
9.00am-10.35am	Spoken session	S73-S78	Sleepy futures: from hospital to home	Rutherford/4th
9.00am-10.35am	Spoken session	S79-S84	Advances in cystic fibrosis	Albert/2nd
9.30am-10.15am	Open meeting		Breathlessness	Victoria/2nd h
10.00am-11.00am	COFFEE/TEA	Whittle & Fleming and Britten/3rd		
10.50am-11.50am	SAG open meeting		Asthma	Abbey/4th
10.50am-11.50am	SAG open meeting		Pleural Disease	Albert/2nd
10.50am-11.50am	SAG open meeting		Sleep	Victoria/2nd
10.45am-12.20pm	Spoken session	S85-S90	Randomised Controlled Trials	Windsor/5th
10.45am-12.30pm	Symposium		Plenary scientific symposium	Churchill/Ground
12.00pm-2.15pm	LUNCH	*Not included in the delegate fee. Card payments only*		Pickwick / 1st and Whittle & Fleming/3rd
1.00pm-1.45pm	The BTS Clinical Lecture		Timing is Everything: What HIV has taught us about when to treat	Churchill/Ground
1.00pm-2.00pm	SAG open meeting		ARTP / BTS Respiratory Physiology	Albert/2nd
2.15pm-3.45pm	Symposium		Breathing Danger: COPD beyond Tobacco	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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Time	Details			Location/Floor
2.15pm-3.45pm	Symposium		Highlights from Thorax/Lancet Respiratory Medicine	Mountbatten/6th
2.15pm-3.45pm	Symposium		Joint BTS/A+LUK/BALR Mid-Career Lectures	Windsor/5th
2.15pm-3.50pm	Spoken session	S91-S96	From the old to the new: innovative phycological assessments	St James/4th
2.15pm-4.00pm	Moderated Poster discussion	M16-M31	Real world pleural medicine: improving care outcomes, training and delivery	Cambridge/5th
2.15pm-4.05pm	Poster discussion	P71-P85	Biologics galore!	Westminster/4th
2.15pm-4.05pm	Poster discussion	P86-P100	Respiratory infections: from disease burden to outcomes	Moore/4th
2.15pm-3.20pm	Poster discussion	P101-P109	Global and public health: future aspects with impact	Abbey/4th
2.15pm-3.50pm	Poster discussion	P110-P122	The breath of childhood: science, innovation and care	Rutherford/4th
2.00pm-3.00pm	SAG open meeting		Lung Cancer and Mesothelioma	Victoria/2nd
3.00pm-4.30pm	Coffee & Tea			Whittle & Fleming and Britten/3rd
4.15pm-5.15pm	SAG open meeting		Global Lung Health (Task and Finish Group)	Victoria/2nd
4.15pm-5.45pm	Symposium		Malignant pleural effusion-from biology to outcomes	Churchill/Ground
4.15pm-5.45pm	Joint BTS BPRS Symposium		Smart Lungs, Smart Care: The Present and Future of Digital Health in Paediatric Respiratory Disease	Mountbatten/6th
4.15pm-5.45pm	Symposium		The physiology of dyspnoea and why patients feel what they feel	Windsor/5th
4.15pm-5.50pm	Symposium	S97-S102	More than a barrier: the airway epithelium	St James/4th
4.15pm-5.50pm	Spoken session	S103-S108	Lung infection: that's just (a)typical	Westminster/4th
4.15pm-6.00pm	Poster discussion	P123-P137	Asthma or something else?	Moore/4th
4.15pm-6.00pm	Poster discussion	P138-P152	Novel approaches to improving COPD care: imaging, AI and acute intervention	Abbey/4th
4.15pm-6.00pm	Poster discussion	P153-P167	ILD: cohort studies and service improvement	Rutherford/4th
4.15pm-5.50pm	Poster discussion	P168-P180	Bronchiectasis unconfined	Albert/2nd
5.45pm-7.00pm	The President's Reception-All welcome!			Britten/3rd

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

FRIDAY 27 NOVEMBER 2026

Time	Details			Location/Floor
8.00am-9.00am	COFFEE/TEA and visit the exhibition			Whittle & Fleming/3rd
8.45am-2.00pm	Poster viewing	P181-P195	Innovation across the lung cancer diagnostic pathway	Whittle & Fleming/3rd
10.00am-11.00am	Authors present	P196-P211	Ventilation: frailty and comorbidity in patients with respiratory failure	
		P212-P221	Opportunities to improve asthma care	
		P222-P229	Advances in thoracic surgery and tobacco harm reduction	
		P230-P244	Improving every breath: pulmonary rehabilitation advances	
		P245-P259	Asthma in transition	
		P260-P272	Type 2 COPD: biomarkers, biologics and outcomes	
		P273-P284	Advances in translational research	
8.45am-5.15pm	Moderated poster viewing	M32-M47	The future of sleep and physiological services	Cambridge/5th
8.30am-9.00am	BTS Journal Club		NTM	Albert/2nd
9.00am-10.30am	Symposium		Finding your niche: Cellular heterogeneity in asthma	Churchill/Ground
9.00am-10.30am	Symposium		When autoimmunity targets the lung	Mountbatten/6th
9.00am-10.30am	Symposium		New Insights from Infection to Inflammation	Windsor/5th
9.00am-10.35am	Spoken session	S109-S114	Decoding lung disease through 'omics	Westminster/4th
9.00am-10.35am	Spoken session	S115-S120	Focused pleural medicine: advances in diagnostic and therapeutics	Moore/4th
9.00am-10.35am	Spoken session	S121-S126	NIV: why outcomes matter	Abbey/4th
9.00am - 10.35am	Open meeting		Women in respiratory	St James/4th
9.00am-10.00am	SAG open meeting		Nurse	Rutherford/4th
9.15am-10.15am	SAG open meeting		Pulmonary Rehabilitation	Albert/2nd
9.00am-10.00am	SAG open meeting		Specialty Trainee	Victoria/2nd
10.00am-11.00am	COFFEE/TEA and visit the exhibition			Whittle & Fleming and Britten/3rd
10.30am-11.30am	SAG open meeting		Acute and Complex Pulmonary Infections	Rutherford/4th
10.30am-11.30am	SAG open meeting		COPD	Gielgud/2nd
10.45am-12.15pm	Symposium		Translation in pleural disease-improving understanding and treatments	Churchill/Ground
10.45am-12.15pm	Symposium		The use of physiological data within specialist clinics	Mountbatten/6th
10.45am-12.15pm	Symposium		BTS Audit & Quality Improvement	Windsor/5th
10.45am-12.20pm	Spoken session	S127-S132	Eosinophilic exacerbations: what do we do?	St James/4th
10.45am-12.20pm	Spoken session	S133-S138	ILDS: ILAS and familial fibrosis	Westminster/4th
10.45am-12.20pm	Spoken session	S139-S144	Pulmonary vasculature: aims and imaging	Moore/4th
10.45am-12.20pm	Spoken session	S145-S150	From defence to damage: the phagocyte	Abbey/4th

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DAILY PROGRAMME (cont.)

FRIDAY 27 NOVEMBER 2026

Time	Details			Location/Floor
12.00pm-2.00pm	LUNCH	*Not included in the delegate fee. Card payments only. Exhibition closes at 2.00pm*		Pickwick / 1st and Whittle & Fleming/3rd
12.45pm-1.30pm	BTS Grand Challenge Guest Lecture		Why this Bill matters: Tobacco, Vaping and the future of Lung Health	Churchill/Ground
1.30pm-3.30pm	Poster discussion	P181-P195	Innovation across the lung cancer diagnostic pathway	St James/4th
1.30pm-3.35pm	Poster discussion	P196-P211	Ventilation: frailty and comorbidity in patients with respiratory failure	Westminster/4th
1.30pm-3.15pm	Poster discussion	P212-P221	Opportunities to improve asthma care	Moore/4th
1.30pm-2.35pm	Poster discussion	P222-P229	Advances in thoracic surgery and tobacco harm reduction	Abbey/4th
1.30pm-3.15pm	Poster discussion	P230-P244	Improving every breath: pulmonary rehabilitation advances	Rutherford/4th
1.45pm-3.15pm	Symposium		Optimising sleep: future directions for treating sleep disordered breathing	Churchill/Ground
1.45pm-3.15pm	Symposium		Is there a role for steroids in CAP?	Mountbatten/6th
1.45pm-3.15pm	Symposium		Cross-disease approaches to treating the onset, exacerbation and progression of chronic lung diseases	Windsor/5th
1.45pm-2.45pm	SAG open meeting		Cystic Fibrosis	Albert/2nd
1.45pm-2.45pm	SAG open meeting		Interstitial and Rare Lung Disease	Victoria/2nd
3.00pm-3.30pm	COFFEE/TEA			Britten/3rd
3.30pm-5.00pm	Symposium		Lung cancer survivorship: Recurrence risk, Second primaries & chemoprevention	Churchill/Ground
3.15pm-5.05pm	Spoken session	S151-S156	The fibrotic niche: epithelial fibroblast interactions	Windsor/5th
3.15pm-5.00pm	Moderated Poster discussion	M32-M47	The future of sleep and physiological services	Cambridge/5th
3.45pm-5.20pm	Poster discussion	P245-P259	Asthma in transition	St James/4th
3.45pm-5.10pm	Poster discussion	P260-P272	Type 2 COPD: biomarkers, biologics and outcomes	Westminster/4th
3.30pm-5.00pm	Poster discussion	P273-P284	Advances in translational research	Moore/4th

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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 25 NOVEMBER

Time	SAG	Location
11.00am-12.00pm	Pulmonary Embolism and other Pulmonary Vascular Diseases	Victoria, 2nd floor
12.30pm-1.30pm	Cough	Albert, 2nd floor
12.30pm-1.30pm	TB and Non-Tuberculous Mycobacteria	Victoria, 2nd floor
2.00pm-3.00pm	Occupational and Environmental Lung Disease	Albert, 2nd floor
2.00pm-3.00pm	Bronchiectasis	Victoria, 2nd floor
3.15pm-4.10pm	Critical Care, Respiratory Failure and Mechanical Ventilation	Albert, 2nd floor
3.15pm-4.10pm	Tobacco Dependency	Gielgud, 2nd floor

THURSDAY 26 NOVEMBER

Time	SAG	Location
8.00am-9.00am	Pharmacist	Gielgud, 2nd floor
10.50am-11.50am	Asthma	Abbey, 4th floor
10.50am-11.50am	Pleural Disease	Albert, 2nd floor
10.50am-11.50am	Sleep	Victoria, 2nd floor
1.00pm-2.00pm	BTS/ARTP-Physiology	Albert, 2nd floor
2.00pm-3.00pm	Lung Cancer and Mesothelioma	Victoria, 2nd floor
4.15pm-5.15pm	Global Lung Health	Victoria, 2nd floor

FRIDAY 27 NOVEMBER

Time	SAG	Location
9.00am-10.00am	Nurse	Rutherford, 4th floor
9.15am-10.15am	Pulmonary Rehabilitation	Albert, 2nd floor
9.00am-10.00am	Specialty Trainee	Victoria, 2nd floor
10.30am-11.30am	Acute and Complex Pulmonary Infections	Rutherford, 4th floor
10.30am-11.30pm	COPD	Gielgud, 2nd floor
1.45pm-2.45pm	Cystic Fibrosis	Albert, 2nd floor
1.45pm-2.45pm	Interstitial and Rare Lung Disease	Victoria, 2nd floor

BTS AWARD PRESENTATIONS

Wednesday 25 November at 4.15pm in the Churchill, Ground floor

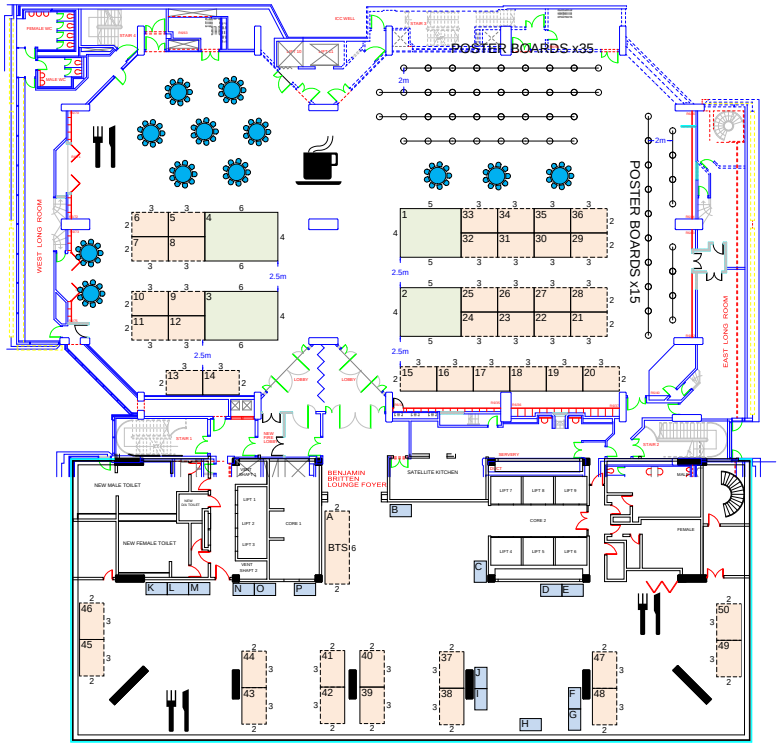
The presentations will be made just before the BTS President's Address, for the annual BTS Medal, BTS President's Award, BTS Award for Meritorious Service, BTS/BALR/A+LUK Early Career Investigator of the Year Awards, BTS Medical Student Awards and BTS/A+LUK/BALR Mid-Career Lecture Awards. Please come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

THE BTS PRESIDENT'S RECEPTION

Thursday 26 November from 5.45pm to 7.00pm in the Britten, 3rd floor

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

FLOOR PLAN OF THE EXHIBITION STANDS



Exhibitors and stand numbers

30	Aerogen
29	APR Medtech
1 & 4	AstraZenaca UK
9,10,11 & 12	Boehringer Ingelheim
5	Celltrion
2	Chiesi
14	Creo Medical
36	Diagenics
8	Erbe
13	Essential Pharma
18	Fannin UK
24	Fisher & Paykel Healthcare
3, 37 & 38	GSK
17	HealthNet Homecare
46	Idorsia Pharmaceuticals UK
19, 21 & 22	Insmed
16	Inspire
7	It's Interventional
47	Merck
6	NeilMed
40	NIOX
23	Olympus Medical
15	Orion Pharma (UK) Ltd
27	Pfizer Medical
41	Pharming
20	Rocket Medical
25, 26, 31, 32, 33 & 34	Sanofi
35	Sterling Anglian
28	Tidal
44	Vitalograph
39	Zoll Medical

Stand Reference Charity / non-commercial organisation

D	Action for Pulmonary Fibrosis
J	Association for Respiratory Technology & Physiology (ARTP)
F	Association of Chartered Physiotherapists in Respiratory Care (ACPRC)
I	Association of Respiratory Nurses (ARNS)
O	Asthma + Lung UK
G	British Association of Lung Research (BALR)
N	British Society for Allergy and Clinical Immunology
E	Health Innovation Oxford
P	Interstitial Lung Disease Interdisciplinary Network
K	Mesothelioma UK
B	National Respiratory Audit Programme (NRAP)
M	NTM Patient Care
C	Pulmonary Rehabilitation Services Accreditation Scheme (PRSAS)
H	Royal College of Speech and Language Therapists
L	SarcoidosisUK
Q	Welsh Thoracic Society

SCIENTIFIC PROGRAMME

8.00am – 9.00am
Whittle & Fleming, 3rd floor
COFFEE/TEA

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

P1-P15
Advancing lung cancer diagnostics and treatment
Discussion of abstracts will take place from 2.15pm to 4.05pm in the St James, 4th floor

P16-P29
Cough clinical profiles and novel interventions
Discussion of abstracts will take place from 2.15pm to 4.05pm in the Westminster, 4th floor

P30-P41
Pulmonary vascular disease – from novel therapies to clinical service improvements
Discussion of abstracts will take place from 2.15pm to 4.00pm in the Moore, 4th floor

P42-P56
Breaking barriers – improving lives, transforming access to healthcare
Discussion of abstracts will take place from 2.15pm to 4.00pm in the Abbey, 4th floor

P57-P70
TB: from diagnosis to care
Discussion of abstracts will take place from 2.15pm to 3.50pm in the Rutherford, 4th floor

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

M1-M16
ILD: biomarkers and beyond
Discussion of abstracts will take place from 2.15pm to 4.00pm in the Cambridge, 5th floor

8.30am – 9.00am
Albert, 2nd floor
JOURNAL CLUB
COUGH

Dr Barnaby Hirons (London)

Learning objective

To review the latest publications and evidence in the field of cough.

Wednesday 25 November 2026

9.00am – 10.30am
Churchill, Ground floor
SYMPOSIUM
THE CHANGING BRONCHIECTASIS LANDSCAPE: INFECTION, BIOLOGICS AND ANTI-INFLAMMATORIES

Chaired by: Professor Melissa McDonnell (Galway) and Dr Phil Mitchelmore (Exeter)

09.00 Microbial Mavericks: Next-Gen science of the bronchiectasis microbiome
Professor Sanjay Chotirmall (Singapore)

09.30 Monoclonal antibodies to target Pseudomonas aeruginosa: What have we learnt from the GREAT trials and mechanistic studies?
Dr Merete Long (Oxford)

10.00 Targeting Type 2 inflammation in Bronchiectasis: Lessons from MAHALE
Professor Stefano Aliberti (Italy)

Learning objectives:

1) Understanding of translational science and mycobiome/ resistome and microbiome. Novel data on gut-lung axis resistome in bronchiectasis

2) New study data on pseudomonas virulence and targeting in bronchiectasis using non-antibiotics. New data on bacterial clearance and patient quality of life using anti-microbial monoclonal antibodies

3) Understand study design and limitations / strengths and role of biologics to date in bronchiectasis. Data from clinical trials (not presented before) on biologics in bronchiectasis will be discussed

9.00am – 10.30am
Mountbatten, 6th floor
SYMPOSIUM
ASTHMA GOING VIRAL

Chaired by: Professor Melissa McDonnell (Galway) and Dr Phil Mitchelmore (Exeter)

09.00 Microbial Mavericks: Next-Gen science of the bronchiectasis microbiome
Professor Sanjay Chotirmall (Singapore)

09.30 Monoclonal antibodies to target Pseudomonas aeruginosa: What have we learnt from the GREAT trials and mechanistic studies?
Dr Merete Long (Oxford)

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3) Understand study design and limitations / strengths and role of biologics to date in bronchiectasis. Data from clinical trials (not presented before) on biologics in bronchiectasis will be discussed

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9.00am – 10.30am
Windsor, 5th floor
JOINT BTS/BALR SYMPOSIUM (Part 1)

PIONEERING NEW FRONTIERS IN RESPIRATORY RESEARCH: THE BIG DATA REVOLUTION

Chaired by: Dr Manuela Plate (London) and Dr Kirsty McGee (Birmingham)

09.00 The power of data integration to progress medical research: lessons from outside the lung
Professor John Terry (Plymouth)

09.30 Applying AI and deep learning to detect lung disease
Professor Liangxiu Han (Manchester)

10.00 Single cell spatial biology reveals early immune events in lung disease
Professor Ling-Pei Ho (Oxford)

Learning objectives:

1) Understand the power of data integration across technologies with mathematical modelling to progress translational medical research

2) Recognise the power of AI and deep learning applications in the diagnosis, monitoring and prediction of chronic respiratory diseases

3) Understand how we use new big data techniques in discovering what matters for patients – defining early immune events means possible therapeutic targets

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9.00am – 10.30am
St James, 4th floor
SYMPOSIUM

THE ENVIRONMENT AND WORKPLACE EXPOSURES: UNCOVERING PATHWAYS FOR CHRONIC RESPIRATORY DISEASES

Chaired by: Dr Huda Badri (Manchester) and Dr Clare Burton (Sheffield)

09.00 Emerging epidemiology of asthma related to work
Orianne Dumas (France)

09.30 From exposure science to case finding in silica-related lung disease
Professor David Fishwick (Sheffield)

10.00 The air we share: short and long-term impacts of air quality on airway diseases
Anna Hansell (Leicester)

Learning objectives:

1) Understand how genetic susceptibility interacts with airborne occupational and environmental exposures to influence risk and outcomes for respiratory disease, including lung cancer and COPD

2) Examine current epidemiological and mechanistic evidence linking indoor and outdoor air quality with chronic airway disorders

3) Explore how exposure science and real-world surveillance are driving change in how we detect, regulate and prevent silica-related lung disease

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9.00am – 10.35am
Westminster, 4th floor
SPOKEN SESSION: S1-S6

Empowering the patient to improve self-management

Chaired by: Dr Natalie Harper and Professor Anna Murphy

9.05am **S1**
Accelerometry-based behavioural phenotypes at five months following hospitalisation for COVID-19 and their associations with health outcomes and recovery at one year
L Wang, RA Evans, C Lawson, TE Yates, K Ntotsis

9.20am **S2**
Home-based body-weight exercise with blood flow restriction in COPD: a randomised feasibility trial
HA Aljohani, LE Latimer, M Baldwin, K Balasundaram, M Fong, NJ Greening, MR Lindley, L Bishop, MC Steiner, TJC Ward, RA Ferguson

9.35am **S3**
Co-producing multilingual breathlessness self-management resources with ethnically diverse communities: early reach and acceptability following national dissemination
Sabrina Bajwah

9.50am **S4**
Why don't more patients manage their own indwelling pleural catheter (IPC)? Barriers and facilitators to IPC self-management.
AM Peel, EM Mishra, S Markham, MC Farquhar

10.05am **S5**
Implementation of a self-referral chest X-ray pathway for cancer detection: early outcomes from a pilot programme.
S Roy, E Parikh, S Foster

10.20am **S6**
Supported "rescue packs" post-discharge for people living with COPD – qualitative interview study
K Kuberska, G Martin, JR Hurst, Mona Bafadhel

9.00am 10.35am
Moore, 4th Floor
SPOKEN SESSION: S7 S11

From RSV prevention to asthma care in childhood

Chaired by: Dr Samantha Sonnappa and Professor Sejal Saglani

9.05am **S7**
Optimising the asthma diagnostic pathway in children and young people – preliminary results from the RADicA study
R Wang, M Bennett, E Barrett, A Simpson, L Healy, S Drake, SJ Fowler, CS Murray

Wednesday 25 November 2026

9.20am **S8**
Evaluating the causal effect of early-life phenotypic traits on lung function trajectory: a Mendelian randomisation study in the Avon Longitudinal Study of Parents and Children (ALSPAC)
J S Grenville, R C Richmond, G M Power, P Yousefi, J W Dodd

9.35am **S9**
Association of preschool wheeze endotypes with wheeze attacks and ICS treatment
A Perikleous, SJ Bowen, C Griffiths, I Pavord, M Rosenthal, L Fleming, A Bush

9.50am **S10**
Outcome of recurrent Preschool Wheeze: Does the season during first wheeze episode matter?
Jui Andharia, Lubna Wajid, Sejal Saglani, Prasad Nagakumar

10.05am **S11**
Real-World Outcomes Following MART Initiation in Paediatric Asthma: A Single-Centre Service Evaluation
SHS Cheng, S Harper, LH Everitt, A Bothra, GJ Connett

9.00am 10.35am
Abbey, 4th Floor
SPOKEN SESSION: S13 S18

Transforming pleural disease – from prediction to patient centred care

Chaired by: Dr Beenish Iqbal and Dr Mohammed Haris

9.05am **S13**
PLUMBing less or more. A mixed-method, multicentre prospective study to understand the management basis of pneumothorax after lung biopsy in the UK (The PLUMB study)
B Iqbal, J Heaton, A Bowes, A Aujayeb, H Bellenberg, C Ross, U Khatana, M Khan, F Altayeb, M Shackcloth, A Baguneid, R Sudhir, R Panchal, G Barton, AT Butt, C Mizzi, B Dharmarajh, A Davies, A Lithgow, J Cheng, E Mishra, B Al-Harbawi, M Haris, S Bhatkal, R Hutchison, G Warwick, B Pippard, A Stanton, S Ibrahim, D De-Fonseka,

C Aday, K Conroy, B Prudon, A Elsheikh, D Addala, E Bedawi, N Rahman, R Hallifax

9.20am **S14**

Real-World Management and Outcomes of Secondary Spontaneous Pneumothorax: An International Multicentre retrospective analysis on Persistent Air LEak in spontaneous pneumothorax (IMPALE)

P Naran, A Aujayeb, J Shaw, S Walker, C Ross

9.35am **S15**

The Risk of Pneumothorax in Cannabis Smokers: A Case-Control Study

L Richman, C Bridge, K Wanniarachchi, S Westwater, M D Hunyadi, P Pharoah, S Marciniak

9.50am **S16**

Wearable-Derived Activity Monitoring Reveals Distinct Recovery Trajectories Following Pleural Intervention in Malignant Pleural Effusion

ORW Walsh, EA Elsheikh, MB Bhatnaghar, DAR Abdul-Rahman, EOB Bedawi, NR Rahman

10.05am **S17**

Longitudinal Improvement in Health-Related Quality of Life Following Pleural Intervention for Malignant Pleural Effusion

DTB Beith, SP Pyne, EKM Mishra

10.20am **S18**

The association of baseline platelet count with clinical outcomes and septation burden in pleural infection: analysis of the PILOT cohort

FA Al-Arrayed, AE Elsheikh, MB Bhatnagar, OW Walsh, DAR Abdul-Rahman, NMR Rahman, EOB Bedawi

9.00am 10.35am
Rutherford, 4th Floor
SPOKEN SESSION: S19 S23

COPD in primary care

Chaired by: Dr Amy Dewar and Dr Stephen Holmes

9.05am **S19**

"Respiratory just isn't sexy": clinicians' experiences of spirometry for COPD diagnosis in UK primary care

C Roleston, M Glogowska, J Wolstenholme, R Koleva-Kolarova, G Hayward, P Turner, HF Ashdown

9.20am **S20**

Spirometry provision for COPD diagnosis across the UK: findings from a national survey of healthcare professionals

C Roleston, M Glogowska, J Wolstenholme, R Koleva-Kolarova, G Hayward, P Turner, HF Ashdown

9.35am **S21**

Standby Rescue Pack Prescribing Creates Substantial Under-Recording of COPD Exacerbations in Primary Care Electronic Health Records

A O Howell, A Schaffer, M Wanat, H Pinnock, M Bafadhel

9.50am **S22**

Prevalence and clinical characteristics of patients with COPD who continue to exacerbate despite triple therapy in England: the SIRIUS-UK study

M Bafadhel, M J Hale, M Watt, A Platonova, R Zhang, C Morgan, V Pradeep, D Halpin, R E K Russell

10.05am **S23**

Beyond-FRONTIER: Utilising Hull Lung Health Study data to investigate the diagnostic accuracy of N-Tidal Diagnose Diagnostic algorithms to identify COPD and pre-COPD among participants in the FRONTIER programme.

MG Crooks, K Brindle, K Watkins, L Pitel, B Proctor, RH Lim, L Talker, G Lambert, AX Patel, S Niaz-Ali, R Malone

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

COFFEE/TEA BREAK

10.45am – 12.15pm
Churchill, Ground floor
SYMPOSIUM

TARGETING COPD: FROM MECHANISTICS TO THERAPEUTICS

Chaired by: Dr Neil Greening (Leicester) and Dr Nicola Smallcombe (London)

10.45 From mechanism to medicine: pathways for targeted therapy in COPD
Professor Francesca Polverino (USA)

11.15 Should we focus on alarmins in COPD treatment?
Professor Chris Brightling (Leicester)

11.45 Biologics in COPD: what the new BTS clinical statement means for practice
Dr Anthony Martinelli (Cambridge)

Learning objectives:

1) To understand how advances in COPD pathophysiology are shaping the development of targeted therapies

2) To assess the potential of alarmins as therapeutic targets in COPD

3) To review how the latest BTS guidance informs the practical use of biologics in COPD care

10.45am – 12.15pm
Mountbatten, 6th floor
SYMPOSIUM

UK TRANSFORMATION PLANS

Chaired by: Claire Hurlin (Wales) and Professor Tom Fardon (Scotland)

10.45 Respiratory Reset in Northern Ireland
Dr Nazia Chaudhuri (Ulster)

11.00 Community diagnostic centres
Dr Martin Allen (Stoke on Trent)

11.15 Outpatient transformation across England
Sleep – Dr Alanna Hare (London)
Breathlessness – Dr Helen Ward (Wolverhampton)
Cough – Dr Sean Parker (Northumbria) / Claire Slinger (Preston)

10.45am – 12.15pm
Windsor, 5th floor
SYMPOSIUM

WHAT'S NEW IN ACUTE RESPIRATORY FAILURE?

Chaired by: Dr Ben Messer (Newcastle) and Ema Swingwood (Bristol)

10.45 Novel therapeutic agents in ARDS
Professor Daniel McAuley (Belfast)

11.15 Biomarker-Driven Stratification and Management for Sepsis in Critical Care
Professor Manu Shanker-Hari (London)

11.45 Beyond Survival: Airway Clearance and Recovery
Professor Bronwen Connolly (Belfast)

Learning objectives:

1) Describe major pathways of lung repair and maladaptive remodelling, and how these can lead to persistent dysfunction or fibrosis after injury

2) Discuss the latest in evidence-based precision medicine in critical care

3) The role of chest physiotherapy, mechanical insufflation: exsufflation and pharmacotherapy

10.50am 12.25pm
St James, 4th Floor
SPOKEN SESSION: S25 S30

Lung cancer – AI & risk

Chaired by: Dr Ambika Talwar and Professor Neal Navani

10.55am **S25**
Imaging-based biological age estimation predicts competing mortality risk in lung cancer screening
DO Cheng, J McCabe, T Patrick, R Chapman, S Wang, T Yang, A Szmul, M Azimbagirad, S Aslani, ML Mullin, SB Naidu, CR Khaw, ACP So, N Navani, A Nair, JR Hurst, A Altmann, A Pennycuick, DC Alexander, NP Oxtoby, AL Young, M Sperrin, SM Janes, J Jacob

11.10am **S26**
Personalising Lung Cancer Screening Eligibility Reassessments Based On Real-world Longitudinal Risk Trajectories.
A Leliveld, D.W. Dumoulin, K Ten Haaf, J.G.J.V. Aerts

11.25am **S27**
The impact of mental health diagnoses on the uptake of lung cancer risk assessment and low-dose CT screening in a lung cancer screening implementation study
C Marshall, A Almatrafi, R Beeken, R Gabe, N Hancock, M Henderson, R Neal, I Simmonds, P A J Crosbie, M E J Callister

11.40am **S28**
Randomised controlled trial of patient navigation for lung cancer screening non responders
S L Quaife, D McInnerney, D Vulkan, L G Akwa, N Hancock, J Lindop, C Marshall, I Simmonds, S Rogerson, P A J Crosbie, R Gabe, M E J Callister

11.55am **S29**
Radiologist-Explainable AI Concordance and Confidence in Explainable AI supported clinical imaging decisions in lung cancer early diagnosis
C Kim, B Hunter, H Kalsi, L Sun, EC Bartlett, B Sharma, S Grubnic, E Aboagye, MD Blackledge, RW Lee, T LIBRA study investigators

12.10pm **S30**
FDG PET/CT for the assessment/staging of ground-glass and subsolid nodules: help or hindrance?
V Sobolewska, AEJ Hogg, J Bott, Z Abbas, DKB Patel, FR Millar

10.50am 12.25pm
Moore, 4th Floor
SPOKEN SESSION: S31 S36

Rewriting the immune landscape of the lung

Chaired by: Professor David Cousins and Dr Madhvi Menon

10.55am **S31**
Broad-spectrum antibiotics induce polarization of inflammatory type 2 immune responses in the lung and alter the immune landscape in a model of asthma
S.A.P. Colombo, N.A. Scott, J. Orr, C.P. McEntee, M.A. Travis, M.A.E. Lawson, T. Felton, L. Pearmain, E.R. Mann

11.10am **S32**
An intact Integrated Stress Response restrains the IL-6-pStat3 pathway
MH Schwiening, QG Gao, M Southwood, A Crosby, S Moore, N Veale, JA Valer, Upton PD, AAR Thompson, NW Morrell, Dunmoree BJ, SJ Marciniak, E Soon

11.25am **S33**
Impact of cigarette smoke extract and IL-17A on T2 and non-T2 inflammatory responses in human airway smooth muscle cells
Abdulrhman S. Alghamdi, Mushabbab A. Alahmari, Abdullah A Alqarni, Omar Alqarni, Dominick E Shaw, Linhua Pang, Amanda L Tatler

11.40am **S34**
Comprehensive characterization of a novel preclinical mouse model mimicking the complex phenotype of Chronic Obstructive Pulmonary Disease
M G Gualandri, G Natali, P Caruso, E Tantillo, S Siragusa, S Sgroi, M L Faietti, B Pioselli, C Bonfini, M Buccardi, E Ferrini, F F Stellari, M Trevisani, G Villetti, M Civelli, I Minato, M Morselli, B Montanini, S Pontis

11.55am **S35**
Inhibition of extracellular Cyclophilin A signaling attenuates acid aspiration-induced acute lung injury
Y Zhang, Y Chen, RF Baldi, M Takata, MR Wilson

12.10pm **S36**
Mechanical ventilation promotes inflammatory and profibrotic signaling in the pleural space following acute lung injury
RF Baldi, Y Zhang, O Perera, C Thomas, Z Luo, J Wan, M Takata, MR Wilson

10.50am 12.25pm
Abbey, 4th Floor
SPOKEN SESSION: S37 S4

Beyond Type 2 asthma

Chaired by: Professor Neil Barnes and Dr Sarah Diver

10.55am **S37**
Proinflammatory non-T2 immunology in the airways of a T2-low severe asthma cohort
PJ McDowell, E Aksenczuk, A Shackleford, J Busby, LG Heaney

11.10am **S38**
Distinct sputum transcriptomic and proteomic signatures associated with airway bacteria in severe asthma

Jiashu Shen, Nazanin Zounemat-Kermani, Sven-Erik Dahlen, Peter Bradding, Kian Fan Chung, Ian Adcock, Timothy Hinks

11.25am **S39**

LBP Elevation Indicates Gut Barrier Dysfunction in Outpatient and Hospitalised Severe Asthma Exacerbations.

LC Bonnard, SJ Abbott, Graham Sharpe, MJ Martin, GF Dodd, NJ Williams

11.40am **S40**

Characteristics and mechanisms of obesity-related asthma: a large-scale multi-omics analysis

Jiashu Shen, Nazanin Zounemat-Kermani, Benjamin Mappin-Kasirer, Chloe Bloom, Sven-Erik Dahlen, Ian Pavord, James Chalmers, Peter Bradding, Ian Adcock, Timothy Hinks

11.55am **S41**

When asthma is not asthma: alternative treatment decisions and safe corticosteroid withdrawal in a cohort with a primary-care diagnosis of uncontrolled asthma

CM Gill, C Ottewill, S Farrell, PJ Kerr, M Gleeson, V Brennan, O Smith, H Doherty, E MacHale, D Ryan, B Cushen, G Greene, RW Costello

12.10pm **S42**

Determining the effectiveness of an online FeNO-guided asthma management intervention in primary care (DEFINE): a pragmatic randomised controlled trial

K Wang, T Becque, R Thornton, B Mundy, L Jones, S Robinson, M Allwood, A Fairbairn, M Martin, B Stuart, N Francis, DE Shaw, ID Pavord, M Thomas

10.50am 12.25pm
Rutherford, 4th Floor
SPOKEN SESSION: S43 S48

Occupational lung disease – focus on silicosis

Chaired by: Dr Johanna Feary and Dr Kim Nettleton

10.55am **S43**

Occupational causes of interstitial lung disease, and trends in silicosis, from a supra-regional UK occupational lung disease service, 2001-25

GI Walters, CC Huntley, VC Moore, S Wallbanks, K Nettleton, PS Bhomra, E Hirons, AS Robertson, AD Vellore, PS Burge

11.10am **S44**

Artificial stone silicosis in the UK: what do we know?

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

11.25am **S45**

Work outcomes and spirometry in patients with silicosis in the North West

R Huang, H Badri, L Byrne, M Van Tongeren, R Wiggans, J Hoyle

11.40am **S46**

Airflow obstruction and crystalline silica exposure in a cohort from a supra-regional NHS occuaptional lung disease service

S Çakmakcı Karakaya, GI Walters, CC Huntley

11.55am **S47**

Understanding the impact of occupational exposures on pulmonary Sarcoidosis

E Hardy, J Shaw, A Thompson, Q Salman, T Raj, T Younas, C Leonard, B Vijayakumar, R Wiggans

12.10pm **S48**

Diverse radiological appearance of welding-related interstitial lung disease: experience from a supra-regional NHS occupational lung disease service

GI Walters, S Wallbanks, VC Moore, PS Burge, CC Huntley

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

Pulmonary Embolism and other Pulmonary Vascular Diseases

12.30pm – 1.30pm
Victoria, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING

Cough

Wednesday 25 November 2026

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

TB and Non-Tuberculous Mycobacteria

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

LUNCH (Lunch is not included in the delegate fee. Card payments only)

1.15pm – 2.00pm
Churchill, Ground floor
BTS SCIENTIFIC GUEST LECTURE

MEDIATING BEST IMMUNE RESPONSES: WHICH IS GREATEST THE NOSE OR THE LUNG?

Professor Daniela Ferreira (Oxford)

Introduced by: Professor Calum Semple OBE (Edinburgh)

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

Occupational and Environmental Lung Disease

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

Bronchiectasis

2.15pm – 4.00pm
Cambridge, 5th floor
MODERATED POSTER DISCUSSION: M1-M15

ILD: biomarkers and beyond

Chaired by: Professor Bibek Gooptu and Dr Amanda Goodwin

M1 Clinical Characteristics and Outcomes of Pleuroparenchymal Fibroelastosis (PPFE): A Retrospective Cohort Study
AC Cotton, MW Wells, JH Hardy, AE Edey, HA Adamali, SLB Barratt

M2 Bigger LITTLE WOMEN: Looking Into anTifibrotic ToLerancE in relation to Weight, dOse, feMale gender and EthNicity
GM Ruffino, N Chaudhuri, S Coughlan, E Patterson, C McCool, S Elshafie, M Elshafie, S Marguerie, SV Fletcher, A Alkhdour, I Adhikari, K Amcha, L Elton-Walters, L Chapman, F Thompson, M Jenkinson, I Nadeem, S Vythilingam, A Varghese

M3 Outcomes following respiratory-related hospitalisation in patients with fibrotic interstitial lung disease in the UK: Interim results from the INDEX Study
G Dixon, K Atkins, L White, A Goodwin, H Virk, I Nadeem, H Emms, S Goodrum, AMN Khin, A Baig, E Berry, C Butter, S Davies, N Dutta, E French, K Kemp, C Kilduff, I Mugal, L Ransome, C Albertyn, S Ali, J Brew, S Ghazal, o Kankam, A Thandamparambil, S Zaidi, FJ Chua, A Ali, J Callum, H Furreed, A Yasmin, J Derry, C Hayton, H Lee Evans, T Mellor, C Rowan, M Thirumaran, SL Barratt, E Osborne, A Parker, I Ufondu, A Ahmad, D Dorrian, P Minnis, C Reid, O Sabgowawa, M Al Belushi, L Crossland, WH Ong, S Smith, R Biju, P Sibanda, Y Shankar, E beri, S Cooper, H Parfrey, G Saini, TM Hartley, A Maqsood, S Wan, P Bhomra, A Fahim, M Mooradun, K Rajalingam, A Tang, K Vassell, S Baynes, J Hutchinson, N Khin, M Thway Ko, A Mishra, N Siribaddana, G Taylor, S Bikmalla, A Maimuna, B Oh, O Abel, SV Fletcher, G Ruffino, J Harridine, C Huntley, A Kan, G Singh, E Cosgriff, A Dey, E Ghanem, S Ghanem, M Keane, F Khan, A Hassan, K Jethwa, C Smith, S Woodmansey, CJ Scotton, MA Gibbons, MA Gibbons, G Dixon

M4 Using a qualitative co-design approach to develop a patient resource on artificial intelligence in interstitial lung disease
A Ahmad, Y Wen, J Ma, S Matchett-Griffiths, REK Russell, M Naqvi

M5 Systemic anti-cancer therapy-related pulmonary toxicity: a tertiary centre experience
A Liedtke, J Duff, E Fraser, LP Ho, R Hoyles, Y West, P Saunders

M6 Nutritional status and malnutrition screening in interstitial lung disease: A systematic review.
K Taylor, K Atkins, R Kahai, U Zanini, J Mandizha, S Airey, R Revuelta Iniesta, CJ Scotton

M7 ILD subtype distribution in patients of South Asian vs Non-South Asian ethnicity across two UK ILD centres
GR Jaiaprasat, DJF Smith, R Roy, F Khan, LF Cuthbertson, S Cai, KJ Myall, B Gooptu, B Gooptu

M8 FLOW-ILD: real-case vignette study of high-flow nasal oxygen and escalation decisions in acute interstitial lung disease deterioration
CKKL Ling, CKKL Ling, IDR Du Rand

M9 Comparison of longitudinal 6-minute walk deterioration, DO-GAP and ODDS score progression for mortality prediction in idiopathic pulmonary fibrosis
LR Robertson, MT Thillai, NS Simler, BKB Knox-Brown

M10 Combined EBUS-TBNA/TBNB and EUS-b-FNA/FNB improves tissue diagnosis of granulomatous lung disease presenting with isolated mediastinal lymphadenopathy: a prospective randomized trial
V Papavasileiou, A Dounavi, V Athanasopoulou, I Vrouvaki, P Siozos, D Chatzis, K-T Vandorou, E-M Antonogiannaki, I Voulgareli, V Tzilas, E Manali, S Loukides, T Raptakis

M11 The role of KL-6 in Pulmonary Sarcoidosis
K Senthil, C Reid, D Chow, A Lui, R Gilpin, D Linden, P Minnis

M12 ELEVATED FeNO: A DIAGNOSTIC CLUE TO UNMASK CHRONIC/FIBROTIC HYPERSENSITIVITY PNEUMONITIS
Salman Ayyaz, Shaheen Fatima, Tayyaba Hafeez, Amina Shabbir, Shakeel Ahmad, Sumbal Ayyaz, Muhammad Ihsaan

M13 A study evaluating the diagnostic utility of chitotriosidase versus serum angiotensin-converting enzyme in sarcoidosis, incorporating clinical and radiological disease activity data
AG Whiteside, A Horne, D Anderson, A Burns, L Pearce, J McNeilly, A Wright

M14 Clinical and humanistic burden of pulmonary hypertension associated with interstitial lung disease (PH-ILD): secondary analysis of the Adelphi Disease Specific Programme (DSP)
H.J. Ford, B. Shah, M. Small, D. Graham, L. Holdstock, J. Fastenau

M15 Efficacy and Radiological Predictors of Response to Pulsed Intravenous Cyclophosphamide (IV-CYC) in Refractory ILD
Wint Soe, Muhammad Abdulmajid, Muhammad Adillshad, Samuel Healy, Mardiya Bako, Michael Ball, Fiona Thompson, Anna Donaldson, Louise Newman, Sowkhya Ramachandraiah, Adam Wallis, Suresh Babu

2.15pm – 3.45pm
Churchill, Ground floor
SYMPOSIUM

THE MIKE MORGAN LEGACY - PRIORITISING RESPIRATORY CARE & DELIVERING THE NHS LONG TERM PLAN

Chaired by: Professor Michael Steiner (Leicester) and Professor Richard Russell (London)

14.15 Enabling early and accurate diagnosis of respiratory disease
Professor William Man (London)

14.45 Expanding Pulmonary Rehabilitation
Professor Sally Singh (Leicester)

15.15 A policy legacy: continuing respiratory transformation
Dr Jonathan Fuld (Cambridge)

Learning objectives:

1) To recognise the contribution of Prof Morgan in championing this area, the importance of enabling early and accurate diagnosis of respiratory disease and where we are now in achieving the aims of the Long Term Plan

2) To recognise the contribution of Prof Morgan in championing this area, the importance of expanding pulmonary rehabilitation and where we are in achieving the aims of the Long Term Plan

2.15pm – 3.45pm
Mountbatten, 6th floor
JOINT BTS/BPRS SYMPOSIUM

BREATHING FROM THE START: THE IMPACT OF AIR POLLUTION ON RESPIRATORY HEALTH FROM BIRTH TO ADOLESCENCE

Chaired by: Dr Sonal Kansra (Sheffield) and Dr Alexandra Adams (Leeds)

14.15 From Womb to Childhood: Biological Mechanisms Linking Air Pollution Exposure to Lung Development and Disease
Professor Pia Hardelid (London)

14.45 Indoor Air Pollution and Child Respiratory Health: Hidden Exposures, Mechanisms, and Modifiable Risks
Dr Shamil Haroon (Birmingham)

15.15 From Evidence to Action: Health Policy Levers to Reduce the Respiratory Impact of Air Pollution in Children
Dr Abigail Whitehouse (London)

Learning objectives:

1) Describe how air pollution exposure from pregnancy through childhood affects lung development and later respiratory disease

2) Identify key indoor air pollution exposures and modifiable risks relevant to paediatric respiratory health

3) Understand how air pollution evidence informs policy actions to reduce respiratory health inequalities

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2.15pm – 3.45pm
Windsor, 5th floor
SYMPOSIUM

Early Career Investigator Awards

Chaired by: Professor Mona Bafadhel, Professor Philip Molyneaux and Dr Amanda Tatler

2.20pm **T1**
Investigating the inflammatory profile of exacerbations in patients with severe asthma receiving tezepelumab: the phase IV TezEx study
F Haris, F Cefaloni, J Gates, P Khooshemehri, A D Rosario, A P Valles, E Woods, V Kanabar, E Scourfield, G d'Ancona, J Dhariwal, A M Nanzer, D J Jackson

2.35pm **T2**
Acquired monocyte reprogramming drives divergent responses to microbial stimuli in Idiopathic Pulmonary Fibrosis.
L Pearmain, F Hunter, S Murtuza Baker, E Jokl, K Piper Hanley, ER Mann

2.50pm **T3**
Feasibility and external validation of the RED score in clinical practice: a multicentre service evaluation
DK Sethi, A Sinha, K Warner, A Aujayeb, M Selvan, S Kuckreja, J Liang, J Zhang, D Jajbhay, J LeCrette, G Tinson, A Kwok, A Bull, R Peries, A Elsheikh, A Clarke, R Bhatnagar, N Rahman, EK Mishra

3.05pm **T4**
FeNO response to benralizumab is determined by bronchial epithelial phenotype
J Gates, J Shen, P Khooshemehri, V Kanabar Raivadera, A del Rosario, A Perez Valles, E Woods, R Martinez Nunez, D J Jackson

3.20pm **T5**
Precision medicine in chronic cough: Predicting low-dose morphine responders
MZ Zhang, AHM Morice, KB Brindle, JN Nielsen

3.35pm **T6**
Integrated multi-omic and functional profiling of immune responses in pleural infection
A Gilmour, H Lind, E Ward, D Sanderson, R C Hull, K Viligorska, J M New, L Marshall, H Richardson, I Petrou, H Liddicoat, R Galloway, N I Kanellakis, N Rahman, D Arnold, M B Long, J D Chalmers, P Short

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

Advancing lung cancer diagnostics and treatment

Chaired by Dr Elizabeth Fuller

P1 Robotic bronchoscopy following prior non-diagnostic biopsy: diagnostic yield and malignancy detection in a salvage diagnostic cohort
BP Patani, HT Trainer, JP Panaguiton, OV Volhina, AK Karir, AT Tana, JLG Garner, JT Tonkin, CMO Orton, PLS Shah

P2 The Impact of Experience on Radiation Dose and Procedure Duration in Shape-Sensing Robotic-Assisted Bronchoscopy.
HJ Trainer, B Patani, J Panaguiton, O Volhina, A Karir, A Tana, J Tonkin, JL Garner, PL Shah, CM Orton

P3 Real-World Outcomes of Navigational Bronchoscopy for Peripheral Pulmonary Lesions: A Single Centre Analysis of Demographics and PDL1 Specimen Adequacy
EF Foley, DT Thong, DB Breen

P4 Adequacy of Tissue Acquired by Shape-Sensing Robotic Bronchoscopy for Molecular Profiling of Non-Small Cell Lung Cancer
R Cuttle, A Askari, R Dunwoody, M Mullin, Y Soo, I Allon, R Shea, N Navani, R Thakrar

P5 Diagnostic yield and safety of CT-guided lung biopsy performed following multi-disciplinary triage of robotic navigational bronchoscopy referrals
A Devaraj, J Tonkin, CM Orton, PL Shah, J Garner, R Shah, S Santhirakumaran, R Walsh, C Watson, S Padley, B Rawal, C Ridge, E Bartlett

P6 Optimising Molecular Characterisation of NSCLC Through Combined Flexible Bronchoscopy and EBUS-TBNA
EF Foley, LT Thong, DB Breen

P7 A longitudinal analysis of the frequency and indication for repeat Endobronchial Ultrasound Transbronchial Needle Aspiration (EBUS-TBNA) sampling in a National Academic Cancer Centre
SA Azman, A Samad, R Murray, L Alhaddah, N McCarthy, M Rasool, A O, MT Henry, J McCarthy, MP Kennedy

P8 Transforming Training in Lung Cancer Diagnostics: Evaluation of novel patient-derived lung phantoms
Y Soo, E Maneas, A Desjardins, R Thakrar, N Navani

P9 An evaluation of EBUS-TBNA training among UK respiratory trainees
R Dunwoody, A Bhamani, R Khan, N Navani, R Thakrar

P10 Assessment of Endobronchial Ultrasound (EBUS) Training Exposure and Confidence Among Respiratory Trainees in Yorkshire & Humber
CA Aday, DS Sagar

P11 Factors influencing patient tolerance and sample adequacy of EBUS procedures: a single-centre retrospective study
D Willans, L Devlin, J Gallois, F Khalid

P12 Evaluation of EBUS procedures performed in a single site over four years against NHS EBUS Standard Specifications
S Pahal, S Babiker, G Olatunde, A Lal

Wednesday 25 November 2026

P13 Management of Moderate/Large Pericardial Effusion in Patients with Lung Cancer in a District General Hospital
M Jenkinson, E Shaji, T Horey, A Varghese, U Ekeowa, PC Russell

P14 The change in the prevalence of central airways obstruction in lung cancer over 10 years
H Liu, P Ridge, C Lo, D Crowle, I Zaki, L Telisinghe, C Daneshvar

P15 Patients with impaired ventilatory function undergoing potential lung cancer surgery. A propensity-matched study
P Gomes-da Silva de Rosenzweig, Y Saif, H Jesani, HD Walji, AE Martin-Ucar, L Hernandez-Arenas

2.15pm - 4.00pm
Westminster, 4th Floor
POSTER DISCUSSION: P16 - P29

Cough: clinical profiles and novel interventions

Chaired by: Dr Barnaby Hirons

P16 Refractory chronic cough patients with low objective cough frequency have a severe cough and burden
KL Rhatigan, EC Mackay, C Wood, R Crickett, RD Turner, CJ Jolley, KJ Myall, JH Hull, SS Birring, PSP Cho

P17 Control of disease in chronic cough: development of a conceptual framework
EC Mackay, KL Rhatigan, RD Turner, KJ Myall, CJ Jolley, WJ Song, JH Hull, SS Birring, PSP Cho

P18 Microbiological, physiological and inflammatory profiles of excessive dynamic airway collapse
F Yang, Z Williams, B Wright, M Kashoub, J Garner, J Tonkin, CM Orton, PH Patel, JH Hull

P19 Evaluating Clinical Delivery Costs of a Virtual Group Physiotherapy Intervention (ViP-Cough) Versus Traditional One-to-One Cough Suppression Therapy
CC Reilly, E Shiner, P May, SV Floyd, D Greir, PS Cho, CJ Jolley, SS Birring

P20 Prevalence and Characteristics of Breathing Pattern Disorders in Adults with Refractory Chronic Cough or Inducible Laryngeal Obstruction: A Systematic Review
K Bui, H Harvey, J Butler, J Haines, G Clunie

P21 Experience of prescribing Gefapixant in a UK Cough Clinic
MZ Zhang, KB Brindle, JN Nielsen, AM Morice

P22 Day-to-Day Variability of Objective Cough Monitoring in Fibrotic Interstitial Lung Disease (fILD)
A S C Lawrence, B Mukherjee, K Rhatigan, P S P Cho, S S Birring, K J Myall

P23 Role of Physiotherapy within a Chronic Cough Service – Review of Referrals
A Folwell, J Harrison, K Prior

P24 Clinical diagnostic confidence and treatment patterns in cough management: a cross-sectional survey among physicians
Q Liu, HQ Chen, R Zhu

P25 Association Between Common Comorbidities and Distribution of Chronic Cough Etiologies in China: An Interim Analysis of a Cross-Sectional Clinician Survey
Siyu Hu, Zifeng Hong, Yahui Shen

P26 From Community Clinics to Tertiary Centers: How Hospital Hierarchies Shape the Characterization of Cough Management
M Su, H Zhang, W Wang, Y Wang

P27 Age-Related Differences in Etiology of Chronic Cough: An Interim Analysis of a Multicenter Cross-Sectional Clinician Survey in China
Zifeng Hong, Siyu Hu, Yahui Shen

P28 Incidence and predictors of future asthma diagnosis in subjects with cough.
J.H. Hämäläinen, A.L. Lätti, A.T. Tolppanen, H.K. Koskela

P29 Physicians' perceptions of factors influencing cough symptom relief: a cross-sectional survey across China's tiered healthcare system
GY Zhou, J Yuan, Q Tang, S Zhao, XB Li

2.15pm - 4.00pm
Moore, 4th Floor
POSTER DISCUSSION: P30 - P41

Pulmonary vascular disease – from novel therapies to clinical service improvements

Chaired by: Dr Rachel Davis and Dr Katherine Bunclark

P30 The Pulse-PH Registry: Real-World UK Data in patients with Interstitial Lung Disease and Sarcoidosis-associated Pulmonary Hypertension
S Herbert, K Dimopoulos, D Kiely, C Church, N Karia, L Howard, J Lordan, K Bunclark, E Cerrone, J Ingram, A Baccelli, I Jenkin, B Khan, TJW Dawes, R Condliffe, N Hamilton, R Thompson, I Armstrong, G Coghlan, W Gin-Sing, C McCabe, H Nashat, B Rawal, A Devaraj, C Harries, V Kouranos, S Bax, AU Wells, G Jenkins, PM George, SJ Wort, LC Price

P31 National cohort study: causes of mortality in patients with chronic thromboembolic pulmonary disease without pulmonary hypertension
L D'Amours, L Dan, M Bhattacharyya, C Y Li Cai, J E Cannon, J Pepke-Zaba, K K Sheares, D Taboada Buasso, K Bunclark, M Toshner

P32 Hidden in plain-sight: ICD-10/SnoMed reasons why thousands of NHS patients with pulmonary arteriovenous malformations are missed by major rare disease initiatives
CL Shovlin, A Alsafi, B Glampson, E Mayer

P33 Intermediate-high risk pulmonary embolism: outcomes with systemic thrombolysis or intravenous heparin in real-world practice.
P Cropley, N Abuhalaweh, H Rupani, M Brown

P34 Prior Prostacyclin Pathway Exposure and Tolerance of Intravenous Epoprostenol Initiation in Pulmonary Arterial Hypertension
WLL Lai, ML Laraghy, AV Vlachou, SW Wort

P35 Thrombolysis for Acute PE: time for rapid multidisciplinary interventional team?
A Goel, S Salloum, R Love, O Elneima

P36 The value of PDE5i therapy in patients with Group 3 pulmonary hypertension: Real world data from a pulmonary hypertension centre
S Barth, A Trachalaki, W Gin-Sing, G Haji, LS Howard, F Lo Giudice, R Davies

P37 Catheter directed Therapy (CDT) of Pulmonary Embolism: An overview of the service run in a South Wales District General Hospital.
E Yarham, J Coventry, M Davies, M Pynn

P38 Can District General Hospitals Deliver Advanced Pulmonary Embolism Care? A Two-Year Real-World Experience of Catheter-Directed Therapy

Z Aung, M Charles-Rudwick, D Alrawashdeh, P Mpisi, K Pannu, B Yung, U Dawar, D Mukherjee, A Zaman

P39 Impact of Frailty on Clinical Outcomes in Acute Pulmonary Embolism: A UK District General Hospital Cohort Study
Z Mer, M Al-Hakim, C Valerio, I Moonsie

P40 Using NEWS to predict outcomes in acute pulmonary embolism: a two-centre cohort study
VK Khemani, JJ Johnson, PK Katarki, AT TARIQ, NA AHMAD

P41 Comparison of clinical and biochemical prediction tools for excluding pulmonary embolism and reducing imaging
MS Anwar, M Masued, M Jenskinson, S Cooper, A Shrestha, R Dhulipala, L Sandhu, N Surendraraj, T Iqbal, M Shafique, A Nawazani, K Hucknall, R Muthurajah, F Saleem, H Hamid

2.15pm - 4.00pm
Abbey, 4th Floor
POSTER DISCUSSION: P42 - P56

Breaking barriers – improving lives, transforming access to healthcare

Chaired by: Dr Dominick Shaw and Professor Rachel Jordan

P42 The Overlooked Respiratory Burden of Homelessness: A UK Electronic Health Records Study
E Carruthers, D Shaw, CI Bloom

P43 Introducing a novel daily Medical Nurse Practitioner (MNP) pleural clinic; assessing clinical efficiency and patient satisfaction
AE Leadbetter, S Bashir, B Romain, VD Masani, S Meiklereid, E Peter, D Griffiths, J Puddy, A Bandasan, JC Harper, J Walters

P44 Abstract Withdrawn

P45 Targeted Lung Health Checks: are we missing the opportunity to diagnose and treat COPD?
SDP Daniel-Papi, TM Macdonald, AK Kong, HB Burke

P46 The role of a Nurse-Led clinic in improving access and efficiency in the management of Latent Tuberculosis Infection (LTBI) in adults at Imperial College Healthcare NHS Trust Tuberculosis services.
A Hoxha, C Luca, H Durkan, V Padmanaban, O M Kon, A Lalvani

P47 Targeted education is a potential strategy to improve the lived experience of young adults (16-25yrs) with severe asthma.
LJ Holmes, K Lovell, G Jones, N Sehgal, M Marshall, S Fowler

P48 Patient experience of a multidisciplinary asthma transition clinic: a retrospective service evaluation
MJ Jurca, DL Lo, LP Pawlick, AC Claydon, VR Rai, EAG Gaillard, MR Ramphul

P49 'Nothing About Me Without Me'. Young people With Asthma and Upper Airway Disorders Transitioning to Adult Services: Service Evaluation of a UK Hospital
Jordan Thomas, Sarah Vollam, Imran Howell

P50 Research equity by design: insights from the A-TREC cohort
PJ Ireland, M Scott, R Sobala, G Mussell, A Prasad, E Pakpahan, AJ Simpson, J Steer, SC Bourke

P51 Same-day text messaging following lung MDT downgrade decisions: improving communication equity in a deprived, multilingual population.
R Rathbone, N Patel, S Ghattas

P52 Developing a blueprint for using chronic obstructive pulmonary disease (COPD) to illustrate best neighbourhood health and outcomes across the UK
K Hickman, On behalf of PCRS committee members, On behalf of Neighbourhood Health project group

P53 Findings from the HERON needs prioritisation partnership: Exploring and mapping unmet needs and potential health technology solutions in chronic respiratory disease
Z Oganesova, S Panchenko, M Wanat, P Turner, G Hayward, H Ashdown

P54 Experiences of specialist nurse review delivered remotely in progressive pulmonary fibrosis
C Ruggiero, M Waite, L Stayt

P55 The value of a multidisciplinary breathlessness service to manage extra pulmonary treatable traits in patients with chronic respiratory disease
AM Cole, R Tatters, V Ord, M Bhabra, J Hughes, J Kibbler, A Prasad, M Jachuck, M Rangar, A MacNair

P56 Right care, every time: Designing patient and healthcare provider checklists to support Non Tuberculous Mycobacteria pulmonary disease (NTM-PD) management across the UK
A Sawka, S Bryant, J Hockley, M Lipman

2.15pm - 3.50pm
Rutherford, 4th Floor
POSTER DISCUSSION: P57 - P70

TB: from diagnosis to care

Chaired by: Dr Claire Calderwood and Dr Martin Dedicoat

P57 Evaluating outcomes of Anti-TB therapy in older people
Z Ahmad, E Hamilton, A Zhuwao, Y Martins, D Hawley, D Creer, S Lee, R Lever, D Lowe, R Moores, M Lipman, J Brown

P58 Characterising non-tuberculous mycobacterial pulmonary disease (NTM-PD): how does this compare with bronchiectasis? An observational cross-sectional study
Naif Sulaiman, Amina Jaffer, Nawal Alotaibi, Dean Creer, Simon Lee, Robert Lever, David Lowe, Rachel Moores, John R Hurst, James Brown, Marc Lipman

P59 An Interim Evaluation of Pharmacy service contributions to Tuberculosis (TB) care pathways in the UK: A Stakeholder practice and perception survey
CM Chen, TG Capstick, M Gilchrist, A Chavda

P60 Introducing antimicrobial stewardship within TB services - A review of Infection pharmacy-led interventions at a London tertiary centre
A Chavda, P Patel, M Gilchrist, H Durkan, A Hoxha, OM Kon

P61 Drug-induced liver injury in active and latent tuberculosis treatment: a retrospective evaluation of monitoring strategies
FC Ó Fearghail, R D'Souza, R Nyame-Satterthwaite, E Cooley, J Pashley, S Stinchcombe

P62 Local, but not systemic, neutrophil-driven inflammation associates with lung pathology in tuberculosis
J Kutschenreuter, DE Kirwan, DLW Chong, MC Loader, O Gayoso, CT Lin, JR Azadi, RH Gilman, JS Friedland

P63 Mapping the dynamics of host transcriptional responses and sputum bacteriological clearance during treatment for pulmonary tuberculosis
F Kloprogge, S Ajagbe, S Assayed, A Dunleavy, C Gonzalez Bridger, S Gurram, V Hack, T Khan, A Keszthelyi, OM Kon, H Kunst, M Noursadeghi, P Papineni, M Ramirez, S Quaid, C Ramsey, X Torres, A Whittington, S Wasserman, M Lipman

P64 TB PCR testing in Non-Sputum Samples to Reduce Diagnostic Delays: A Retrospective Case Series
G Long, F Hopkins, O Dixon, J H Y Chiu, R Hussain, R Ahmed

P65 Survey of Adenosine Deaminase (ADA) Testing Practices for Pleural Tuberculosis Across Hospitals in England
A Kaur, D Creer, S Brill, J Brown, M Lipman, A Thayanandan, S Lee, S Sayers

P66 Genomic characteristics of Mycobacterium tuberculosis among small-scale miners in Northern Tanzania
P Howlett, B Said, E Mwanga, I Godfrey, A Mbuya, M Van Zweltsaar, OM Kon, A Amaral, J Feary

P67 Can an RD-1 based skin test strategy improve the TB infection care cascade where IGRA is standard?
Y HAMADA, A Kilic, S Fabiane, C Layton, T Duong, R Horne, S Jhuree, H Kunst, M Lipman, N Mandelbaum, E Owen-Powell, D Trathen, D Zenner, I Abubakar, MX Rangaka

P68 Tuberculosis infection care in migrants needs improved access to adequate online educational resources to improve outcomes
H Kunst, S Ali-Sheikh, J Zhang, Y Long, P Hernandez-Jimenez, L Kabalan

P69 Decision-Making in Latent Tuberculosis Testing: Qualitative Insights on Novel TB-Specific Skin Tests
AK Kılıç, ZM Moon, AHYC Chan, CL Layton, DZ Zenner, EOP Owen-Powell, HK Kunst, KS Sanders, ML Lipman, MM Mandelbaum, PP Papineni, TD Duong, YH Hamada, IA Abubakar, MXR Rangaka, RH Horne

P70 What is the impact of radiology referrer alerts on tuberculosis diagnoses? Experience from a single centre
T Fisayo, P Ojo, COF Dean, P Palchaudhuri, E Skyllberg, N Jayasekera, A Gray, L Baker

3.00pm – 4.30pm
Whittle & Fleming and Britten, 3rd floor
COFFEE/TEA BREAK

3.15pm – 4.15pm
Albert, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Critical Care, Respiratory Failure and Mechanical Ventilation

3.15pm – 4.15pm
Gielgud, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Tobacco Dependency

Wednesday 25 November 2026

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 Awards, BTS/A+LUK/BALR Mid-Career Lecture Awards and BTS Medical Student Awards.

4.45pm – 5.30pm
Churchill, Ground floor
BTS PRESIDENT'S ADDRESS
Education and Networking: The Power to Shape the Future

Professor Adam Hill (Edinburgh)

Introduced by Professor Robina Coker (London)

5.35pm – 6.05pm
Churchill, Ground floor
BTS ANNUAL GENERAL MEETING
British Thoracic Society members only

8.00am – 9.00am
Whittle & Fleming, 3rd floor
COFFEE/TEA

8.00am – 9.00am
Gielgud, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Pharmacist

8.45am – 4.00pm
Whittle & Fleming, 3rd floor
POSTER VIEWING

Authors present: 10.00am – 11.00am

P71-P85
Biologics galore!
Discussion of abstracts will take place from 2.15pm to 4.05pm in the Westminster, 4th floor

P86-P100
Respiratory infections: from disease burden to outcomes
Discussion of abstracts will take place from 2.15pm to 4.05pm in the Moore, 4th floor

P101-109
Global and public health: future aspects with impact
Discussion of abstracts will take place from 2.00pm to 3.20pm in the Abbey, 4th floor

P110-P122
The breath of childhood: science, innovation and care
Discussion of abstracts will take place from 2.00pm to 3.50pm in the Rutherford, 4th floor

P123-P137
Asthma or something else?
Discussion of abstracts will take place from 4.15pm to 6.00pm in the Moore, 4th floor

P138-P152
Novel approaches to improving COPD care: imaging, AI and acute intervention
Discussion of abstracts will take place from 4.15pm to 6.00pm in the Abbey, 2nd floor

P153-P167
ILD: cohort studies and service improvement
Discussion of abstracts will take place from 4.15pm to 6.05pm in the Rutherford, 4th floor

P168-P1180
Bronchiectasis unconfined
Discussion of abstracts will take place from 4.15pm to 5.50pm in the Albert, 4th floor

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8.45am – 4.00pm
Cambridge, 5th floor
MODERATED POSTER VIEWING: M16-M31
Real world pleural medicine: improving care outcomes, training and delivery

Discussion of abstracts will take place from 2.15pm to 4.00pm in the Cambridge, 5th floor

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8.30am – 9.00am
Victoria, 2nd floor
JOURNAL CLUB
Pulmonary Embolism

Dr Elaine Soon (Cambridge)

Learning objective

To review the latest publications and evidence in the field of Pulmonary Embolism and other Pulmonary Vascular Diseases

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9.00am – 10.30am
Churchill, Ground floor
SYMPOSIUM
LUNG CANCER IN NEVER SMOKERS; HOW CAN WE IDENTIFY THOSE AT HIGHEST RISK?

Chaired by: Professor Mat Callister (Leeds) and Dr Tanya Patrick (London)

09.00	Integrating epidemiologic, genomic, and experimental data to outline a unifying framework for lung adenocarcinoma in never smokers Dr Maria Theresa Landi (USA)
09.30	Understanding and integrating environmental and inherited risk Dr Frank McCaughan (Cambridge)
10.00	How do we identify high risk people? Biomarkers, risk profiling and screening Dr Hillary Robbins (Lyon)

SCIENTIFIC PROGRAMME

Learning objectives:

1) To understand the key genetic drivers of lung cancer in never-smokers, understand how these differ from smoking-related disease, and recognise the clinical implications for diagnosis and treatment pathways

2) To describe the distinctive features of the tumour micro-environment in lung cancer in never-smokers, including inflammatory pathways and immune cell interactions, and understand their implications for tumour development, progression and treatment

3) To understand current and emerging approaches to risk stratification in never-smokers, including the role of biomarkers and risk prediction models, with reference to the principles of screening, balancing benefit against harm and maximising mortality benefit

9.00am – 10.30am
Mountbatten, 6th floor
SYMPOSIUM

EVOLVING PRACTICE IN THE DELIVERY OF PULMONARY REHABILITATION: WHERE NEXT?

Chaired by: Dr Samantha Kon (London) and Dr Enya Daynes (Leicester)

09.00	Delivering effective PR with minimal equipment Dr Claire Nolan (London)
09.30	Adapting exercise prescription for clinically relevant co-morbidities Professor Frits Franssen (Netherlands)
10.00	New horizons in exercise training in patients with severe dyspnoea Professor Ioannis Vogiatzis (Northumbria)

Learning objectives:

1) To understand how pulmonary rehabilitation can be adapted where there is a lack of access to specialised gym equipment and increase accessibility

2) To review the evidence and provide practical recommendations for tailoring exercise interventions in individuals with COPD and relevant comorbidities

3) Reviewing the latest evidence and practical implications for the prescription of exercise in those with severe dyspnoea

Thursday 26 November 2026

9.00am – 10.30am
Windsor, 5th floor
SYMPOSIUM

JOINT BTS/BALR (PART 2) SYMPOSIUM
PIONEERING NEW FRONTIERS IN RESPIRATORY RESEARCH: THE BIG DATA REVOLUTION

Chaired by: Dr Jonathan Baker (London) and Dr Bettina Schock (Belfast)

09.00	Multi-omics and data integration in translational respiratory research Dr Iain Stewart (London)
09.30	Integrative 'omic analyses to delineate fibrogenesis Dr Janine Gote-Schniering (Switzerland)
10.00	Multi-layer network analysis for COPD stratification Professor Rosa Faner (Spain)

Learning objectives:

1) Discover approaches to integrate multi-omic datasets across respiratory diseases to progress basic, translational, and clinical respiratory research

2) Understand the molecular insights in to fibrosing lung diseases resulting from mult-omic approaches

3) Learn how multilayer network analysis can be applied to disease stratification

9.00am - 10.35am
St James, 4th Floor
SPOKEN SESSION: S49 - S54

Next generation asthma care

Chaired by: Dr Jane McDowell and Dr Freda Yang

9.05am	**S49** Mechanistic insights into steroid chronotherapy in asthma. FK Khanom, VD Deugi, RM Maidstone, RW Wang, JB Blaikley, AC Chakraborty, HD Durrington
9.20am	**S50** Exacerbation trajectories in biologic-naïve severe asthma patients F Yang, DC Marshall, I Howell, TSC Hinks, KF Chung, ID Pavord, DJ Jackson, CI Bloom

9.35am **S51**
Exacerbation-free proportions in placebo arms of asthma biologic trials: a meta-analysis and meta-regression of predictors
F Yang, J Dhariwal, CI Bloom, DJ Jackson

9.50am **S52**
Effect of tezepelumab on lung function in patients with OCS-dependent severe asthma by baseline inflammatory biomarker levels: results from the phase 3 SUNRISE study
CE Brightling, ME Wechsler, I Capala-Szczurko, H Lal, P Scrigner, G Hunter, P Kabelis, N Molfino, SS Ponnarambil

10.05am **S53**
Clinical response to Tezepelumab in a real-world setting is preserved regardless of baseline biomarker status or presence of comorbidities
S Kharbanda, N Khine, H Keelan, V Singh, C Eames, S Kerley, M Pantaleon, E Rayala-Montaniel, P Cook, J McCreery, A Freeman, HM Haitchi, RJ Kurukulaaratchy, P Dennison, H Rupani

10.20am **S54**
Twice-yearly depemokimab demonstrates efficacy in patients with asthma and comorbid chronic rhinosinusitis with nasal polyps (CRSwNP): Phase III SWIFT-1/-2 studies
E Heffler, L Jacques, CQ Zhu, A Vichiendilokkul, P Howarth, ID Pavord

9.00am - 10.35am
Westminster, 4th Floor
SPOKEN SESSION: S55 - S60

Bronchiectasis: biology, biomarkers and better care

Chaired by: Professor Jeremy Brown and Dr Emma Johnson

9.05am **S55**
Molecular detection reveals pathogen-specific inflammatory signatures in bronchiectasis
R C Hull, H Richardson, H Lind, C Hennayake, D Alferes de Lima Headley, J Pollock, E McIntosh, R Galloway, E Johnson, M Shuttleworth, P C Goeminne, M Shteinberg, A De Soyza, S Aliberti, J Altenburg, C S Haworth, O Sibila, E Polverino, M Loebinger, F C Ringshausen, N Lorent, K Dimako, J Stobo, A Shoemark, M B Long, J D Chalmers

9.20am **S56**
Bronchoalveolar lavage proteomics in bronchiectasis and protracted bacterial bronchitis provides novel insight into airway inflammation
Isavella Petrou, Jeffrey T Huang, Merete B Long, Hannah Dickson, Melissa McDonnell, Rebecca C Hull, Chloe Hughes, James D Chalmers

9.35am **S57**
Cross-compartment inflammatory programmes identified by multi-omic integration are associated with disease severity and infection in bronchiectasis: results from EMBARC-BRIDGE
M B Long, L Perea, R C Hull, C Hughes, E Johnson, M K Shuttleworth, J Pollock, M Shteinberg, S Aliberti, S H Chotirmall, E Polverino, P Goeminne, N Lorent, F Ringhausen, R Faner, O Sibila, A Shoemark, J D Chalmers

9.50am **S58**
Patient-Reported Sputum Color and Its Association With Neutrophil Serine Proteases and Exacerbations in Patients With Bronchiectasis: A Post Hoc Analysis From the ASPEN Trial
PA Flume, S Aliberti, Pieter Goeminne, X Wei, M Lauterio, A Teper, JD Chalmers

10.05am **S59**
The BRACE study: a multicentre UK audit of multidisciplinary bronchiectasis care
M Carling, J Kibbler, A Konkoth, E Stamp, A De Soyza, H Upadhyay, R Anderson, F Fyles, D George, J Bradley, K Bielosludtseva, J Chalmers, E Ward, D Sanderson, F Waddell, J Lawson, R Haider, M Adamu, E Lyka, P Mitchelmore, A Carter, D Lau, J Sutanto, V Truong, L Gutierrez, J Dunleavy, J De Soyza, S Hyatali, H Anderson, S Higham, M Ting, P Jones

10.20am **S60**

Effect of Brensocatib on Patient-Reported Symptoms in Patients with Non-Cystic Fibrosis Bronchiectasis: A Post Hoc Analysis of QOL-B RSS Individual Items From the ASPEN Phase 3 Trial

PA Flume, JJ Tolle, GM Solomon, S Aliberti, K Furuuchi, X Wei, J Fastenau, M Lauterio, C Fernandez, JD Chalmers

9.00am - 10.35am
Moore, 4th Floor
SPOKEN SESSION: S61 - S66

ILD: risk prediction

Chaired by: Dr Pilar Rivera Ortega and Dr Giles Dixon

9.05am **S61**

SpO_2 recovery time post 6MWT improves risk stratification in interstitial lung disease

L.R Robertson, M.T Thillai, NS Simler, BKB Knox-Brown

9.20am **S62**

Intermittent hypoxaemia is associated with the quality-of-life of patients with idiopathic pulmonary fibrosis

LE Crowley, LE Crowley, E Jenkins, C Mafham, RY Mahida, RY Mahida, BG Cooper, T Cunnington, A Lomas, A Scott, A Scott, DR Thickett, DR Thickett, DR Thickett, D Parekh, D Parekh, D Parekh, D Parekh

9.35am **S63**

Acute Respiratory Worsening and Survival Outcomes in patients with Idiopathic Pulmonary Fibrosis: A Population-Based Cohort Study

L Yazbeck,T.M Maher,C Bloom, P. L Molyneaux

9.50am **S64**

External validation of mortality prediction models in idiopathic pulmonary fibrosis using real-world data from the British Thoracic Society (BTS) interstitial lung disease (ILD) registry

A Chao, X Nie, KA Betts, M Sundar, A Carabajal-Johnson, A Desai, Y Zhong, PL Molyneaux

Thursday 26 November 2026

10.05am **S65**

Social, geographic and environmental determinants of mortality in pulmonary fibrosis: a prospective PROFILE cohort analysis

AT Trachalaki, GG Goobie, PM Molyneaux, FC Chua, LVW Wain, AUW Wells, ER Renzoni, GS Sain, SJ Johnson, TMM Maher, GJ Jenkins, IS Stewart

10.20am **S66**

The impact of race and gender on different spirometric reference equations used to assess %-predicted FVC for antifibrotic eligibility in idiopathic pulmonary fibrosis

A Opaleye, C Roberts, PL Molyneaux, S Bax, EA Renzoni, RJ Hewitt, V Kouranos, P George, M Kokosi, GJ Jenkins, AU Wells, F Chua

9.00am - 10.35am
Abbey, 4th Floor
SPOKEN SESSION: S67 - S72

Paediatric respiratory medicine: vaccines, asthma and innovation

Chaired by: Dr Wanda Kozlowska and Dr Lena Thia

9.05am **S67**

Blowing Away Inequality: 12-month outcomes from a Children and Young People (CYP) asthma diagnostic hub.

C K Konkal Matt, T Evans, S Frost, M Swallow, P Nagakumar, V Vijay

9.20am **S68**

Left breathless: gaps in basic asthma care for children and young people in England — a retrospective cohort study

A Gawlik-Lipinski, JK Quint, CL Gillies, DKH Lo

9.35am **S72**

Efficacy and safety of twice-yearly depemokimab in adolescent patients with asthma: Subgroup analysis of the SWIFT-1/-2 studies

DJ Jackson, S Schalkwijk, D Jarrreta, N Bird, C Tacon, MR Gupta

9.00am - 10.35am
Rutherford, 4th Floor
SPOKEN SESSION: S73 - S78

Sleepy futures: from hospital to home

Chaired by: Dr Johan Meurling and Dr Annabel Nickol

9.05am **S73**
Information Flow from Oxygen Saturation to Heart Rate Predicts Cardiovascular Mortality and Morbidity in Obstructive Sleep Apnoea
AJ Shah, FF Neves, S Mittal, N Devani, A Saigal, JR Hurst, AR Mani, S Mandal

9.20am **S74**
Transforming Sleep Diagnostic Pathways: Implementing a Clinical Decision Support System (CDSS) across a District General Hospital and Community Diagnostic Centre (CDC) setting
CA Coombs, RH Mason

9.35am **S75**
Multi-Night Home Sleep Apnea Testing Improves Diagnostic Accuracy Compared with Single-Night Assessment
N F Watson, Y N Nygate, S Waters, S Rusk, A Manuel, C R Fernandez

9.50am **S76**
Do scoring methods matter? The effect of WatchPAT autoscoring on treatment decision-making
VC Moore, T Matharu, ED Parkes, J Shakespeare

10.05am **S77**
Cardiovascular outcomes in chronic obstructive pulmonary disease, obstructive sleep apnoea and the overlap syndrome
AJ Shah, MQ Arfeen, S Denaxas, N Fitzpatrick, P Murphy, JR Hurst, S Mandal

10.20am **S78**
CPAP is Associated with improvements in Real-World Driving Performance and Sleep Quality in Obstructive Sleep Apnoea
K O Lee, T A Bekinschtein, I E Smith

9.00am - 10.35am
Albert, 2nd Floor
SPOKEN SESSION: S79 - S84

Advances in cystic fibrosis

Chaired by: Dr Suzanne Roche

9.05am **S79**
Development and validation of phenotyping algorithms for accurate identification of adults with cystic fibrosis in electronic health records
S Humfress, G Lip, F Frost

9.20am **S80**
Subjective evaluation of sputum characteristics shows poor interobserver reliability
R Gyte, SE Weinberg, G Einarsson, A Coy, F Frost, E Caamano Gutierrez, R Maher, AM Jones, MM Tunney, A Horsley

9.35am **S81**
Factors associated with respiratory exacerbations in adults with cystic fibrosis in the post-modulator era – a UK registry data study
R. C. Robey, Z. Gozde Sahin, S. Weinberg, J. Duckers, S. C. Charman, R Lord, A Jones, R Maher, M Tunney, E Caamano Gutierrez, A Horsley

9.50am **S82**
Associations Between Perception of Exacerbations, Viral Infection and Symptom Burden in Cystic Fibrosis
H Cheema, SE Weinberg, S Ahmad, E Caamano Gutierrez, S Doe, J Duckers, F Frost, AM Jones, R Lord, RE Maher, D Peckham, MM Tunney, A Horsley

10.05am **S83**
Lung function recovery following inpatient treatment of pulmonary exacerbations in adults with primary ciliary dyskinesia
B Powell, A Jones, P Barry, H Green

10.20am **S84**
Increasing age is associated with improved home spirometry quality and distinct error phenotypes in people with cystic fibrosis
A. Azam, R.J. Bright-Thomas, A. Horsley, P.J. Barry, A.M. Jones

9.30am - 10.15am
Victoria, 2nd floor
OPEN MEETING
Breathlessness

This is an inclusive session – all delegates are welcome.

10.00am – 11.00am
Whittle & Fleming and Britten, 3rd floor
COFFEE/TEA BREAK

10.45am – 12.30pm
Churchill, Ground floor
SYMPOSIUM
PLENARY SCIENTIFIC

Chaired by: Professor Mona Bafadhel (London) and Professor Nick Maskell (Bristol)

10.45 How to SELF-BREATHE
Dr Charles Reilly (London)

11.10 Adaptability of the Neutrophil
Dr Emily Watts (Edinburgh)

11.35 Repairing the lung: dissecting immune regulation of airway epithelial regeneration
Professor Chris Lucas (Edinburgh)

12.00 Improving cancer outcomes through early diagnosis
Dr Anna Bibby (Bristol)

Learning objectives:

1) To enhance understanding of non-pharmacological approaches to breathlessness self-management, and to introduce SELF-BREATHE, a co-designed digital intervention developed to improve patients' confidence and capability in managing breathlessness.

2) To learn about neutrophil adaptations in the lung microenvironment

Thursday 26 November 2026

3) To highlight the mechanisms that allow the airway epithelium to repair and regenerate following injury

4) To learn about new diagnostic approaches and strategies to in increase early detection in lung cancer and mesothelioma.

10.50am – 11.50am
Abbey, 4th floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Asthma

10.50am – 11.50am
Albert, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Pleural Disease

10.50am – 11.50am
Victoria, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Sleep

10.45am – 12.30pm
Windsor, 5th floor
SPOKEN: S85-S90
Randomised Controlled Trials

Chaired by Dr Alexandros Mathioudakis and Professor Janice Leung

10.45am **S85**
CORMORANT: Prospective diagnostic accuracy study of hand-held capnography using N-Tidal Diagnose compared to spirometry for the diagnosis of COPD in UK primary and community care settings
H F Ashdown, S Fleming, G Lambert, C Latimer-Bell, J Cook, P J Turner, C Roleston, M Glogowska, R Hallifax, K Woodward, E Kennington, L Campbell, J Carter, A X Patel, J Wolstenholme, C C Butler, G N Hayward

11.00am **S86**
Improving outcomes for primary care patients with undiagnosed or diagnosed COPD on monotherapy or less through targeted case-finding and proactive optimisation: results from the PREVAIL cluster-randomised clinical trial
M Singh, J R Hurst, A P Dickens, M Kerr, K Hickman, M G Crooks, L A Alves, C Ariti, M Bafadhel, M Bhutani, J Busby, V Carter, J D Chalmers, R Chen, A Couper, G Devereux, N Eleangovan, A Evans, D Freeman, C P Gale, M Gibson, D M G Halpin, W Henley, J Marshall, F Martinez, M Miravitlles, T Morris, H Muellerova, S Muro, J Natman, R Pullen, J K Quint, A Sharma, D Singh, D Skinner, D Stolz, D Varghese, E Vijaykumar, T Winders, T Wilkinson, D Price

11:15am **S87**
Tozorakimab for prevention of exacerbations in COPD: pooled analysis of the OBERON and TITANIA trials
C E Brightling, F C Sciurba, H Watz, A Bourdin, F J Martinez, M K Han, G J Criner, D Singh, A Papi, S P Bhatt, M Jenkins, M Fageras, R Patel, N Martin, I Psallidas

11:30am **S88**
IMPALA-2: A randomized, double-blind, placebo-controlled clinical trial of once-daily inhaled molgramostim in patients with autoimmune pulmonary alveolar proteinosis (aPAP)
C McCarthy, B C Trapnell, Y Inoue, F Bonella, T Wang, A Ataya, B Robinson, R Fleming, R Pratt, Y Wasfi

11:45am **S89**
Reintroduction of anti-tuberculosis therapy following drug-induced liver injury: study protocol for a randomised controlled trial (TB-DILI)
W S Lim, G Meakin, S Sadiq, C Partlett, G P Aithal, L Nicholson-Scott, A Montgomery, O M Kon, M Lipman, T Gorsuch, M Dedicoat, K T Shenoy, L K Balakumaran, P White, G Davies, L John, A Rich, A Elsharkawy, C Bruce

12:00pm **S90**
RAPTOR-f: a multicentre randomised feasibility trial of RAPID-guided management in pleural infection
A Elsheikh, M Bhatnagar, B Iqbal, E Hedley, D Addala, O Walsh, D Abdul-Rahman, F Al-arrayed, J Sadler-Ladell, T Panduro, M Dobson, I Mechie, C Pearce, A Budds, P Ridge, M Mwadeyi, C Hardy, W Talbot, T Sarkar, Z Sein, V Lennox, Y Feng, J Chu, N Kanellakis, C Daneshvar, J Corcoran, R Miller, G Collins, R Hallifax, E Mishra, N M Rahman, E O Bedawi

12.00pm – 2.15pm
Pickwick, 1st floor and Whittle & Fleming, 3rd floor
LUNCH (Lunch is not included in the delegate fee. Card payments only)

1.00pm – 1.45pm
Churchill, Ground floor
BTS CLINICAL LECTURE
TIMING IS EVERYTHING: WHAT HIV HAS TAUGHT US ABOUT WHEN TO TREAT

Professor Marc Lipman (London)

Introduced by: Professor Adam Hill (Edinburgh)

SCIENTIFIC PROGRAMME

1.00pm – 2.00pm
Albert, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING

ARTP / BTS Respiratory Physiology

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

Lung Cancer and Mesothelioma

2.15pm – 3.15pm
Churchill, Ground floor
SYMPOSIUM

BREATHING DANGER: COPD BEYOND TOBACCO

Chaired by: Professor Sarah Walmsley (Edinburgh) and Dr Marie Fisk (Cambridge)

14.15 Mechanistic insights into vaping and e-cigarette exposure in COPD
Professor Aaron Scott (Birmingham)

14.45 What are the multidimensional effects of cannabis smoking on the lung?
Professor Janice Leung (Canada)

15.15 Air pollution as a driver of COPD
Professor John Hurst (London)

Learning objectives:

1) To understand the cellular and molecular mechanisms by which vaping and e-cigarette exposure contribute to COPD pathogenesis

2) To evaluate the respiratory effects of cannabis smoking and its role in development and progression of COPD

3) To understand how air pollution contributes to the development, exacerbation and global burden of COPD

2.15pm – 3.45pm
Mountbatten, 6th floor
SYMPOSIUM

HIGHLIGHTS FROM THORAX/LANCET RESPIRATORY MEDICINE

Chaired by: Professor Jennifer Quint (Thorax), Dr Sheetu Singh (Thorax) and Bianca Brandon (Lancet Respiratory Medicine)
Selected articles highlighting the diversity of excellent respiratory research published by the journals will be presented by their authors and followed by a discussion with the editors.

Thursday 26 November 2026

2.15pm – 3.45pm
Windsor, 5th floor
SYMPOSIUM

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

Chaired by: Professor Mona Bafadhel, Professor Philip Molyneaux and Dr Amanda Tatler

2.30pm Pneumothorax: trends in epidemiology, impacting guidelines and future directions
Professor Rob Hallifax

3.00pm COPD, a disease of accelerated ageing. The causes and consequences
(30 minutes)
Dr Jonathan Baker

2.15pm - 3.50pm
St James, 4th Floor
SPOKEN SESSION: S91 - S96

From the old to the new: innovative physiological assessments

Chaired by: Dr Caroline Jolley and Professor Gerrard Rafferty

2.20pm **S91**
Investigation of a non-invasive, non-volitional index of diaphragm strength using twitch surface mechanomyography
X. Ma, N. N. Costa, M. R. G. Toscano, E. N. Kamavuako, G. F. Rafferty, C. J. Jolley

2.35pm **S92**
Beyond FEV1 for the early detection of COPD: Unlocking the silent zone through novel measurement of small airways function
NMJ Smith, B Langford, D Fleming-Brown, C Wilbur, A Wild, D Sandhu, G Richmond, M Tsakok, R Fox, HF Ashdown, GAD Ritchie, PA Robbins, ID Pavord, NP Talbot, N Petousi

2.50pm **S93**
Long Term Patterns of Aerobic Capacity Change in ACHD Patients
JB Burge, EP Parkes, DA Adamson, GT Talone, AZ Zhupaj

3.05pm **S94**
Measuring Respiratory Volumes with a 3D Depth Camera
A Handley, S Preece

3.20pm **S95**
Cardiopulmonary exercise testing insights into patients referred with unexplained dyspnoea
M Afzal, S Wallbanks, Y Khan, R Gregory, O Stackhouse, A Halder, R Mukherjee

3.35pm **S96**
Role of exercise in the functional capacity and clinical outcomes of smokers
King-Pui Florence Chan, Lam Priscillia Lam, Jackson Ka-Chun Leung, Yim-Wah Lynn Shong, Jin-Ning Clarabel Wong, Aaron Cheung, Macy Mei-Sze Lui, Siu Leung Fung, Ivan Fan-Ngai Hung, David Chi-Leung Lam

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2.15pm - 4.05pm
Westminster, 4th Floor
POSTER DISCUSSION: P71 - P85

Biologics galore!

Chaired by: Dr Paul Pfeiffer and Professor Rekha Chaudhuri

P71 A global clinician survey of OCS stewardship in asthma exacerbations: time for T2-guided treatment?
D L Sykes, S Couillard, R G Figueiredo, J Charriot, H Pott, A Manji, M Abdo, I Dimiene, W J Song, S Ramakrishnan, S Faruqi, H Cummings, M G Crooks

P72 Real-world exacerbation phenotyping in severe asthma treated with anti-IL5/5Ra, anti-IL4Ra and anti-TSLP
AM Bucataru, J Bourne, I Berrar Torre, B Wright, D Watchorn, P Kerr, R Despiney, P H Patel, F Yang

P73 Benralizumab does not alter the airway microbiome in severe asthma, but exacerbations are associated with increased dysbiosis identified by the $\log_2$ Pseudomonadota:Bacillota ratio
PAS Shah, KH Haldar, VM Mistry, JL Logan, ZSO Ong, KM Martin, LG Gillespie, AM McConnachie, WTNL Lee, TB Brown, SF Faruqi, DJJ Jackson, RK Kurukulaaratchy, AHM Mansur, DS Saralaya, SJF Fowler, PP Patel, JB Brown, JL Lordan, SS Siddiqui, SM Megremis, SAH Harrison, RB Brown, CN Nelson, VB Brown, JDC Chalmers, RD Djukanovic, IDP Pavord, LGH Heaney, SD Diver, RC Chaudhuri, CEB Brightling

P74 Benralizumab in the modern biologic era: comparison of clinical outcomes following the expansion of biologic treatment options in severe asthma
MG Gleeson, JG Gates, FH Harris, CR Roxas, JL Lam, LG Green, MF Fernandes, LT Thomson, GDA D'Ancona, JD Dhariwal, ANK Nanzer Kelly, DJ Jackson

P75 Tezepelumab in patients receiving benralizumab for EGPA - steroid sparing effect of dual biologic therapy
A Nanzer, L Green, C Roxas, J Lam, M Fernandes, L Thomson, J Gates, F Haris, M Fernando, T Ismail, J Dhariwal, G D'Ancona, D Jackson

P76 Effectiveness of Tezepelumab treatment in patients with severe asthma: real world outcomes from a severe asthma network
MD Drew, SK Khan, LW White, AM Mansur, AP Pillai, JS Sullivan, JM Marsh, EL Lyng, MS Sheego, SG Gilby, EV Verrier, BS Sutton, RB Berair, DS Subramanian, SM Message, PR Ryan, MD Doherty

P77 Malignancy outcomes in severe eosinophilic asthma patients treated with benralizumab across two UK centres
JL Romer, F Yang, DJ Jackson

P78 Effect of tezepelumab on type 2 inflammatory biomarker levels in the nasal lining fluid of patients with CRSwNP and comorbid asthma
BJ Lipworth, C Marchese, C Chen, A Foster, SS Ponnarambil, J Reddy, JR Parnes, J Publicover, JD Sherrill

P79 Effect of tezepelumab on lung function in oral corticosteroid-dependent patients with severe asthma and comorbid CRSwNP or CRSsNP in the phase 3b WAYFINDER study
DJ Jackson, P Chanez, NL Lugogo, M Gurnell, LG Heaney, S Korn, G Brusselle, R del Olmo, L Baylis, K Salapa, C Marchese, S Chandarana

P80 Treatment Response and Clinical Remission After One Year of Tezepelumab in Biologic-Naive Severe Asthma: A Real-World Study
Abdul Qadir Vapiwala, Hnin Aung, Shamsa Naveed

P81 Tezepelumab to Improve Symptom and Treatment Burden in Severe Asthma: An England Based Single-Centre Retrospective Cohort Study
AJF Orford, J To, E Idris

P82 A comprehensive assessment of the impact of anti-IL5 therapy on wellbeing, illness perception and cognition in severe asthma: longitudinal data from the CONTENTed study.
GW Nava, M da Silva, V Cobham, C Morgan, J Grenville, R Shrimanker, B Ainsworth, P White, PW Jones, TR Barrick, JW Dodd

P83 Dupilumab improves spirometry- and oscillometry-derived ratios indicating large and small airway function in patients with moderate-to-severe type 2 asthma: VESTIGE
BJ Lipworth, C Taube, P Rogliani, T Shirai, F Wen, FJ González-Barcala, A Mohan, A Altincatal, R Gall, H Awad, M Castro

P84 Twice-yearly depemokimab maintains exacerbation reduction and clinical remission over 2 years in patients with asthma, regardless of baseline asthma duration: SWIFT/AGILE integrated analysis
M Kraft, N Jarjour, R Buhl, C Tacon, D Muccino, CQ ZHU, DJ Jackson

P85 Never Too Old for Biologics? Real-World Outcomes in Severe Asthma Patients Aged 75 Years and Older
M Abououf, M Saleh, I Arnetoli, K Prior

2.15pm - 4.05pm
Moore, 4th Floor
POSTER DISCUSSION: P86 - P100

Respiratory infections: from disease burden to outcomes

Chaired by: Dr Heinke Kunst and Dr Huw Ellis

P86 Public Awareness and Understanding of Hospital-Acquired Pneumonia: a Cross-Sectional Survey
DK Jackson, DG Wootton, DG Wootton

P87 Fever-dependent WHO SARI criteria incompletely ascertain adult hospitalised respiratory viral infection
T G Morelli, M Purcell, A Freeman, TMA Wilkinson

P88 Viral Aetiology of ARDS in Neutropenic Patients with Malignancy: A Retrospective Cohort Study
Sarah Hereth Al Azawei, Halin Bruno, Zen Yap, Amina Abdulla

P89 Healthcare utilisation after hospitalisation for Influenza in England: a population-based study.
S Middleton, S Middleton, TM McKeever, TM McKeever, CE Bolton

P90 Implementation of digital tools to improve length of stay and antimicrobial stewardship in community-acquired pneumonia
MD Knolle, S Sarker, V Frechter, D Lee, R Santos, P Lynch, S Trivedi, T Gouliouris

P91 Not-so- 'Simple': Parapneumonic Effusion as an Independent Marker of Excess Mortality and Prolonged Hospitalisation in Pneumonia
M Bhatnagar, NM Rahman, DW Eyre

P92 Impact of statins on outcomes of older inpatients with community-acquired pneumonia
WJ Cheeng, Richard CH Davidson, Lucy Rimmer, John H Thompson, Thomas P Hellyer, Anthony J Rostron, A John Simpson, Wezi Sendama

P93 Value of procalcitonin in identifying alternative diagnoses in patients with suspected community-acquired pneumonia
SM Zubair, SS Nair, RV Reddy

P94 Diagnostic accuracy of an artificial intelligence system for identifying pulmonary tuberculosis on chest radiographs in a moderate incidence global north setting
P Gosal, C Juli, S Ellis, A Hakim, H Kunst, WM Ricketts

P95 MAC-PD, the cost of care: a comprehensive assessment of actual healthcare costs associated with Mycobacterium avium complex (MAC) pulmonary disease (PD)
F Dixon, D. J Dhasmana

P96 Understanding patient and health system delays in tuberculosis diagnosis
T Gandhi, S Matthews, A Dubey, N Hussain, M Nalmpanti, S Varma, S Khaliq, S Rahman, A Rahman, H Kunst

P97 Quantifying the Secondary Care Burden and Social Complexity of Tuberculosis to Support Community Pathway Improvement
Shobika Gomahan, Rory Mcdermott

P98 Title: Missed Opportunities for Tuberculosis Prevention: An Audit of LTBI Screening Among Recent Migrants Subsequently Diagnosed with Active Tuberculosis

S Khalil, M Siddiqui, M Ashwini, A Maliekal, A Ainley, A Rahman

P99 Social risk burden and treatment outcomes among patients with tuberculosis: a retrospective cohort study
S Matthews, T Gandhi, A Dubey, N Hussain, M Nalmpanti, S Varma, S Khaliq, S Rahman, A Rahman, H Kunst

P100 Tuberculosis (TB) related deaths:10 year analysis and comparison of outcomes between White UK-born and other ethnic groups
U Agboje, M Adamu, J Panayi, K Srinivasan, H Moudgil

2.15pm - 3.20pm
Abbey, 4th Floor
POSTER DISCUSSION: P101 - P109

Global and public health: future aspects with impact

Chaired by: Dr Nick Hopkinson and Dr Shamanthi Jayasooriya

P101 Reusing inhalers in lung function reduces cost and carbon footprint
LJE Smith, V Shah, L Morgan, R Tolhurst, C Curtis, L Desborough, C Wood

P102 Temporal trends in mortality due to alpha-1-antitrypsin deficiency across 30 European countries from 2011-2021
A Gupta, F Gonnelli, T M McKeever, F A Pearce, C E Bolton

P103 Lung function, asthma, and respiratory symptoms in Nepali children and adolescents: an analysis from Nepal Family Cohort Study (NeFCoS)
Dr Monica Shrestha, Prof Nagendra Chaudhary, Dr Ashesh Dhungana, Prof Charlotte E Bolton, Mr Prashil Raj Dali, Prof Paramjit Gill, Dr Ioannis Kyrou, Prof Harpal S Randeva, Prof Om P Kurmi

P104 Integrating Field Surveillance and Immunological Assessment to Enhance Tuberculosis Contact Tracing in The Gambia Insights From the TBVAC Study
ML Jatta, H Jobe, B Njai, OA Owolabi, A Sillah, M Sonko, J Sunderland

P105 Five-minute exposure to traffic-related air pollution and adherence of particulate matter on circulating red blood cells
GL Lekka, YR Rajeev, JG Grigg, NL Liu

P106 Cross-regional disparities in access to tuberculosis hospital care in Indonesia, 2020–2024
Putri Shafirra Rakita, Zamharirah Tamzil

P107 High ambient temperature and respiratory emergency admissions during the warm season: a daily time-series analysis
J Liao, K Anand, T Riveron, K Eminson, R Panchal, C Jephcote, AL Hansell, NS Ray-Bennett, JD Vande Hey, YS Cai

P108 Reducing the environmental impact of inhaler disposal in secondary care: A Quality Improvement Project
A.H Hamdani, F.S Suliman, H.R Ridha, C.S Scott, M.L Leighton, J.N Nixon

P109 Respiratory Implications of Recent Weight Loss Pharmacotherapies: A Narrative Review of GLP-1 and Dual GIP/GLP-1 Receptor Agonists
Aarthy Aravind, Thinuri Mendis, Lakshmi Saranya

2.15pm - 3.50pm
Rutherford, 4th Floor
POSTER DISCUSSION: P110 - P122

The breath of childhood: science, innovation and care

Chaired by: Dr Rebecca Thursfield and Dr Chris Hine

P110 Impact of a clinician referral pathway to Citizens Advice Service in addressing health inequalities in paediatric respiratory medicine
C Pearson, J Livesey, E Sheppard, S Baker, S Kansra

P111 Short-term air pollution exposure and pediatric respiratory hospital admissions in Athens: a time-series study
G Kontonasiou, A Analitis, D Stamatiou, K Dimakopoulou, E Verykakis, A Polytarchou, N Mihalopoulos, N Mihalopoulos, E Samoli, A Karakatsani

P112 Short-term Air Pollution Exposure and Risk of Airway Inflammatory Response in Children (CHERISH): A Randomised Mixed Factorial Study.
S Moloney, A C Thomson, I Gonzalez Calvo, H Hajmohammadi, H E Wood, M I Mead, I S Mudway, G Mosler, J Scales, A Whitehouse

P113 What is the association between household environmental exposures and respiratory

function among children aged 4-16 attending the Air Pollution Clinic? A Cross-Sectional, Observational Study
A Parmar, A Whitehouse, J Kennedy

P114 Endotracheal aspirates demonstrate higher bacterial biomass and similar microbiota composition compared with bronchoalveolar lavage in children with chronic wet cough
S Campbell, K Gerasimidis, S Milling, R Langley

P115 Treatment patterns and 12-month outcomes after acute preschool wheeze presentations: the PICTURE study
Jui Andharia, Lubna Wajid, Sejal Saglani, Prasad Nagakumar

P116 A tough pill to swallow - assessing the impact of virtual pill school on children with cystic fibrosis.
D Fisher, M Sathiyakumar, N Murray, M Ovens, IM Balfour-Lynn

P117 Predictive accuracy of STARWave, Difficulty Drawing Breath, and Feverkidstool scores for hospital admission in children with suspected lower respiratory tract infection (LRTI): a retrospective cohort study
KB Bansal, CC Chan, IG Garcia, NL Leek, VB Bansal, LH Hole

P118 Current Practices and Barriers in Paediatric Non-Invasive Ventilatory Support: A Multi-Centre Survey
ASY Chee, R Langley, P Davies, J Twynam-Perkins, P Burns, A Brown, E Buchan

P119 Outcomes in children with Trisomy 21 undergoing drug-induced sleep endoscopy due to persistent obstructive sleep apnoea post-adenotonsillectomy.
L Venditto, O Wright, E Chan, V Patelis, L Sutton, B Ahmed

P120 Variation in Immunological Investigation Following Paediatric Empyema and Necrotising Pneumonia
MF Farinha, EP Potter, SK Kansra, BS Shillitoe

P121 Outcomes following paediatric lung transplantation for childhood diffuse parenchymal lung disease in the UK
HM Makrinioti, BA Ahmed, MT Thomas, MB Brodlie, RB Brugha

P122 Opportunity for ex-vivo lung perfusion to increase paediatric lung transplantation
R Brugha, SJ Mead-Regan, H Spencer

Thursday 26 November 2026

2.15pm - 4.00pm
Cambridge, 5th Floor
POSTER DISCUSSION: M16 - M31

Real world pleural medicine: improving care outcomes, training and delivery

Chaired by: Dr Poppy Denniston and Dr Anand Sundaralingam

M16 Correlation of chest CT patterns with pleural fluid biochemistry, inflammatory markers and microbiology in adult pleural infection: an exploratory study.
A Elsheikh, M Dembele, M Bhatnagar, O Walsh, D Abdul-Rahman, F Al-arrayed, Y Feng, J Chu, N Kanellakis, R Hallifax, E Mishra, N M Rahman

M17 Management of Primary Spontaneous Pneumothorax before and after implementation of 2023 BTS pleural disease guidelines: experience from a tertiary centre
CK Ho, S Mok, TQ Tuen, S Shakir, B Pippard

M18 Clinical outcomes of pleural infection in a tertiary respiratory centre despite high adherence to British Thoracic Society Guidelines for pleural disease.
A Elsheikh, M Bhatnagar, O Walsh, D Abdul-Rahman, F Al-arrayed, M Samarage, J Cabildo, J Sadler-ladell, R Hallifax, E O Bedawi, A Moore, E Mishra, N M Rahman

M19 Waiting times to Medical Thoracoscopy and its impact on outcomes: a retrospective descriptive study at a tertiary care respiratory center
S M Hettiarachchi, B Vernon, S Muhammed, S Gudur, M Munavvar

M20 Indwelling pleural catheter versus medical pleurodesis in malignant pleural effusions: UK-based single-centre experience over 3 years
M Lu, A Sinha, S Bala, Y Myint, M Selvan, T Win, A McMillan, S Lok

M21 Local MIST- foggier than expected: A real world view on management of pleural infection
MZ Zakariya, S Gudur

M22 Do elective admission pathways improve outcomes in definitive management of malignant pleural effusion? A retrospective cohort study
EC Coleby, OR Walsh, I Rahman, AN Hipgrave, NM Rahman

M23 Indwelling pleural catheters for the management of persistent air leaks: a retrospective review
R Westley, S Johnstone, F Hinchcliffe, N Newnham, A Yousuf, S Mohammad, R Sudhir, R Panchal

M24 Cell-free DNA in suspected malignant pleural effusion: diagnostic and molecular applications
D Abdul-Rahman, L Watts, N Kanellakis, B Iqbal, A Elsheikh, O Walsh, M Bhatnagar, R Hallifax, E Bedawi, NM Rahman

M25 Practice of Intrapleural fibrinolytic and enzyme therapy (IET) in adult patients on veno-venous extracorporeal membrane oxygenation (VV ECMO) - a case series
AD D'Souza, BI Iqbal, JL Liang, OK Kadwani, PD Denniston, GG Glover

M26 Reviewing National Pleural Services Through a Safety-II Lens: recommendations for the ideal pleural unit
Janey McKane, Erin Gaughan, Avinash Aujayeb, Sarah Galbraith

M27 Higher intercostal drain dislodgement rates outside respiratory medicine: A multicentre service evaluation
H G Kamil, Q Gohir, Q Shahzad, C Jayawardena, F Ali, H Asif, F Afridi, M Murshed, M Nomani, M A Khalil, J Bhat, M Haris

M28 Out-of-hours pleural procedures: a UK national survey of general medical, emergency medicine and intensive care registrars
D Jajbhay, A Aujayeb, P Denniston, M Juniper, H Shotton, J Liang, J Zhang, O Kadwani

M29 UK Pleural practice review – still not getting it right
J Zhang, A Loveridge, A Aujayeb, S Mahendran

M30 Confident at the bedside, unsupported by the system: a multi-centre survey of awareness, training and self-reported practice in pleural procedures across 17 East of England hospitals following the 2023 BTS guidance
MAZ Zahid, GM Movio, SB Bala, BA Arifaj, MS Sait-rosenberg

M31 Thoracic ultrasound training in the UK: a snapshot perspective
R Westley, R Panchal, A Aujayeb

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

COFFEE/TEA BREAK

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

Global Lung Health (Task and Finish Group)

4.15pm – 5.45pm
Churchill, Ground floor
SYMPOSIUM

MALIGNANT PLEURAL EFFUSION – FROM BIOLOGY TO OUTCOMES

Chaired by: Professor Kevin Blyth (Glasgow) and Dr Eleanor Mishra (Norfolk and Norwich)

16.15 The biological effects of pleural fluid on malignant progression - translational evidence on mechanism
Dr Dinesh Addala (Oxford)

16.45 Changing the paradigm for IPCs and Thoracoscopy- the AMPLE 3 RCT results
Prof Gary Lee (Australia)

17.15 Outcomes in randomised trials of malignant effusion - core outcomes and patient priorities
Alexandra Dipper (Bristol)

Learning objectives

1) To understand the current scientific evidence on pleural fluid biological effects on malignant growth

2) To review evidence on optimal treatment of MPE including surgical options on the basis of RCT data

3) To review best outcomes in clinical trials of malignant effusion, and how these should be applied in clinical practice

SCIENTIFIC PROGRAMME

4.15pm – 5.45pm
Mountbatten, 6th floor
SYMPOSIUM

JOINT BTS/BPRS SYMPOSIUM

SMART LUNGS, SMART CARE: THE PRESENT AND FUTURE OF DIGITAL HEALTH IN PAEDIATRIC RESPIRATORY DISEASE

Chaired by: Dr Hazel Evans (Southampton) and Dr Don Urquhart (Edinburgh)

16.15	Digital Monitoring in Paediatric Sleep and Long-Term Ventilation: Transforming Assessment, Adherence, and Outcomes Dr Hui Leng Tan (London)
16.45	Smart Monitoring in Childhood Asthma and Bronchiectasis: From Early Detection to Personalised Care Dr Prasad Nagakumar (Birmingham)
17.15	From Innovation to Implementation: challenges in implementing Digital Respiratory Technologies into Routine Paediatric Practice Professor Heather Elphick (Sheffield)

Learning objectives:

1) Understand how digital technologies are used to monitor paediatric respiratory disease, sleep, and long-term ventilation

2) Evaluate the clinical value of remote monitoring for early detection and personalised management

3) Identify key challenges and enablers for implementing digital respiratory technologies in routine paediatric practice

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4.15pm – 5.45pm
Windsor, 5th floor

THE PHYSIOLOGY OF DYSPNOEA AND WHY PATIENTS FEEL WHAT THEY FEEL

Chaired by: Dr Karl Sylvester (Cambridge) and Dr Helen Ward (Wolverhampton)

16.15	How the brain constructs dyspnoea Dr Kyle Pattinson (Oxford)
16.45	Insights from exercise and disease Paul Burns (Glasgow)
17.15	From sensation to intervention Dr Lucy Speakman (Oxford)

Thursday 26 November 2026

Learning objectives

1) Understand that disorders of respiratory control are implicated in a wide range of diseases such as COPD, asthma, sleep apnoea and sudden infant death syndrome to name a few

2) Be knowledgeable in the use of exercise in the assessment of diseases associated with dyspnoea

3) Understand how the science behind breathlessness then translates into intervention and improved clinical outcomes including quality of life

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4.15pm - 5.50pm
St James, 4th Floor
SPOKEN SESSION: S97 - S102

More than a barrier: the airway epithelium

Chaired by: Dr Rachel Clifford and Professor Emily Swindle

4.20pm	**S97** Limited IL-5 receptor expression is associated with absence of direct antiviral effects of IL-5 in asthma A Nikonova, E Fiorenzo, C Chan, S Cayir, MA Ye, T Kebadze, J Aniscenko, SL Johnston
4.35pm	**S98** JAK inhibition attenuates rhinovirus-driven interferon responses in COPD airway epithelium without enhancing viral replication: a single-cell transcriptomic study H Guo-Parke, O Cappa, M Glenn, M McKelvey, A Mousnier, A Krasnodembskaya, J Kidney, D Simpson, D Linden
4.50pm	**S99** ORMDL3 regulates non-typeable Haemophilus influenzae infection in airway epithelial cells H Huang, S Chi, J Bozeman, K Ito, W Cookson, M Moffatt, Y Zhang
5.05pm	**S100** ORMDL3 regulates HMOX1 expression and lipid peroxidation for ferroptosis in human airway epithelial cells S Chi, H Zhao, D Ma, K Ito, W Cookson, M Moffatt, Y Zhang
5.20pm	**S101** Epithelial-fibroblast crosstalk

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induces transcriptional differences in Smoothened-Hedgehog signalling in COPD-derived airway epithelial cells
Telma Sancheira Freitas, Sneha Pinto, Yashwanth Subbannayya, Marine Petit, Lindsay Broadbent

5.35pm **S102**
A multicellular human COPD bronchial organoid model recapitulates epithelial dysfunction, endothelial disorganisation and cellular senescence
H Guo-Parke, HIA Alhamdan, JC Kidney, A Krasnodembskaya, DA Linden

4.15pm - 5.50pm
Westminster, 4th Floor
SPOKEN SESSION: S103 - S108

Lung infection: that's just (a)typical

Chaired by: Professor Michel Loebinger and Ms Jennie Keane

4.20pm **S103**
Radiological findings, inter-observer agreement and imaging burden in patients with possible non-ventilator associated hospital acquired pneumonia randomised to CXR or CT.
H Clay, F Khalid, J Hare, K Irwin, C McLeavy, E Thwaite, S Aston, R Bazaz, K Edwards, J Hucker, H Singh, J Howells, D Wootton

4.35pm **S104**
Exploring costs associated with suspected non-ventilator-associated Hospital-Acquired Pneumonia (nv-HAP) within the HAP-FAST trial
P Evans, A Guldemond, I Powell, A Jones, S Willshaw, A Tao, J Wood, N Shafiqa, E Adegbenro, K Cook, D Mclaughlan, H Singh, K Kalapurakal, N Senior, S Aston, L Pilling, R Bazaz, S Trewick, I Poku, A Alvarez Nishio, E Lam, DG Wootton

4.50pm **S106**
The British Thoracic Society Multidrug Resistant-Tuberculosis Clinical Advice Service: Activity Summary 2018-2025
P Haldar, P Papineni, S Howard, M Dedicoat, OM Kon, G Bothamley, T Capstick, S Coles, S Hindle, M Lipman, A Mackin, F Perrin, E Robinson, M Shadab

5.05pm **S107**
A randomised trial evaluating adherence and safety of rifapentine-based TB preventive treatment
T Duong, Y Hamada, A Kilic, S Assayed, R Horne, H Kunst, M Lipman, M Mandelbaum, J Brigden, K Sanders, D Zenner, I Abubakar, MX Rangaka

5.20pm **S108**
Microbiologic outcomes and recurrence in patients with newly diagnosed Mycobacterium avium complex lung disease (MACLD) in the phase 3b ENCORE trial
J. van Ingen, E. Van Braeckel, E. Van Braeckel, J. Feng, J. Feng, L. Morgan, L. Morgan, L. Morgan, C.L. Daley, P.A. Flume, M.R. Loebinger, J.D. Chalmers, D. May, P. Wu, M. Nevoret, D.W. Yuen

4.15pm - 6.00pm
Moore, 4th Floor
POSTER DISCUSSION: P123 - P137

Asthma or something else?

Chaired by: Professor Adel Mansur and Dr Sonay Beyatli

P123 Optimising diagnostic test thresholds and pathways for adult asthma
M Bennett, E Barrett, R Wang, L Healy, A Simpson, S Drake, C Murray, S Fowler

P124 Fractional Exhaled Nitric Oxide and Airflow Variation in Adults Without Respiratory Disease: Cross-Sectional Analyses in Singapore and U.S. NHANES
A Yii, I Fong, N Sieow, XN Choo, WJ Chen, MS Koh, J Koh, TR Tay

P125 Symptom profiles and diagnostic patterns in a multi-morbid respiratory cohort: findings from the SMART cohort
SA Farrell, C Ottewill, CM Gill, PJ Kerr, V Brennan, H Doherty, O Smith, E MacHale, B Cushen, D Ryan, G Greene, RW Costello

P126 Multimorbidity clusters and associated characteristics in mild, moderate and severe asthma cohorts
R Wang, C Gill, C Otterwill, A Simpson, C Murray, R Kurukulaaratchy, HM Haitchi, B Ainsworth, S Farrell, SJ Fowler, R Costello, A Freeman

P127 Beyond the Wheeze: A Phenotype-Driven Approach to Asthma Exacerbations
AP Pradhan, KS Shaw, JE Eastham, MM Martin, RH Hall, TM McKeever, IS Sayers, TWH Harrison

P128 Longer asthma duration is associated with elevated non-T2 sputum biomarkers and reduced T2 inflammation in severe asthma
F Yang, S Seo, T Hasegawa, CI Bloom, N Zounemat-Kermani, PK Bhavsar, IM Adcock, S Won, TB Kim, KF Chung

P129 Early Identification of "High-Risk" Asthma and progression to bio-eligibility: A Retrospective Cohort Study Using UK Data
LC Coyle, JB Busby, PJMcD McDowell, CR Redmond, DP Price, GAD Davies, LGH Heaney, JKQ Quint

P130 Digitally assessed home FEV1 and inhaler adherence to identify severe asthma as a cause of poorly controlled asthma
C Ottewill, C Gill, P Kerr, S Farrell, C Campbell, M Gleeson, O Smith, H Doherty, E Mac Hale, V Brennan, B Cushen, G Greene, RW Costello

P131 Sarcopenia in asthma: a cross-sectional assessment of muscle strength, mass and physical performance Reem Alajmi, Ian J. Clifton, Daniel Peckham, T. Scott Bowen, Oliver J. Price
Reem Alajmi, Ian J. Clifton, Daniel Peckham, T. Scott Bowen, Oliver J. Price

P132 Blood eosinophil count is associated with treatment response to oral corticosteroids for asthma attacks
Jiashu Shen, William Henley, Heath Heatley, Fleur Meulmeester, Samuel Mailhot-Larouche, Sanjay Ramakrishnan, Michael Wechsler, Guy Brusselle, Sarah Diver, Christopher Brightling, Mario Castro, Nicola Hanania, David Jackson, Neil Martin, Alison Moore, Peter Howarth, Megan Hardin, Rebecca Gall, Cecile Holweg, Vennila Dharman, Richard Beasley, Jacob Sont, Ewout Steyerberg, Timothy Hinks, Ian Pavord, David Price, Simon Couillard, Imran Howell

P133 When to send in the troops - Optimal timing for Breathing Pattern Disorder Assessment in Severe Asthma
J Harrison, A Folwell, K Prior

P134 Laryngeal dysfunction in a district general hospital asthma clinic: a service needs assessment in an urban population
R Rathbone, J Gudenus-Oribe, P E Pfeffer, A P Hearn

P135 Sound and vision: do video-recordings with sound impact diagnostic accuracy and inter-rater agreement of a diagnosis of Inducible laryngeal obstruction?
CD Slinger, B Tidmarsh, R Slinger, A Ali, H Lever

P136 Implementation of a provocation laryngoscopy service within a difficult asthma clinic: a service evaluation at one year
J Gudenus-Oribe, A Ramdin, P E Pfeffer, A P Hearn

P137 Excessive Dynamic Airways Collapse (EDAC): An under-recognised cause of symptoms in the severe asthma clinic?
Y L Pang, P Smith, M Sovani, SA Nouraei, M J Martin

4.15pm - 6.00pm
Abbey, 4th Floor
POSTER DISCUSSION: P138 - P152

Novel approaches to improving COPD care: imaging, AI and acute intervention

Chaired by: Dr Hnin Aung and Dr Lydia Finney

P138 Cardiopulmonary Event Outcomes and Risks in COPD according to GOLD stage - An Observational Study in UK Primary and Secondary Care
D Shrikrishna, MJ Hale, M Watt, J Lipworth, DMG Halpin, CP Gale

P139 Opportunistic Thoracic CT-AI body composition as a Sarcopaenia case-finding flag in respiratory disease: DXA validation
D P O'Riordan, P Novotny, R Evans, E Daynes, C Brightling, N Greening, K Ntotsis, H McAuley

P140 Is lung volume reduction candidate case finding using quantitative computed tomography feasible in lung cancer screening?
TS FitzMaurice, M Abdulhakeem, M Diab, M Ledson, F Frost, S Chee, M Shackcloth

P141 Regional Mucus Plug Burden Predicts Adverse Outcomes in COPD Patients Identified Through Lung Cancer Screening: An Automated Quantitative CT Analysis
MZ Zahid, SW Wang, RC Chapman, JM McCabe, YC Chen, MA Azimbagirad, TY Yang, DC Cheng, AS So, PV Vasudev, JL Lim, GB Baxter, DY Yamada, RE Egashira, AS Szmul,

AB Bhamani, TP Patrick, AN Nair, SUMMIT Consortium, SJ Janes, JH Hurst, JJ Jacob

P142 Systemic cardiovascular burden in COPD: CT-detected aortic and coronary atherosclerosis as a predictor of frequent exacerbations.
C. Amaral De Sousa, L. Bravo Quiroga, M. Moreno Justicia, E. López González, M. Jiménez Méndez, R. Duque Alzate

P143 A study of use of MRI to measure modulation of eosinophil related inflammation in COPD
SJ Kirby-Smith, H Marshall, L Watson, LJ Smith, PJC Hughes, GJ Collier, AM Biancardi, LC Saunders, J Bray, NJ Stewart, R Thompson, JM Wild, R Lawson

P144 50 Shades of Green: Proof-of-Concept for Digital Sputum Colour Quantification
F Barker, L Jackson, D Wat, D Nazareth, K Mohan, J Fothergill, D Wootton, F Frost

P145 Abstract Withdrawn
E Dogu, N Smallcombe, M Bafadhel

P146 Impact of prophylactic azithromycin on hospital admissions, inpatient bed days and community steroid use in chronic respiratory disease: a retrospective service evaluation.
F Hopkins, O Dixon, F Khan, K Ibrahim, E Taylor, H Brierley

P147 Aminophylline during an acute exacerbation of obstructive airways disease: Is it worth it?
Mujtaba Kaouas, Omna Khawaja, Moez Nayyar

P148 A Pilot Study of the Effect of Metformin on Airway Glucose in COPD: The Metformin and Airway Glucose In COPD (MAGIC) Trial
E Fiorenzo, JS Tregoning, I Fahidi, MR Edwards, T Kebadze, P Mallia, SL Johnston, HA Farne

P149 Equity of Access to Virtual Ward (VW) Care for Exacerbations of COPD: A Retrospective Cohort Study
B Alenazi, Cat Atkin, S Gallier, Liz Sapey

P150 Acute exacerbation of COPD: Standard inpatient care versus virtual ward stepdown care in a District General Hospital
J Carter, R Whelan, J Zhang, E West, L Sridaran, L Gajendran, HA Go, S Harvey-Porter, A Loveridge, V Smith

P151 Virtual Oxygen Wards For Individuals Recovering From Acute Respiratory Illness: A Real-World Observational Study
Y Rahim, T Ward, N Haroun, C Nyein-OO, J Danns, M Naeem, G Tsaknis, RV Reddy

P152 Influence of Admission Troponin during COPD exacerbation and Survival
L Lindsay, A Harte, S Shahid, S McNeill, J Cannie, P Minnis

4.15pm - 6.00pm
Rutherford, 4th Floor
POSTER DISCUSSION: P153 - P167

ILD: cohort studies and service improvement

Chaired by: Dr Shaney Barratt and Dr Katherine Myall

P153 Pulmonary Hypertension in Nintedanib-Treated Fibrotic ILD: Real-World Prevalence and Safety in a Theoretically At-Risk Cohort from a Single Centre Experience
H de Silva, D Tilakaratne, A Hayley, J Derry, V Nanda, B Vijayakumar, LMH Bandara

P154 PPFE: Pulmonary Function, Nutritional Status and Outcomes from a single center
Wint Soe, Maheen Iftikhar, Samuel Healy, Lovena Nawoor, Adam Wallis, Sowkhya Ramachandraiah, Fiona Thompson, Michael Ball, Anna Donaldson, Suresh Babu

P155 Survival after surgical lung biopsy in interstitial lung disease: influence of histopathological diagnosis. A retrospective cohort study.
MH Hafeez, ZA Azad, DA Anderson

P156 Rituximab in connective tissue disease–associated interstitial lung disease : A Real-World Cohort Study
D Chatzis, C Koukouvitaki, S Flouda, K Chavatza, A Banos, F Karageorgas, DT Boumpas, S Loukides, E Manali, A Fanouriakis, V Tzilas

P157 The Interdisciplinary Interstitial Lung Disease (ILD IN) group highlights the need to optimise the ILD specialist practitioner's role across the ILD Clinical Pathway
M Koulopoulou, L Fabbri, R Boorsma, L Youngs, N Hussein, C Walker, A Fahim

P158 Expert ILD review improves diagnostic confidence and reduces unclassifiable disease in fibrotic ILDs
AT Trachalaki, MK Kokosi, FC Chua, VK Kouranos, ER Renzoni, SD Desai, AD Devaraj, RH Hewitt, PG George, AUW Wells

P159 Regional interstitial lung disease MDT collaboration between a tertiary centre and district general hospitals: acceptability, feasibility, and educational impact
K Abernethy, K Osborne, S Crawley, N Chen, R Boorsma, K Brignall, K Myall, A West

P160 Incidence, Prevalence and the Diagnostic Odyssey in Rare Cystic Lung disease
SNB Abd Hadi, TM McKeever, SR Johnson

P161 Real-World Nintedanib Outcomes Following Antifibrotic Eligibility Expansion and ILD Service Redesign at a UK Specialist Centre
N Khine, N Khine, A Laith, L Elton-Walters, P Prime, M Pipet, S Quarrell, E Hunter, S V Fletcher, S V Fletcher, R Limbrey, B Marshall, B Marshall, K Spinks, T Wallis, T Wallis, M G Jones, M G Jones

P162 Prevalence and Clinical Management of Patients with ILD Associated with PH – A UK Regional ILD Centre Perspective
CS Crook, J Francesqui, SC Stanel, P Hughes, J Lentaigne, S Lines, C Young, MA Gibbons, MA Gibbons, MA Gibbons, P Rivera-Ortega

P163 Navigating Treatment Intolerance: Real-World Outcomes of Antifibrotic Switching in IPF and PPF
J Lim, G O'Sullivan, I Haydar, MT Henry

P164 The clinical value and cost-effectiveness of routine blood monitoring in patients on pirfenidone for idiopathic pulmonary fibrosis: a retrospective cohort study
J Waddon, C Hawkins, K Kemp

P165 Anti-carbamylated protein antibodies in Fibroinflammatory lung Disease including Rheumatoid Arthritis & Sarcoidosis
A Liu, C Tan, R Gilpin, JA Doherty, S Carmichael, B Schock, J Burns, P Minnis

P166 Intravenous Methylprednisolone in Acute Exacerbations of Interstitial Lung Disease: A Single-Centre Experience
S Liyanage, V Joshi, N Ramsundar, E Ioannou, S Arul

P167 Survival after antifibrotic initiation in rheumatoid arthritis-associated usual interstitial pneumonia compared with idiopathic pulmonary fibrosis: A real-world retrospective cohort study
MH Hafeez, DA Anderson, GJ Jayasekera

Thursday 26 November 2026

4.15pm - 5.50pm
Albert, 2nd Floor
POSTER DISCUSSION: P168 - P180

Bronchiectasis unconfined

Chaired by: Ms Rachel Daley and Dr Fiona Mosgrove

P168 Immune signatures and inflammatory cell profiles differ between diseased and non-diseased lung segments of anatomically heterogenous bronchiectasis
R Nixon, J Pinkerton, A Kamenova, C Carter, K Myncham, M Jackson, C Orton, L Chan, A Tana, J Tonkin, M Kusumoto, L O, R Snelgrove, J Garner, A Singanayagam, A Shah

P169 Clinical, physiological and microbiological characteristics by Type 2 inflammatory status in bronchiectasis.
A. Vrouvaki, M. Moustaka Christodoulou, S. Loukides, E. Fouka

P170 The diagnosis detectives: improved recognition of aetiology in bronchiectasis through a regional diagnostic clinic
N Johl, C Addy, A Smith, A Sayers, J Duckers

P171 Review of the management plan of new patients seen in the bronchiectasis clinic: a retrospective observational study.
N Iqbal, S Kalam, L Arshad, E Avraam

P172 Understanding Barriers to Recruitment in Bronchiectasis Human Challenge Study: A Qualitative Analysis
FA Ahmed, MO Omer, CC Charlotte Carter, SLJ Johnston, AS Shah

P173 Bronchiectasis doesn't come with a user manual - do interdisciplinary conversations improve decision making?
S Wigmore-Sykes, J Harrison, A Horsley, K Prior

P174 Feasibility and acceptability of using a Shared Decision Making intervention to support patient choice of Airway Clearance Techniques in bronchiectasis
P McCallion, J Bradley, J Lally, L Robinson, A De Soyza

P175 Airway clearance therapy switching patterns in Primary Ciliary Dyskinesia: A tertiary center cohort study.
S A Jagtap, S Mahmood, A Sawant

P176 Nebulised Therapy as an Independent Driver of High Intensity Airway Clearance in Bronchiectasis
S Mahmood, M S Rashid, A P Sawant

P177 Abstract withdrawn

P178 Outcomes of IV antibiotic monotherapy for Pseudomonas aeruginosa in bronchiectasis: A 10-year OPAT cohort
N. Flora, L. Bedford, C. Sander

P179 Serum Azurocidin-1 is associated with frequent exacerbations in patients with bronchiectasis: Preliminary data from the EMBARC-BRIDGE study
Jiahui He, Merete Long, James Chalmers

P180 LiveBE: Living life with Bronchiectasis - results from a patient survey on how Bronchiectasis affects patient lives.
H Lind, E M Ward, J D Chalmers

5.45pm – 7.00pm
Britten, 3rd floor

BTS PRESIDENT'S RECEPTION

All participants registered to attend on Thursday 26 November are warmly invited to attend

SCIENTIFIC PROGRAMME

8.00am – 9.00am
Whittle & Fleming, 3rd floor
COFFEE/TEA

8.45am – 2.00pm
Whittle & Fleming, 3rd floor
POSTER VIEWING

Authors present: 10.00am – 11.00am

P181-P195
Innovation across the lung cancer diagnostic pathway
Discussion of abstracts will take place from 1.30pm to 3.30pm in the St James, 4th floor

P196-P211
Ventilation: frailty and comorbidity in patients with respiratory failure
Discussion of abstracts will take place from 1.30pm to 3.20pm in the Westminster, 4th floor

P212-P221
Opportunities to improve asthma care
Discussion of abstracts will take place from 1.30pm to 3.15pm in the Moore, 4th floor

P222-229
Advances in thoracic surgery and tobacco harm reduction
Discussion of abstracts will take place from 1.30pm to 2.35pm in the Abbey, 4th floor

P230-244
Improving every breath: pulmonary rehabilitation advances
Discussion of abstracts will take place from 1.30pm to 3.15pm in the Rutherford, 4th floor

P245-259
Asthma in transition
Discussion of abstracts will take place from 3.45pm to 5.20pm in the St James, 4th floor

P260-272
Type 2 COPD: biomarkers, biologics and outcomes
Discussion of abstracts will take place from 3.45pm to 5.10pm in the Westminster, 4th floor

P273-P284
Advances in translational research
Discussion of abstracts will take place from 3.30pm to 5.00pm in the Moore, 4th floor

Friday 27 November 2026

8.45am – 5.15pm
Cambridge, 5th floor
MODERATED POSTER VIEWING
M32-M47
The future of sleep and physiological services
Discussion of abstracts will take place from 3.15pm to 5.00pm in the Cambridge, 5th floor

8.30am – 9.00am
Albert, 2nd floor
JOURNAL CLUB
TB

Chaired by Dr Pranab Haldar (Leicester)

Learning objective

To review the latest publications and evidence in Tuberculosis

9.00am – 10.30am
Churchill, Ground floor
SYMPOSIUM
FINDING YOUR NICHE: CELLULAR HETEROGENEITY IN ASTHMA

Chaired by: Professor Liam Heaney (Belfast) and Dr Maisha Jabeen (Oxford)

09.00	Overcrowding in the asthmatic airway: the consequences of bronchospasm Professor Jody Rosenblatt (London)
09.30	Greater than the sum of their parts: Mast cell – eosinophil interactions in asthma Professor Sven-Erik Dahlén (Sweden)
10.00	Upside down: Epithelial responses to eosinophil targeting Dr Hitasha Rupani (Southampton)

Learning objectives:

1) To understand the mechanobiological effects of bronchoconstriction on the airway epithelium, and how these processes may contribute to airways remodelling in asthma

2) To explore the role of mast cells in asthma immunopathology and how bidirectional interactions with eosinophils impact the effector function of both cells

3) To understand how targeting the downstream IL-5/ eosinophil pathway may lead to improvements in upstream epithelial function in asthma

9.00am – 10.30am
Windsor, 5th floor
SYMPOSIUM

WHEN AUTOIMMUNITY TARGETS THE LUNG

Chaired by: Dr Katie Ward (London) and Dr Rahul Mahida (Birmingham)

09.00 Mechanisms of autoimmunity in ILD
Professor Joanna Porter (London)

09.30 Navigating the spectrum of vasculitis in ILD
Professor David Jayne (Cambridge)

10.00 Phenotyping the myositis patient and tailoring management to specific subtypes
Dr Harsha Gunawardena (Bristol)

Learning objectives:

1) Understand range of vasculopathy in systemic sclerosis and novel targets

2) Spectrum of vasculitis of lung and key management principals, including newer treatments on the horizon

3) To understand how clinical phenotypes may alter disease outcomes and enable tailored treatment in myositis-spectrum lung disease

9.00am – 10.30am
Mountbatten, 6th floor
SYMPOSIUM

CYSTIC FIBROSIS: NEW INSIGHTS FROM INFECTION TO INFLAMMATION

Chaired by: Professor Robert Gray (Glasgow) and Dr Dawn Lau (Cardiff)

09.00 Covering changing microbial ecology and potential new approached such as phage therapy
Professor Jo Fothergil (Liverpool)

09.30 What can we learn from large animal models of CF and non-CF Bronchiectasis?
Professor David Stoltz (USA)

10.00 Exploring how understanding non-pathogen driven autoinflammation may be the key to treating residual inflammation in the CF lung following CFTR modulator therapy
Professor Daniel Peckham (Leeds)

Learning objectives:

1) To understand how lung microbiology has changed in CF and how new approaches are needed in anti-microbial therapy.

2) To learn how pre-clinical models in CF and non-CF muco-obstructive diseases can lead to the development of new treatments.

3) To determine the role of autoinflammatory pathways in CF and how they might be modulated

9.00am – 10.35am
St James, 4th Floor
OPEN SESSION

SUPPORTING WOMEN IN RESPIRATORY MEDICINE

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.

9.00am - 10.35am
Westminster, 4th Floor
SPOKEN SESSION: S109 - S114

Decoding lung disease through 'omics

Chaired by: Dr Kevin Liu and Dr Janine Gote-Schniering

9.05am **S109**
Single-cell and spatial dissection of airway niches reveal distinct immune and epithelial drivers of asthma phenotypes
W Lason, M Jabeen, D Agrawal, J Melhorn, A Fries, RM Schreiner, B Brassenyi, R Yemeni, X Zhang, M Greco, JC Knight, CA Dendrou, TSC Hinks

9.20am **S110**
Germline and somatic genetic sequencing reveal clonal haematopoiesis and rare, high-impact variants in IL-17RA in late-onset severe eosinophilic asthma
J Melhorn, N Jones, R Yamani, N Jakobsen, M Metzner, P Vyas, P Bradding, ID Pavord, TSC Hinks

9.35am **S111**
Single cell profiling of corticosteroid-responsive and non-responsive endotypes of severe asthma
MF Jabeen, W Lason, LC Garner, B Brassanyi, R Yamani, M Mahdi, P Klenerman, ID Pavord, CA Dendrou, TSC Hinks

9.50am **S112**
Single-cell transcriptomic analysis reveals the bronchoalveolar landscape in interstitial lung abnormalities
M D Andhari, H M J Chong, V Kanabar, R M Nunez, S Cass, D Freydina, R Lee, A Trachalaki, L Yazbeck5, F Chua, S Desai, A Devaraj, P George, C Orton, RJ Hewitt

10.05am **S113**
Initiators and drivers of lung fibrosis – insights from spatial biology and in vitro studies using alveolar organoids
VKN Nathwani, PW Weeratunga, TH Hu, LB Baghaarabai, JW Woo, CV Vuppusetty, CC Clelland, AA Antanaviciute, LPH Ho, VKN Nathwani, LB Baghaarabai

10.20am **S114**
A 3D Multi-Omics Atlas of Human Lung Fibrosis Defines Molecular–Architectural Niches
JA Bell, SK Robinson, R Pedley, J Norman, S James, I Reddin, S Jogai, D Johnston, J Thomas, J Parkin, R Allen, L Sayer, A Alzetani, B Marshall, T Wallis, G Thomas, DE Davies, G Marshall, K Geillinger-Kästle, P Seither, M Thomas, A Fabre, M Eyres, OL Katsamenis, MG Jones

9.00am - 10.35am
Moore, 4th Floor
SPOKEN SESSION: S115 - S120

Focused pleural medicine: advances in diagnostic and therapeutics

Chaired by: Dr David Arnold and Dr Helen Davies

9.05am **S115**
Small effusion, big impact: the role of ultrasound-guided aspiration of minimal pleural effusions in cancer diagnosis and staging
SM Zubair, M Naeem, G Tsaknis, RV Reddy

9.20am **S116**
Standardising pleural management in suspected malignant pleural effusion using unified pre-test probability and the RED score: a quality improvement project
A Fadel, H Al-Sarireh, A Alrayyes, N Chinnappa

9.35am **S117**
Malignant Pleural Effusion Liquid Biopsy Evaluation (MAPLE): Exploring the diagnostic utility of pleural fluid ctDNA
R Westley, K Page, RA Allsopp, MK Wadsley, M Abraham, A Baguneid, JA Shaw, R Sudhir, R Panchal

9.50am **S118**
Pleural complications associated with tyrosine kinase inhibitors
J. Treves, A. Warrier, J. Harrington, S. J. Marciniak

10.05am **S119**
Severity Meets Vulnerability: Integrating Frailty and RAPID for Risk Stratification in Pleural Infection
S Ahmad, A P Sawant

10.20am **S120**
Lipids in parapneumonic pleural effusions drive tissue factor–dependent thrombin generation in macrophages
E McNally, A Newing, J McGrath, K Slattery, A Dyer, A Rehill, P Klavina, R Preston, L Gleeson

Friday 27 November 2026

9.00am - 10.35am
Abbey, 4th Floor
SPOKEN SESSION: S121 - S126

NIV: why outcomes matter

Chaired by: Dr Michael Davies and Dr Swapna Mandal

9.05am **S121**
Diagnosis-Specific Patterns of Healthcare Utilisation and Outcomes Following Acute Non-Invasive Ventilation for Hypercapnic Respiratory Failure
A Ozsancak Ugurlu, G Pereira, B Tanveer, M Magashi-Ali, J Malla, A Halder, R Mukherjee

9.20am **S122**
The impact of adherence to long-term NIV in patients with COPD
A Bikov, B Csoma, N Al Ieman Haji Mostapha, A Bentley

9.35am **S123**
Beyond Age and Acidosis: Frailty Independently Predicts Inpatient Mortality After Acute NIV in a Two-Site Cohort
O Kafi, MS Chohan, S Afifi, A Ode, Z Ammar, V Spillane, T Dhlandhlara, R Mukherjee, S Nafisa

9.50am **S125**
Transcutaneous CO_2 Monitoring Versus Arterial $PaCO_2$ During Acute NIV in Acute Hypercapnic Respiratory Failure: A Systematic Review
SN Nafisa, MG Gordon, MM Miah, ZH Huma, MS Sovani

10.05am **S126**
Cost-effectiveness of accelerated versus standard weaning from NIV in COPD: an economic evaluation within a randomised controlled trial
K Brewin, MH Naseer, R Arries, ND Lane, A Scadding, D Ghosh, B Prudon, M Sovani, TM Hartley, A McCarthy, J Gray, E Pakpahan, SC Bourke

9.00am – 10.00am
Rutherford, 4th floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Nurse

9.00am – 10.00am
Victoria, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Specialty Trainee

9.15am – 10.15am
Albert, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Pulmonary Rehabilitation

10.00am – 11.00am
Whittle & Fleming and Britten, 3rd floor
COFFEE/TEA BREAK

10.30am – 11.30am
Rutherford, 4th floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
Acute and Complex Pulmonary Infections

10.30am – 11.30am
Gielgud, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING
COPD

10.45am – 12.15pm
Churchill, Ground floor
SYMPOSIUM
TRANSLATION IN PLEURAL DISEASE – IMPROVING UNDERSTANDING AND TREATMENTS

Chaired by: Professor Gary Lee (Perth) and Dr Selina Tsim (Glasgow)

10.45 Genetic mechanisms in pneumothorax and cystic lung diseases
Professor Stefan Marciniak (Cambridge)

11.15 Macrophage subtypes and cellular immunity in pleural infection
Professor Laura Gleeson (Dublin)

11.45 Pleural septation detection, formation and biology – insights from early phase studies
Dr Eihab Bedawi (Oxford)

Learning objectives:

1) To understand the importance and impact of genetic testing in patients with pneumothorax and cystic lung disease

2) To appraise the current state of the art in macrophage and cellular biology in pleural infection

3. To understand the current understanding of the meaning and formation of septations in pleural disease

SCIENTIFIC PROGRAMME

10.45am – 12.15pm
Mountbatten, 6th floor
SYMPOSIUM

THE USE OF PHYSIOLOGICAL DATA WITHIN SPECIALIST CLINICS

Chaired by: Dr Vicky Moore (Coventry) and Professor Luke Howard (London)

10.45 Specialist Nursing: Physiological data within severe asthma, the path to therapeutics
Katie Borg (Oxford)

11.15 Physiotherapy: Physiological data for dysfunctional breathing, a growing evidence base
Chelliah Paramasivan (Cambridge)

11.45 Physician Clinics: Physiology data in pulmonary vascular disease, what's new?
Martin Johnson (Scotland)

Learning objectives:

1) To be able to assess the evidence from various physiological tests that provide the pathway to diagnosis and therapeutics in severe asthma. To understand the specialist nursing role in assessing scientific evidence in this area for decision making in CNS clinics

2) To understand that breathing pattern disorder is becoming a more common and debilitating phenomenon and there is emerging evidence from physiology that can support and aid diagnosis and treatment via physiotherapy

3) To review what is new alongside current evidence for the use of physiological data in Pulmonary vascular disease, and how this is used in clinical practise for accurate diagnosis

10.45am – 12.15pm
Windsor, 5th floor
SYMPOSIUM

BTS AUDIT AND QUALITY IMPROVEMENT

Chaired by: Dr Andy Molyneux, (Mansfield)

10.45 New Assessment Pathways for patients attending hospital with exacerbations of COPD or Asthma
Dr James Dodd (Bristol)

11.15 Findings from analysis of oxygen safety incidents related to type 2 acute respiratory failure
Dr Hamish Duff (Bristol)

Friday 27 November 2026

11.45 National Confidential Enquiry into Patient Outcomes and Death (NCEPOD) report on pleural procedures
Dr Mark Juniper (Swindon)

Learning objectives:

1) To learn more about the recently created assessment pathways for patients arriving to hospital with exacerbations of COPD or Asthma.

2) To gain insight on oxygen safety incidents related to type 2 acute respiratory failure.

3) To examine the NCEPOD report on pleural procedures.

10.45am - 12.20pm
St James, 4th Floor
SPOKEN SESSION: S127 - S132

Eosinophilic exacerbations: what do we do?

Chaired by: Professor Jadwiga Wedzicha and Dr Hamish Mcauley

10.50am **S127**
Not all eosinophil-associated exacerbations are the same : Inflammatory phenotypes of untreated COPD exacerbations
H.WW.A Aung, K.N Ntotsis, W.R.M Monteiro, P.N Novotny, K.B Balasundaram, C.E.B Brightling, A.KA.W Wright, N.J.G Greening

11.05am **S128**
Repeated blood eosinophil counts predict exacerbation sub-types in the Characterisation of COPD Exacerbations study (COPE).
AB Beech, RD Danby, ZP Paterson, TJ James, TN Nagarajan, AM Mathioudakis, DS Singh

11.20am **S129**
The Burden and Clinical Characterisation of the Eosinophilic Phenotype in Patients with Recurrent Severe COPD Exacerbations
B Brown, T Melor, A Dwarakanath, S Boot

11.35am **S130**
Most hospitalised COPD exacerbations lack a definable viral or eosinophilic endotype: a real world cohort study
C Morgan, R Granell, C Hyams, JW Dodd

11.50am **S131**
Robustness of time-to-event benefits associated with benralizumab-containing treatment in eosinophilic asthma and COPD: sensitivity analyses from the ABRA trial
M Zheng, N Smallcombe, S Ramakrishnan, REK Russell, E Dogu, M Bafadhel

12.05pm **S132**
Validation of the BAt risk score in a multi-centre cohort of hospitalised COPD exacerbations
A Beersaerts, H W W Aung, N Smallcombe, E Dogu, K Vermeersch, P Moghames, H J C McAuley, C A Flynn, C E Brightling, S Everaerts, S Cass, R E K Russell, W Janssens, N J Greening, M Bafadhel

10.45am - 12.20pm
Westminster, 4th Floor
SPOKEN SESSION: S133 - S138

ILDS: ILAS and familial fibrosis

Chaired by: Dr Anjali Cranshaw and Dr Laurence Pearman

10.50am **S133**
Risk Stratification and Longitudinal Assessment of Interstitial Lung Abnormalities Using e-Lung Quantitative CT Biomarkers
J Tadjkarimi, D Torres, AC de Freitas Cravo, A Devaraj, PM George, RJ Hewitt

11.05am **S134**
Mediastinal Lymphadenopathy predicts radiological progression of interstitial lung abnormalities (ILA) in Lung Cancer Screening Programme
N Khine, S Marguerie, J Naftel, M Morgan, V Gnanananthan, M G Jones, J Dunbar, T Wallis

11.20am **S135**
Characterisation of Interstitial Lung Abnormalities in a UK Multicentre ILA Clinic Cohort: Early Findings from the ILARC-UK Pilot
RJ Hewitt, D Torres, J Tadjkarimi, H Morris, K Zakis, J Swale, Z Ahmed, C Ruggiero, N Bracey, A Devaraj, PM George, MA Gibbons, P Rivera-Ortega, SC Stanel, L Fabbri, E Fraser, B Selvarajah, C Hayton

11.35am **S136**
Clinical Characteristics Associated with Progression of Interstitial Lung Abnormalities to Interstitial Lung Disease: A Single-Centre Cohort Study
D Torres, J Tadjkarimi, E Renzoni, A Wells, PM George, S Desai, A Devaraj, RJ Hewitt

11.50am **S137**
An Overview of the Clinical Characteristics of Familial Pulmonary Fibrosis Patients in the United Kingdom: Data from the British Thoracic Society ILD Registry 2013-2026
A Carolan*, J Callum*, C S Crook*, M Loughenbury, V Elworthy, S C Stanel, H Hanson, M Bradford, A Duckworth, L Fabbri, A Achaiah, G Bloye, F Chua, G Dixon, S Fletcher, R Hewitt, F Khan, H Morris, M Naqvi, B Price, G Thomas, S Toor, N Chaudhuri, C J Scotton, M A Gibbons, P Rivera-Ortega

12.05pm **S138**
Telomere Length Analysis and Next Generation Sequencing Results in Patients with Fibrotic Interstitial Lung Disease
A Carolan, J Jasmin, W L Ng, C Kannengiesser, R Borie, R Morgan, K Norris, D Baird, M Ozaki, K Hurley

10.45am - 12.20pm
Moore, 4th Floor
SPOKEN SESSION: S139 - S144

Pulmonary vasculature: aims and imaging

Chaired by: Dr Michael Newnham and Dr Sarah Scrivener

10.50am **S139**
Genetic associations of rare disease complications - brain abscess and migraine in patients with pulmonary arteriovenous malformations
A L Newman-Jones, S Soeiro, K Al-Dahleh, B Glampson, E Mayer, A Alsafi, C L Shovlin

11.05am **S140**
Ischaemic stroke in patients with pulmonary arteriovenous malformations: new associations with fractional exhaled nitric oxide and atrial fibrillation.
H Janbon, R Sheth, H McKernan, J Springett, H C Tighe, H Prabhudev, N Read, B M Milanes, J A Cabantug, J Ranger, M Al-Sahaf, N Coote, A Alsafi, B Glampson, E Mayer, C L Shovlin

11.20am **S141**
Targeting 5-HT to prevent ischaemic stroke due to untreatable pulmonary arteriovenous malformations without risking haemorrhage from conventional anti-platelet agents
CL Shovlin, M Iyer, K Al-Dahleh, A Modarresi, SSC Soeiro, SM Jarvis, K Rekopoulou, HJ Whitwell, M Storch, N Matthews, H Prabhudev, M Al-Sahaf, NM Coote, A Alsafi, B Glampson, E Mayer

11.35am **S142**
Rethinking Hypoxaemia in Pulmonary Arteriovenous Malformations: Associations with Pulmonary Artery Pressure and Gas Transfer
R Sheth, R Sheth, H Janbon, H Janbon, J Springett, H McKernan, H C Tighe, H Prabhudev, N Read, B M Milanes, M Al-Sahaf, N Coote, J A Cabantug, J Ranger, B Glampson, E Mayer, E Mayer, A Alsafi, C L Shovlin, C L Shovlin

11.50am **S143**
Using hyperpolarised 129Xenon and proton MRI to detect impaired gas exchange in structurally normal lung regions in systemic sclerosis patients with pulmonary arterial hypertension
M Caraher, LC Saunders, S Strickland, G Collier, N Stewart, S Kulkarni, F Hitchcock, AJ Swift, AMK Rothman, RA Condliffe, DJ Kiely, JM Wild, AAR Thompson

12.05pm **S144**
Anthropometry, sex and age at diagnosis influence AI-derived pulmonary blood volumes from CT pulmonary angiography in pulmonary hypertension
Hakim Ghani, Muhunthan Thillai, Simon Walsh, Elliott Bussell, Joanna Pepke-Zaba

10.45am 12.20pm
Abbey, 4th Floor
SPOKEN SESSION: S145 S150

From defence to damage: the phagocyte

Chaired by: Dr Louise Crawley and Professor Louise Donnelly

10.50am **S145**
Pleural fluid multi-omics reveals heterogeneous neutrophil populations in pleural infection
Yinzhou Feng, Kiki Cano-Gamez, Julia Chu, Alguili Elsheikh, Elie Antoun, Yaqi Li, Luyang Chen, Yichun Zhu, Lei Gao, Andrew Brown, Beenish Iqbal, Nikita Manoharan, Tao Dong, Yuanlin Song, James Chalmers, Najib Rahman, Julian Knight, Nikolaos Kanellakis

11.05am **S146**
Acute systemic hypoxia increases circulating neutrophil activation and extravasation potential
S Oruganti, S Venkataraman, S Nakanishi, T Peel, I Stewart, AS Cowburn, KM Lodge

11.20am **S147**
Distinct Pathological Neutrophils in Idiopathic Pulmonary Fibrosis Identify Disease Endotypes
KM Lodge, S Nakanishi, L Yazbeck, GK Ramadas-Dhondalay, J Guck, PL Molyneaux, AS Cowburn

11.35am **S148**
Effects of hypercapnia lead to differential phenotypes in COPD
P De Souza, S Cass, N Hart, RF DCruz, J Baker, M Bafadhel

11.50am **S149**
Human inhaled lipopolysaccharide challenge induces coordinated pulmonary and systemic inflammasome activation

TJ Mawhinney, DA Simpson, M Glenn, H Guo-Parke, M McKelvey, r McMullan, CC taggart, DF McAuley, RC Coll, DA Linden

12.05pm **S150**

Impaired ALOX15/5/FPR2 axis in COPD macrophages

JSY Mah, J Devulder, A Lobosco, P Fenwick, Y Guo, S Bartlett-Pestell, R Lopez, D Gaboriau, L Donnelly, RJ Snelgrove, JA Wedzicha, LJ Finney

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12.00pm – 2.00pm
Pickwick, 1st floor and Whittle & Fleming, 3rd floor

LUNCH (Lunch is not included in the delegate fee. Card payments only)

Exhibition closes at 2.00pm

12.45pm – 1.30pm
Churchill, ground floor
BTS GRAND CHALLENGE LECTURE

WHY THIS BILL MATTERS: TOBACCO, VAPING AND THE FUTURE OF LUNG HEALTH

Professor Sanjay Agrawal (Leicester)

Introduced by: Professor Elizabeth Sapey (Birmingham)

1.30pm - 3.30pm
St James, 4th Floor
POSTER DISCUSSION: P181 - P195

Innovation across the lung cancer diagnostic pathway

Chaired by: Dr Kimuli Ryanna and Dr Alastair Moore

P181 Clinical Impact of Thoracic Radiologist Input in a Lung Nodule Virtual Clinic: Improving Triage and Patient Outcomes
Ei Ei Khine Phyu, Qandeel Khan, Khola Nazim, Muhammad Majid, Prajakta Pinglay, Ziad Khan, Yiewfah Fong, Jonathan Bennett

P182 Anterior mediastinal masses identified through lung cancer screening: how should we measure, how should we manage and when to refer?
R Haggie, A Morjaria, S Gunning, A Edey, A Bibby

P183 Emergency Admissions with Lung Cancer – Who and Why?
S Raza, K Verbeke, F Kon, J Ong, A Talwar, CL Tey, G Ghidoni, A Moore, J E Park

P184 Discussing the Effectiveness of Geographic Mapping on Geospatial Analysis of Lung Cancer in Berkshire
S. H. I. Sung, I. Zaki, E. McKeown

P185 Self Request Chest X Ray Pathway: A Low Cost Strategy to Accelerate Lung Cancer Detection in Symptomatic Patients
Ei Ei Khine Phyu, Richard Westley, Syed Rizvi, Andrew Creeden, Marta da Silva, Helen Lang, Daniel Togher, Rajini Sudhir, Indrajeet Das, Ben Noble

P186 From Late Diagnosis and Palliation to Early Detection and Cure: The Sustained Impact of a Lung Cancer Screening Programme
R Biswas, M Lee, N Davies, A Nasimudeen, M Kyi

P187 Interval and incident lung cancers in a lung cancer screening program
A Iyer, O Mole, C Daneshvar

P188 Are early cancer detection and cure missed from the lung cancer screening programme?
M Lee, R Biswas, N Davies, A Nasimudeen, M Kyi

P189 Enhancing early lung cancer diagnosis - Efficacy of a strategy to increase primary care referrals
T Heather, P Paing, J Tuggey, S Roberts

P190 When It's Not Lung Cancer: Alternative Diagnoses and Diagnostic Delays
EM McGuigan, HH Hubbard, CM MacAulay, TP Plant

P191 Streamlining lung multi-disciplinary team meetings using AI based clinical decision support system: retrospective data analysis to validate standard of care
WM Ricketts, S Mathew, M Beauchamp, A Chambers, K Dodgson, V Patkar

P192 Lung cancer with cystic air spaces (LCCA) – a comprehensive review of the literature.
L Kimani, G Hyde, E O'Dowd, C Johns, G Fitzmaurice, M Majid, A Kar, K Jayaprakash, A Sharman, M Evison, A Bibby

P193 The value of circulating tumour DNA in secondary care thoracic oncology: search and rescue
K S Jethwa, J O Edwards, O N Mann, U Gupta

P194 The Impact of concordance between Liquid and tissue biopsy for the molecular profiling of actionable mutations in Non-small cell lung cancers (NSCLC)
Danne Shaji Philip, Aishwarya Krishnan, Maria Hill, Sanjay Kumar

P195 Outcomes of Imaging Surveillance in Virtual Lung Cancer Triage: Safety, Discharge Rates, and Cancer Detection within the National Optimal Lung Cancer Pathway
Ei Ei Khine Phyu, Eltayeb Ahmed, Qandeel Khan, Laraib Arshad, Ziad Alhaj, Asfandyar Yousuf, Veeresh Patil, Sanjay Agrawal, Jonathan Bennett, Syed Mohammad, Rajini Sudhir

1.30pm - 3.30pm
Westminster, 4th Floor
POSTER DISCUSSION: P196 - P211

Ventilation: frailty and comorbidity in patients with respiratory failure

Chaired by: Dr Michelle Ramsay and Dr Milind Sovani

P196 Combination of Non-Invasive Ventilation Outcome (NIVO) and Rockwood Clinical Frailty Scale (CFS) Scores in Predicting In-Hospital Mortality in COPD Exacerbations Requiring Non-Invasive Ventilation
A Shrestha, A Mahdi, R Varghese, G Wabara, I Nadeem, MS Anwar, S Kuckreja, U Ekeowa, PC Russell

P197 Frailty independently predicts mortality in patients receiving acute non-invasive ventilation for type 2 respiratory failure: a multicentre cohort study
ZZ Zubair, AB Babu, AC Collins, MHR Rahaman, ID Nadeem

P198 Prevalence and Influence of Oxygen toxicity in COPD exacerbations requiring non-invasive ventilation.
MH Naseer, K Brewin, N Lane, A Scadding, D Ghosh, B Prudon, M Sovani, T Hartley, J Gray, E Pakpahan, SC Bourke

P199 Evaluating the Impact of a Post-Exacerbation Respiratory Failure Pathway on Home Mechanical Ventilation Referrals and Clinical Outcomes: Mind the Gap
K Earnshaw, S Spikings, M Atkinson, A Gondker, S Boot, A Dwarakanath

P200 Changes in respiratory parameters obtained from remote NIV monitoring are associated with exacerbations in patients with COPD
Lay Min GAN, Andrew BENTLEY, Andras BIKOV

P201 Agreement and Accuracy of Ventilator-Derived VCO_2 for Estimating Resting Energy Expenditure in Mechanically Ventilated Critically Ill Adults: A Systematic Review and Meta-analysis
Jan Henrick Foscablo

P202 Morbid Obesity and Acute NIV: Preserved Acute Outcomes and Improved One-year Survival
A Ozsancak Ugurlu, G Pereira, B Tanveer, M Magashi-Ali, J Malla, A Halder, R Mukherjee

P203 Evolution of Acute NIV Practice Following the COVID-19 Pandemic: Changes in Case-Mix, Care Pathways and Outcomes
A Ozsancak Ugurlu, G Pereira, B Tanveer, M MagashiAli, J Malla, D Mukherjee, A Halder, R Mukherjee

P204 Outcomes of high flow nasal oxygen therapy in a respiratory support unit outside critical care
Aamir Ali, Mohammed Alhamadi, Rabeea Asghar, Donna Ward, Aileen Molesworth, Shyam Madathil, Syed Huq

P205 Clinical Outcomes of Non-Invasive Ventilation in Patients with Post-Tuberculosis Lung Disease Presenting with Acute Hypercapnic Respiratory Failure
Kewal Sethi, Arti Julka, J.C. Agrawat, Anil Badal

P206 Comprehensive Clinical Profile and Respiratory Outcomes of Patients with Limb-Girdle Muscular Dystrophy in a Specialised Long-Term Ventilation Service
L M H Bandara, M Roberts, JB Lilleker, A Bentley, A Bikov

P207 Respiratory support of facioscapulohumeral muscular dystrophy
A Clark, H Bandara, M Roberts, J Lilleker, A Bentley, A Bikov

P208 A single centre experience of non-invasive ventilation (NIV) and High flow therapy (HFT) for patients with motor neuron disease (MND) undergoing a percutaneous endoscopic

gastrostomy (PEG) insertion with propofol sedation.
S Bokhari, N Shah, A Reddy, G Kaltsakas, N Desmond, K Whitby, K Al-Arafi, S Srivastava, R D'Cruz, J Simpson, S Anderson, L Chong, H Thomas, L Arrandale, J McCanny, J Steier, N Hart, E-S Suh, P Murphy, MC Ramsay

P209 Analysis of outcome of Percutaneous Endoscopic Gastrostomy (PEG) tube insertion under non-invasive ventilation (NIV) support for Motor Neuron Disease (MND) patients: A three-year retrospective study
A Sivaramakrishnan, R Haider, A Khan, D Strachan, O Wilde, H Elsey, A Mukunda, S Hanna, S Irshad, A Rajhan, A Ali, A Thomas, N Mustfa

P210 Oximetry plus pulse wave tonometry for detecting respiratory failure in patients with amyotrophic lateral sclerosis (ALS); interim report
L Redmond, J Alcada, M.I Polkey

P211 Longitudinal clinical and therapeutic trajectories in those with motor neurone disease receiving tracheostomy ventilation at a specialist community long-term ventilation unit: a retrospective qualitative study
E Winterson, M Bartlett, L Vardy

1.30pm - 3.15pm
Moore, 4th Floor
POSTER DISCUSSION: P212 - P221

Opportunities to improve asthma care

Chaired by: Dr Alexandra Nanzer-Kelly and Dr Wadah Ibrahim

P212 Evaluation of screening tools to identify inappropriate SABA use in patients with asthma: The CHECK-SABA study.
JM González-Moro Rodríguez, S Alonso Viteri, C Amaral De Sousa, C Martínez Rivera, I Boira Enrique, M De la Poza Abad

P213 Using FeNO to predict the risk of asthma exacerbations: Impact of 'other' factors
Hafsa Ahmed, Lowri Hunt, Moez Nayyar

P214 Developing Interventions to Tackle Ethnicity-based Disparities in Asthma Management in British South Asians "TEAMS STUDY"
N Pervez, AH Mansur, I Williams, R Knibb, MT Krishna

P215 An Evaluation of Inhaler Optimisation in Asthma Patients During Hospital Attendance: Clinical Effectiveness, Sustainability and Patient Involvement
H Hussain, L Elsey

P216 High ICS/Formoterol Inhaler Utilisation and Potential Medicines Oversupply: A National Prescribing Analysis 2022–2025
AC Murphy, S Khatri, R Seeley, TG Capstick

P217 Airway variability across methacholine challenge and bronchodilator recovery improves sensitivity for diagnosing asthma
AJ Simpson, L Healy, R Wang, M Bennett, S Drake, H Wardman, SJ Fowler, C Murray, A Simpson

P218 Barriers and facilitators to implementing anti-inflammatory reliever-based asthma care in community pharmacy: a qualitative study
H Harvey, J Dyson, D Sykes, K Shah, G Godfrey, M Watt, G Chiu, J Lipworth, T Morris, M Bhalla, L Hubanova, M G Crooks

P219 Can home spirometry and FeNO testing improve asthma diagnosis? – a feasibility study
R Wang, A Thompson, M Bennett, A Simpson, SJ Fowler, HJ Durrington, CS Murray

P220 Depemokimab demonstrates sustained long-term efficacy and consistent patient-reported outcomes over 2 years in patients with type 2 asthma irrespective of baseline inhaled corticosteroid dose: An integrated analysis of the phase III SWIFT-1/-2 and the open-label extension AGILE studies
RA Panettieri, M Bonini, CQ Zhu, D Jarreta, C Tacon, P Howarth, DJ Jackson

P221 Phenotyping Severe Asthma Attacks in Hospitalised Patients
M J Martin, M Aljehani, I Sayers, T McKeever, T W Harrison

1.30pm - 2.35pm
Abbey, 4th Floor
POSTER DISCUSSION: P222 - P229

Advances in thoracic surgery and tobacco harm reduction

Chaired by: Dr Nick Weatherley and Dr Leanne Ashrafian

P222 Echocardiographic Probability of PulmonaryHypertension and the Association With 90-Day Mortality and Post-Operative Complications in People Undergoing Lung Cancer Resection Surgery

NM Shakeel, V Kumar, S Sothynathan, R Maulana, H Ahmad, A Dawson, R Sudhir

P223 Long-term bronchoscopic management of localised tracheobronchial amyloidosis
A Askari, R Cuttle, R Shea, M Mullin, R Dunwoody, N Navani, R Thakrar

P224 Investigating regional variation in referral for lung volume reduction: who sends us what?
TS FitzMaurice, L Anderson, S Woolley, A Reghunath, M Shackcloth, K Mohan

P225 Real-world outcomes of a multidisciplinary hyperinflation service for severe emphysema
H G Kamil, Q Gohir, B Al-Harbawi, S Ghosh, M Haris

P226 Smoking Cessation in COPD: A Mixed-Methods Analysis of Behavioural, Socioeconomic and Healthcare Determinants in Real-World Practice
A Beale, C Yerramasu, T Walsh, S Waite, R Preece, J Farquhar, G Choudhury

P227 The prescription of Home Oxygen to people who vape: a cross-sectional survey study
J Webb, N Tunstall, F Fyles, K Lippiett

P228 Nicotine Dependence Among Respiratory Therapy Students in Saudi Arabia: Findings From a National Cross-Sectional Survey
ASA Alsulayyim, RAA Alyami, ASA Al Sharifi, AH Hakamy, AMA Alasmari, ZA Alshehri, FHA Alahmadi, SMA Alghamdi, MAA Almeshari, ASA Alghamdi, MMA Alyami, JSA Alqahtani, NYA Alobaidi, BSA Alkhathlan, KAA Alqahtani, HHA Alowiwi, FHA Algharbi, AAA Alqarni, AMA Aldhahir

P229 The Impact of Varenicline in Combination with Nicotine Replacement Therapy and Specialist Support on Morbidity and Healthcare Utilisation for Inpatients with Tobacco Dependency.
S Patel, E Fraser, E Doran, L Restrick

1.30pm - 3.15pm
Rutherford, 4th Floor
POSTER DISCUSSION: P230 - P244

Improving every breath: pulmonary rehabilitation advances

Chaired by: Dr Adam Lewis and Professor Charlotte Bolton

P230 Effectiveness of pulmonary rehabilitation across individual characteristics: findings from the National Respiratory Audit Programme
H Drover, H Drover, MW Orme, L Wang, M Richardson, JK Quint, RA Evans, SJ Singh, E Daynes

P231 Designing the UPTURN intervention to support uptake of pulmonary rehabilitation: a collaborative co-design process
A.P. Moore, J. Ward, T. Kipouros, S. Langston, L. Lawrence, M.M.G. Rabbani, A. Achininaya, T. Tabassum, S. Prinjha, F. Lorencatto

P232 Post exacerbation Pulmonary Rehabilitation: has the population changed?
E Chaplin, S Ward, E Daynes, S Harvey, E Vincent, N Gardiner, SJ Singh

P233 Systematic review of interventions to improve referral and uptake of pulmonary rehabilitation in patients with COPD
RABN Idham, E Marcus, J S Watson, W D-C Man, M Maddocks

P234 Experience based co-design to adapt pulmonary rehabilitation for South Asian individuals
H Drover, J McAllister, L Gardiner, MW Orme, SJ Singh, E Daynes

P235 Co-design of a very brief behavioural change approach to assist referrers discuss pulmonary rehabilitation with COPD patients
R Tuffnell, C Kilbride, E Norris, M Steiner, B Newman, Nithi Sivasubramaniam, S Papadakis, A McEwen, E Bevan-Smith, W D C Man, S J C Taylor, E A Steed, C M Nolan

P236 "Designing rehabilitation for individuals with Long Covid – The 4Ps of Acceptability"
K Kontou, G Doe, L Houchen-Wolloff, N Bateman, J Creese

P237 A behavioural intervention for patients with long-COVID
SJ Preece, A Handley, L Gifford, T De Souza, W Maltinsky

P238 The benefits of a novel group-based chest management education pathway for chronic respiratory disease: improving patient outcomes and reducing clinician time.
Inge Vermeulen, Urvashi Soneji

P239 Mucus plugging, eosinophilia, chronic lung disease and mucolytic therapy: Identifying a phenotype
JJ Johnson, NA Ahmad

P240 Long-Term Recovery Following Physical Rehabilitation for Post-COVID Syndrome: A Qualitative Exploration of Persistent Symptoms and Impact of Exercise
SC Chakraverty, GAB Baker, EH Hume, DM Megaritis, ED Daynes, RAE Evans, SS Singh, CE Echevarria, CH Haighton, IV Vogiatzis

P241 Can observable breathing abnormalities identify breathing pattern disorder? Evaluation of the Breathing Pattern Assessment Tool
LJF Grillo, H Shannon, A-M Russell, NS Hopkinson, JH Hull, CI Bloom

P242 Beyond the score: Physiotherapists' experiences of using the Breathing Pattern Assessment Tool in clinical practice
LJF Grillo, H Shannon, A-M Russell, NS Hopkinson, CI Bloom

P243 The PneumoRater chest-worn sensor can continually measure respiratory rate and signs of air hunger in adults with and without breathing pattern disorders.
J Hudson-Colby, F Yates, S Brider, M Harding, H Baston, M Shaban, N White, A Lewis

P244 An online breathing and wellbeing programme (ENO Breathe) for people with long COVID breathlessness: a mixed methods analysis of 3-month post-intervention follow-up data
SMM Macleod, PJW Williams, HO Owles, CL Lee, SM McVey, TP Pagnuco, KB Bruce, BW Warnock, AC Chomacki, HB Brunjes, JM Mollica, AL Lound, SZ Zumpe, AMA Abrahams, VP Padmanaban, THH Hardy, AL Lewis, AL Lalvani, SLE Elkin, NSH Hopkinson, KEJP Philip

1.45pm – 3.15pm
Churchill, Ground floor
SYMPOSIUM

OPTIMISING SLEEP: FUTURE DIRECTIONS FOR TREATING SLEEP DISORDERED BREATHING

Chaired by: Dr Sonya Craig (Liverpool) and Helen May (Manchester)

13.45 Gender Differences in Obstructive Sleep Apnoea
Dr Renata Riha (Scotland)

14.15 MAD plus CPAP: does combining two established treatments for Obstructive Sleep Apnoea give added benefits?
Dr Tim Quinnell (Cambridge)

14.45 Complex Sleep Apnoea and ASV: Evidence, Controversies, and Patient Selection
Professor Esther Schwarz (Zurich)

Learning objectives:

1) To discuss the reasons for delayed diagnoses in women and understand the differences in OSA between men and women and the impact of treatment

2) To understand the treatment efficacy of CPAP and MADs in comparison to each other and in combination

3) To understand the indications for ASV and the current evidence in the context of the ADVENT-HF and SERVE-HF trials, with some case examples

1.45pm – 3.15pm
Mountbatten, 6th floor
SYMPOSIUM

IS THERE A ROLE FOR STEROIDS IN CAP?

Chaired by: Dr Frances Grudzinska (Nottingham) and Dr Meg Coleman (London)

13.45 Host immune responses and corticosteroid effects in severe CAP
Professor Thomas Wilkinson (Southampton)

14.15 Effect of steroids on mortality in patients with severe community-acquired pneumonia: The REMAP-CAP Corticosteroid Domain Randomized Clinical Trial in context
Professor Anthony Gordon (London)

14.45 NICE guidelines in context – steroids in pneumonia
Dr Tom Bewick (Derby)

Learning objectives:

1) Understand how digital technologies are used to monitor paediatric respiratory disease, sleep, and long-term ventilation

2) Evaluate the clinical value of remote monitoring for early detection and personalised management

3) Identify key challenges and enablers for implementing digital respiratory technologies in routine paediatric practice

1.45pm – 3.15pm
Windsor, 5th floor

SYMPOSIUM

CROSS-DISEASE APPROACHES TO TREATING THE ONSET, EXACERBATION AND PROGRESSION OF CHRONIC LUNG DISEASES

Chaired by: Professor David Lomas (London) and Dr Samantha Walker (ALUK)

13.45 New and known pathological pathways as novel targets during the early phases of chronic respiratory disease.
Dr Renata Jurkowska (Cardiff)

14.15 Preventing acute microbial-driven exacerbations from destabilising lung function and worsening chronic disease.
Dr Aurelie Crabbe (Belgium)

14.45 Opportunities for targeting common cross-disease mechanisms to prevent progression of chronic lung diseases.
Dr Amanda Tatler (Nottingham)

Learning objectives:

1) Explore common pathways and targets, with treatment potential across different stages of chronic respiratory disease, where focused research would accelerate drug discovery

2) Understand how increased efforts in developing specific research tools are needed to make significant progress towards the development of new treatments, including increased effort towards biomarker development across respiratory diseases

3) Provide insight into how taking a cross-disease and cross-discipline approach can facilitate collaborations that drive the development of tools (biomarkers) and treatments (drugs) that are truly disease-modifying, and what key elements can drive impactful collaborations

1.45pm – 2.45pm
Albert, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING

Cystic Fibrosis

Friday 27 November 2026

1.45pm – 2.45pm
Victoria, 2nd floor
BTS SPECIALIST ADVISORY GROUP OPEN MEETING

Interstitial and Rare Lung Disease

3.00pm – 3.30pm
Britten, 3rd floor

COFFEE/TEA BREAK

3.15pm - 5.00pm
Cambridge, 5th Floor
POSTER DISCUSSION: M32 - M47

The future of sleep and physiological services

Chaired by: Dr Sophie West

M32 A Multidisciplinary Secondary Triage Model for Obstructive Sleep Apnoea Referrals: Improving Efficiency in Sleep Services
A Peace-Witton, L Earnshaw, A Dwarakanath

M33 Home sleep study quality and clinical yield across diagnostic modalities in an NHS sleep service: a service evaluation
A Bonham-Carter, C Francis, A Manuel

M34 Effect of a Telemonitoring-Enabled Multidisciplinary Clinic on CPAP Adherence in Obstructive Sleep Apnoea
E Green, S Wallbanks, A Ugurlu, D Mukherjee, M Afzal, R Mukherjee

M35 Does Where You Live Affect Early CPAP Use? Three-Month Adherence Across Rural Herefordshire
A Manuel, S Coelho, C Francis, R Leach, H Cullen, N Pious

M36 Sensitivity of Overnight Oximetry between Ethnic Groups
U R Ehioghae, K Lee, A S Patel, R Madula, M Pittman

M37 Does hypoxic burden better explain sex differences in functional impairments than apnoea-hypopnoea index in obstructive sleep apnoea?
U Nanda, YS Bin, P de Chazal, ASL Chan, A Piper, K Kairaitis, PA Cistulli, BJ Yee

M38 Residual Apnoea Hypopnea Index Measurement from PAP Devices Versus Home

Polygraphy: A Method Comparison Study Using Real World Data
S K Mansell, S Augustt, F Gowing, D Ridout, S T Hilton, C Kilbride, E Main, S Schievano, S Mandal

M39 Follow-up After CPAP Therapy Initiation in Pediatric Obstructive Sleep Apnea Syndrome (OSAS) – Insights from a Retrospective Analysis
CB Bernold, IR Rubira, AB Bush, HLT Tan

M40 Optimising Transcutaneous Electrical Stimulation Strategies for Obstructive Sleep Apnoea: Comparing Continuous and Intermittent Stimulation Using a Skeletal Muscle Model
Mohammed Alkhathami, Ahmed Almaghrabi, Asim Madkhali, Georgios Kaltsakas, Gerrard F Rafferty, Joerg Steier

M41 Obesity Management in Obstructive Sleep Apnoea: Patient Perspectives and a Quality Improvement Study
G Haffenden, J Cartledge, C Huon, P Marino

M42 Obstructive Sleep Apnoea and Difficult Asthma: Provisional Findings from the OSADA Trial
L Piggott, L Monaghan, C Buckley, E Mulligan, A Franciosi, B Kennedy, Rory ODonnell, D Grimes, P Mitchell, D Fitzgerald, B Kent

M43 Delivering the NHS Long Term Plan: Outcomes from a digitally enabled community breathlessness service
K P Sylvester, K Pauley, E ONeill, M Knolle

M44 Respiratory functions in adult with perinatal HIV infection
Y Myat, G Zouridaki, C Foster, O M Kon

M45 Impulse oscillometry in extrathoracic restriction friend or foe?
WM Ricketts, S Joseph, GV Kopernicka, PS Marino

M46 Exercise limitation in COPD: insights from cardiopulmonary exercise testing
M Afzal, S Wallbanks, R Gregory, A Ozsancak-Ugurlu, A Watson, R Mukherjee

M47 Mind the gap: Comparison between GLI race neutral lung function with ECCS in different ethnicities
CJ Froneman, S Lovage, S Goyal, P Bhowmick, K Sanyal, R Sarka**r**

3.30pm – 5.00pm
Churchill, Ground floor
SYMPOSIUM

LUNG CANCER SURVIVORSHIP: RECURRENCE RISK, SECOND PRIMARIES & CHEMOPREVENTION

Chaired by: Dr Liz Fuller (Newcastle) and Dr Alexandra Teagle (Plymouth)

15.30 Expanding roles of liquid biopsies for lung cancer: detection, residual disease, surveillance and beyond
Professor Nitzan Rosenfeld (London)

16.00 Second primary lung cancers – who is at risk and how long should we follow people up?
Professor Robert Rintoul (Cambridge)

16.30 LungVax: early interception and prevention in the lung cancer setting
Professor Mariam Jamal-Hanjani (London)

Learning objectives:

1) To recognise the risk of second primary lung cancers in radically treated patients, appreciate potential factors associated with increased risk and be aware of future tools for risk assessment and early detection

2) To understand the concept of minimal residual disease in lung cancer, its relationship with recurrence and biomarkers to aid surveillance and early diagnosis

3) To describe current and emerging chemoprevention strategies with a focus on vaccine therapy to prevent or delay the development of second primary lung cancers and tumour recurrence after radical treatment

3.30pm – 5.00pm
Mountbatten, 6th floor
OPEN SESSION

NRAP: DRIVING IMPROVEMENTS IN RESPIRATORY OUTCOMES FROM INNOVATIONS IN RESPIRATORY CARE

Chaired by: *Professor Alice Turner (Birmingham) and Dr Hannah Burke (Southampton)*

15.30 Patient centred innovation – overcoming health inequalities
Professor Ian Sinha (Liverpool)

15.50 Respiratory innovation and improvement in primary care
Dr Katherine Hickman (Leeds)

16.10 Driving impact from new therapies
Dr Irem Patel (London)

16.30 Understanding new models of care
Professor Alice Turner (Birmingham)

Learning Objectives:

1) To involve the respiratory community in the national drive to improve outcomes through understanding innovations in respiratory care. Focus will be given to a review of respiratory care delivered across the whole respiratory pathway and the implementation science to deliver change including the equitable roll out of biologics.

2) To understand the emerging data on the intersection of health inequality and respiratory outcomes and how service models need to evolve to address these.

3) To explore innovations in improving quality of care in novel settings including virtual wards and emergency departments.

3.30pm - 5.05pm
Windsor, 5th Floor
SPOKEN SESSION: S151 - S156

The fibrotic niche: epithelial fibroblast interactions

Chaired by: Dr Alison John and Professor Chris Scotton

3.35pm **S151**
Epithelial Cell Detachment as an Origin Model that Promotes Basal to Aberrant Basaloid Trans-differentiation
Konain F. Bhatti, Gisli Jenkins, Alison E. John, Iain D. Stewart

3.50pm **S152**
Lung epithelial cell-derived leucine-rich alpha2 glycoprotein-1 (LRG1) drives the development of Tuberculosis-induced pulmonary fibrosis
DLW Chong, J Kutschenreuter, JS Friedland

4.05pm **S153**
Targeting AKAP13 RhoGEF activity ameliorates pro-fibrotic phenotypes driven by the IPF associated AKAP13 risk variant
B Liu, J May, G Contento, S Gangi, L Organ, E Pyman, I Kibreab, L Zhao, HY Yao, ID Stewart, AK Reed, AE John, RG Jenkins

Friday 27 November 2026

4.20pm **S154**
Gαq/11 modulates the extracellular matrix and fibroblast-epithelial interactions to control alveolar repair
AT Goodwin, J Patel, S Henson, C Sainz Zuniga, N Hannan, AL Tatler

4.35pm **S155**
Hemicentin-1 Mediates Profibrotic Signalling In Lung Fibroblasts In Idiopathic Pulmonary Fibrosis
H Yao, B Liu, J W P May, K Bhatti, RG Jenkins, ID Stewart, AE John

4.50pm **S156**
Factor-inhibiting HIF (FIH) regulates fibroblast phenotype and extracellular matrix remodelling in the ageing human lung
L N Sayer, F Conforti, R A Ridley, J A Bell, S Wang, J G H Parkin, R Allen, J Ceccato, F Cinetto, A Alzetani, D E Davies, Y Wang, M G Jones

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3.30pm - 5.00pm
Moore, 4th Floor
POSTER DISCUSSION: P273 - P284

Advances in translational research

Chaired by: Professor Hannah Durrington and Professor Robert Gray

P273 Comparing app-entry and calendar-day prediction of COPD exacerbations using real-world myCOPD data
HMG Glyde, AM Blythin, TMA Wilkinson, IT Nabney, JW Dodd

P274 Combining pleural fluid proteomics and microbiology data to identify pleural infection biological endotypes
Yinzhou Feng, Elie Antoun, Julia Chu, Justin Whalley, Kiki Cano-Gamez, Alguili Elsheikh, Yaqi Li, Luyang Chen, Yichun Zhu, Lei Gao, Tao Dong, Yuanlin Song, James Chalmers, Najib Rahman, Julian Knight, Nikolaos Kanellakis

P275 Prior exacerbation history shapes short-term prediction of COPD exacerbations in real-world myCOPD data
HMG Glyde, AM Blythin, TMA Wilkinson, IT Nabney, JW Dodd

P276 Platelet function in chronic obstructive pulmonary disease (COPD) patients, with and without cardiovascular co-morbidities
N Hajmahmoud, SP Cass, SC Pitchford, M Bafadhel

P277 A multivariate sputum proteomic signature associated with airflow limitation in COPD: baseline findings from the phase 2a, randomised, double-blind, placebo-controlled COPD-ST2OP trial
KN Ntotsis, MS Siow, VM Mistry, KH Haldar, PN Novotny, AW Wright, DFC Choy, MAG Grimbaldeston, NJG Greening, CEB Brightling

P278 Baseline serum IL1RL1 identifies a COPD subgroup with increased exacerbation burden and reduced exacerbation rates following Astegolimab: a post-hoc analysis of the phase 2a randomised, double-blind, placebo-controlled COPD-ST2OP trial
MS Siow, KN Ntotsis, VM Mistry, KH Haldar, PN Novotny, AW Wright, DFC Choy, MAG Grimbaldeston, NJG Greening, CEB Brightling

P279 Mapping the Prodrome Phase of Rhinovirus-Induced Chronic Obstructive Pulmonary Disease (COPD) Exacerbation
D. P. Wilkins, C. Morgan, S. J. Park, M. B. Trujillo-Torralbo, P. Mallia, J. S. Mar, D. Dugger, H. Chen, D. S. Cheung, O. M. Kon, M. R. Edwards, M. A. Grimbaldeston, D. F. Choy, S. L. Johnston

P280 Eosinophil depletion unmasks distinct cytokine profiles in EGPA and severe eosinophilic asthma
P Khooshemehri, J Gates, J Dhariwal, A M Nanzer, A M Nanzer, D J Jackson

P281 Admission IL-33/ST2 profiling identifies compartment-specific severity signals in adults hospitalised with respiratory viral infections
T G Morelli, M Purcell, A Freeman, TMA Wilkinson

P282 Beyond the Eosinophil: Benralizumab Drives Airway-Specific T cell Immune Remodelling in Severe Eosinophilic Asthma
J Gates, P Khooshemehri, V Kanabar Raivadera, A del Rosario, A Perez Valles, E Woods, R Martinez Nunez, D J Jackson

P283 Host-derived clinical phenotypes delineate distinct severity trajectories in adult respiratory viral hospitalisation
TG Morelli, M Purcell, A Freeman, TMA Wilkinson

P284 Intrapleural enzymatic therapy modulates neutrophil-mediated inflammatory responses - The Iffley study
YYJ Chu, Alguili Elsheikh, Yinzhou Feng, Amy Gilmour, Nikita Manoharan, Luyang Chen, M.Alba Sorolla, Jose M Procel, Federico Mei, Federica Monaco, Gianluigi Giangregorio, Marco Tomasetti, Argyrios Tzouvelekis, Dimitris Komninos, Fotios Sampsonas, Sina Ahmadzai, Gitte Alstrup, Uffe Bodtger, Tao Dong, Yichun Zhu, Lei Gao, Yuanling Song, Julian C. Knight, Eihab O Bewadi, James D. Chalmers, Naijib M. Rahman, Nikolaos I. Kanellakis

3.45pm - 5.20pm
St James, 4th Floor
POSTER DISCUSSION: P245 - P259

Asthma in transition

Chaired by: Dr Laura-Jane Smith and Dr Andy Chadwick

P245 Does the carbon footprint of inhalers impact patient's inhaler choice. Evidence from the REDUCE Carbon for Patients RCT.
L Wiffen, I Kostakis, D Neville, TL Jones, KS Babu, AJ Chauhan, TP Brown

P246 Are the inhaler prescribing preferences of healthcare professionals influenced by the carbon footprint - evidence from the REDUCE Carbon RCT.
L Wiffen, I Kostakis, D Neville, TL Jones, KS Babu, AJ Chauhan, TP Brown

P247 Shifting Patterns of ICS/Formoterol Utilisation Following Implementation of AIR/MART Therapy: A National Prescribing Analysis 2022–2025
TG Capstick, S Khatri, R Seeley, AC Murphy

P248 Exploring prescribing patterns following switching between Fostair® and Luforbec® in a Scottish NHS board : a retrospective analysis of PIS prescribing data
LH Haddock, VS Sharma, GB Bryson, BM Melia, NW Weir, MP Patel, AS Smith, PP Paxton

P249 Budesonide/glycopyrronium/formoterol fumarate dihydrate effects on lung function and asthma exacerbations by persistent airflow

limitation status: post hoc analyses of KALOS and LOGOS studies
DJ Jackson, A Papi, RA Wise, NL Lugogo, R Chen, T Trasieva, J Marshall, K Bowen, H Pandya, A Megally, M Patel

P250 Inhaler Mismatch in Athletes: Inspiratory Flow and Technique Favour Dry Powder Inhalers Over Pressurised Metered-Dose Inhalers
TH Burrell, MG Crooks, BW Kolosz, AJ Simpson

P251 Effectiveness of a facilitated implementation strategy to improve routine delivery of supported asthma self-management in UK primary care: a cluster randomised implementation trial (IMP2ART)
HJ Pinnock, V Hammersley, F Appiagyei, A Barat, B Delaney, D Fitzsimmons, T Hamborg, S Holmes, T Jackson, M Jones, S Julious, C MacLeod, K McClatchey, V Marsh, N Morgan, D Price, J Sheringham, L Steed, B Stuart, SJC Taylor

P252 An Economic Evaluation of the IMP²ART Whole-Systems Intervention to Support Asthma Self-Management in Primary Care
M Jones, F Davies, D Fitzsimmons, TJ Driscoll, T Hamborg, V Hammersley, C Macleod, V Marsh, JW Parsons, J Sheringham, EA Steed, BL Stuart, SJC Taylor, H Pinnock

P253 Reducing the number of healthcare interactions to achieve a diagnosis in asthma using the Astec score vs BTS/SIGN/NICE guidelines
SA Farrell, C Ottewill, CM Gill, PJ Kerr, V Brennan, H Doherty, O Smith, E McHale, B Cushen, D Ryan, G Greene, RW Costello

P254 Physical Activity and Mortality in Asthma, COPD, and Obstructive Spirometry: An 8-Year Population-Based Cohort Study
TT Itkonen, TM Mattila, TJ Vasankari, K Tokola, MJ Lopmeri, W Mazur, J Lahti, TM Vasankari

P255 Identifying the residual unmet need in severe asthma
J Gates, J Kavanagh, F Haris, J Lam, M Fernandes, L Green, L Thomson, C Roxas, G d Ancona, J Dhariwal, A M Nanzer, D J Jackson

P256 Assessing the role of social determinants of health in asthma hospitalisations using the Clinical Practice Research Datalink.
I Adejumo, C E Bolton, D E Shaw, T M McKeever

Friday 27 November 2026

P257 Assessing Demographic Disparities in Severe Asthma: Access to Care, Lung Function, and Treatment Patterns at a Tertiary Respiratory Care Centre
S.B Biswas, A.V Vyas

P258 Socioeconomic Deprivation and Risk of ICU Admission in Asthma: A Retrospective Cohort Study
K Gaffney, B Sloan, A Gupta, C Thurlow, LM Jones

P259 Recruiting and retaining participants from deprived communities into an asthma diagnostic study
LS Whipday, M Bennett, L Willmore, L Healey, S Drake, A Simpson, S Fowler, C Murray, R Wang

3.45pm - 5.10pm
Westminster, 4th Floor
POSTER DISCUSSION: P260 - P272

Type 2 COPD: biomarkers, biologics and outcomes

Chaired by: Professor Stephen Bourke and Professor Wisia Wedzicha

P260 Blood eosinophil counts and incident asthma and COPD: prospective analyses in UK adults
B Mappin-Kasirer, N Petousi, RG Walters, HF Ashdown, KBH Lam, ID Pavord, A Edris

P261 Predictors of blood eosinophil testing in moderate-to-severe COPD: A UK retrospective cohort study
J Park, J Bolaji, S Yang, T Tritton, P Payne, D Main, W Kay, JK Quint

P262 Persistent exacerbation and mortality in COPD with type 2 inflammation despite triple therapy: A UK retrospective cohort study
J Park, S Yang, J Bolaji, T Tritton, P Payne, D Main, W kay, JK Quint

P263 What Does FeNO Add Beyond Blood Eosinophils? Joint Biomarker Phenotyping for Assessing Inhaled Corticosteroids Response in COPD
H.WW.Aung Aung, K.N Ntotsis, P.N Novotny, K.B Balasundaram, C.B Brightling, A.KA.W Wright, N.J.G Greening

P264 COPD Assessment Test Scores in Patients Meeting NICE/MHRA Dupilumab Eligibility Criteria: A Real World Analysis

AE Eitezaz, MH Hussain, MRG Ghani, WF Fazal, SFW Wood, KJ Jackson, GO Owen, AR Rajhan, AT Thomas

P265 Service Evaluation of Dupilumab Eligibility in COPD Patients with Acute Exacerbations Across Wirral
JW Goh, S Meikle, J Sola Garcia, J Seaborne, J Ayathamattam, O To, R Robinson, K Liatsikos

P266 Mepolizumab reduces systemic corticosteroid utilisation in chronic obstructive pulmonary disease with type 2 inflammation: Pooled results from phase III randomized trials
NA Hanania, M Kraft, E Burrows, A Biswas, S Gould, J Min

P267 Sustained FEV1 improvements with mepolizumab up to 104 weeks in patients with COPD and less severe airflow obstruction: Post hoc analysis of the MATINEE phase III study
D Singh, SP Bhatt, ID Pavord, E Penz, B Mayer, J Min, S Kolterer, CF Vogelmeier

P268 Dupilumab is efficacious in chronic obstructive pulmonary disease with type 2 inflammation regardless of smoking status
Jadwiga A. Wedzicha, Surya P Bhatt, Guy G. Brusselle, Helena Backman, Hisatoshi Sugiura, Henrik Watz, Paramita Saha Chaudhuri, Mena Soliman, Alberto Papi, Jigna Heble

P270 Treatment failure at 1 year following benralizumab during an eosinophilic exacerbation: data from ABRA
N Smallcombe, E Dogu, M Zheng, S Ramakrishnan, R E K Russell, M Bafadhel

P271 CRP modifies response to mepolizumab following hospitalisation for COPD exacerbation: post-hoc analysis of COPD-HELP
CA Flynn, HJC McAuley, O Elneima, AKA Wright, M Richardson, CE Brightling, NJ Greening

P272 Biologic response in severe asthma patients with overlapping asthma and COPD features: a real-world cohort study
J Bales, I Akbar, A Lawton, J Bales, A Gonzales, A Suson, A Villagracia, J Brown, S Siddiqui, E Wong, H Farne

SPEAKERS' BIOGRAPHICAL DETAILS

Biographical Details for Speakers and Chairs in Symposia, Guest Lectures and Journal Clubs

Dr Alex Adams is a Paediatric Respiratory Consultant at Leeds Children's hospital and is clinical lead for asthma and the severe asthma service for West Yorkshire. She did her MD(Res) in severe asthma looking at the role of IL-17 at the Royal Brompton Hospital and has been principal investigator for a number of national studies in asthma in Leeds. Alex also has specialist interests in congenital abnormalities affecting the airways and bronchiectasis.

Dr Dinesh Addala is a Respiratory Consultant and pleural specialist at Frimley Health NHS Foundation Trust, and a clinical researcher at the University of Oxford and Oxford Respiratory Trials Unit. His research focuses on improving diagnostic and treatment pathways for patients with malignant pleural disease, which formed the basis of his doctoral fellowship. He is PI of the NIHR-funded STREAMLINE trial, evaluating a streamlined approach to the diagnosis and definitive management of suspected malignant pleural effusion.

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.

Professor Stefano Aliberti is Full Professor of Respiratory Medicine, Vice-Rector for Social Responsibility, and Director of the Fellowship Program in Respiratory Diseases at Humanitas University. He is also Chief of the Respiratory Unit at IRCCS Humanitas Research Hospital (Milan, Italy), where he leads the Bronchiectasis and Non-Tuberculous Mycobacteria Programs. His major clinical and research interests are both acute and chronic respiratory infections. He has extensive experience in epidemiological and clinical research in pneumonia since early-2000s when he was working for the Community-Acquired Pneumonia Organization. He has extensively published clinical and translational research papers on community-acquired pneumonia, especially in the field of clinical failure, cardiovascular events and antimicrobial resistance. He received the Young Researcher Award in Respiratory Infections from the European Respiratory Society for his contribution on community-acquired pneumonia. Over the past 20 years he has been heavily involved in acute and chronic respiratory infections, including bronchiectasis and non-tuberculous mycobacteria. He co-chairs the European Bronchiectasis Registry (EMBARC) and chairs both the Italian Bronchiectasis Registry and the Italian Registry of Pulmonary Non-Tuberculous Mycobacteria. Prof. Aliberti published more than 470 peer-reviewed articles especially on respiratory infections over the past 20 years and he was part of different international task forces to produce guidelines on bronchiectasis, COVID-19 and severe pneumonia, as well as statements on tuberculosis. Prof. Aliberti has been heavily involved in different international societies over the past decade, including the European Respiratory Society, the American College of Chest Physicians, and the European Cystic Fibrosis Society, in different educational and scientific committees.

Dr Martin Allen is a consultant physician at the University Hospital of North Staffordshire where he has had a major interest in respiratory and sleep medicine. He has fulfilled a variety of management and transformational roles within the hospital and nationally. Currently these include: Chair of the expert working group on coding for respiratory medicine, National respiratory GIRFT lead, lead for the GIRFT review of TB services across England and Scotland and National Speciality Advisor for physiological science in the National Diagnostics Program, In the latter role he has been involved in the development and roll out of CDCs since their inception.

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 Jonathan Baker is a senior research associate at King's College London. His research interests focus on investigating aberrant signalling and inflammatory mechanisms in the respiratory disease, chronic obstructive pulmonary disease (COPD). Jonathan previously worked with Professors Peter Barnes, Louise Donnelly, and Kaz Ito at Imperial College London, examining the role cellular senescence in driving COPD pathogenesis and whether this can be targeted therapeutically using both senolytic and senomorphic therapy. He continues this work, examining the role cellular senescence plays in disease exacerbations, working in collaboration with Professor Mona Bafadhel at King's.

Dr Eihab Bedawi is a Consultant Respiratory Physician and Clinical Lead of the Oxford Pleural Unit at Oxford University Hospital. He completed his PhD in pleural disease at Oxford and conducts clinical and translational research in pleural infection and advanced ultrasound. He is Key Investigator of the MIST-4 trial and Co-Chair of the ERS supported EMPIRE pleural infection translation research network. He co-chairs European Respiratory Society taskforces on pleural infection and pleural tuberculosis and contributes to British Thoracic Society pleural guidelines, education and training standards.

Dr Tom Bewick is a Consultant in Respiratory Medicine at the Royal Derby Hospital. He has a clinical and research interest in acute lung infection, in particular developing and testing novel interventions to enhance recovery from community-acquired pneumonia and long COVID. He was a committee member for the NICE pneumonia guideline update in 2025, and has previously held positions in the NHSE pneumonia working group and BTS lung infection Specialist Advisory Group.

Professor Anna Bibby is a Professor of Respiratory Medicine at the University of Bristol. She is Clinical Director for the Somerset Wiltshire Avon and Gloucestershire Lung Cancer Screening Programme and co-chairs the BTS Lung Cancer and Mesothelioma SAG. 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 sits on the Roy Castle Lung Cancer Foundation Clinical Expert Group.

Katie Borg is a trainee advanced nurse practitioner, and she is the lead nurse for the Oxford Special Airways Service at Oxford University Hospitals NHS Foundation Trust. Until recently, she was a clinical research nurse for the University of Oxford, and is an author on over 50 publications. She is still actively involved in clinical research in severe asthma. She helped developed Oxford into an NHS England commissioned Severe Asthma Centre, and she set up and runs specialist services, including biologics, specialist adherence and rapid assessment clinics. She is a prescriber, a member of the Association of Respiratory Nurse Specialists and a member of the BTS Nurse Advisory Group.

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 leads pioneering research that 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 Hannah Burke is a Consultant Respiratory Physician at University Hospital Southampton NHS Foundation Trust, where she works within the integrated COPD service. She has a strong research background, including a Wellcome Trust-funded PhD exploring the mechanisms driving frequent COPD exacerbations, with publications in this area.

She has led several COPD pathway transformation programmes across Hampshire, focusing on early and accurate diagnosis, identification and optimisation of rising-risk patients, early specialist input, and digital technology to support self-management and access to specialist care. Her work focuses on delivering sustainable pathway change and improving outcomes. Nationally, she works with the National Respiratory Audit Programme (NRAP) to support high-quality COPD care and implementation of COPD biologics. She is passionate about medical education and is an

SPEAKERS' BIOGRAPHICAL DETAILS

Honorary Senior Clinical Lecturer at the University of Southampton, shaping the next generation of healthcare professionals.

Paul Burns is a Respiratory and Sleep Clinical Scientist and Head of the Respiratory and Sleep Physiology Service at the Royal Hospital for Children, Glasgow. Following early clinical experience in adult services, he has worked in paediatric respiratory and sleep medicine for the past 17 years.

He leads specialised respiratory and sleep diagnostic services and have expertise in the performance and interpretation of advanced physiological investigations. His clinical and research interests include asthma, congenital lung disease, exercise-induced laryngeal obstruction (EILO), and the physiological assessment of breathlessness. He has a particular interest in cardiopulmonary exercise testing (CPET), using exercise physiology to diagnose, characterise, and monitor respiratory and cardiac disease. His work is focused on translating complex physiological data into clinically meaningful answers that improve patient care.

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) and a 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 section chair for the IARC Lung Cancer Screening Handbook currently in development.

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 Nov 2020 to 2023. She is also a member of the European Respiratory Society (ERS) ILD long range planning committee, an ERS ILD mentor and the secretary of the ERS idiopathic interstitial pneumonia ILD assembly. She is the secretary of the Irish Thoracic Society and member of the ILD working group. She is a trustee of the patient charity Pulmonary Fibrosis Northern Ireland (PFNI) and the NI rare disease partnership.

She graduated from the University of Leeds with an honours degree in medicine and a BSc honours in genetics. She performed a PhD and published her work looking at the cellular interactions and signalling in response to infection and air pollution. She is the principal investigator on a number of clinical research trials (>20 over 5 years) in idiopathic pulmonary fibrosis (IPF) and was the UK Chief Investigator of a major clinical trial on progressive ILDs and a trial in IPF. She is collaborator on a number of grants exceeding £5 million. She is also co-applicant for a British Lung foundation grant for a study in Sarcoidosis.

Dr Chaudhuri has published her experience in prescribing antifibrotics and delivering MDT care and has presented over 40 abstracts pertaining to IPF, antifibrotics and the importance of a multi-disciplinary team approach at all major respiratory conferences. She is collaborator on a number of grants and has co-authored over 50 peer reviewed articles.

Professor Sanjay Chotirmall is an internationally recognized clinician-scientist with an established translational respiratory research group at the Lee Kong Chian School of Medicine, NTU Singapore. To date, he has performed key work on endo-phenotyping pulmonary infection, including the use of next generation sequencing approaches, in the context of chronic inflammatory respiratory diseases that have led to >200 publications including those in Nature Medicine, the NEJM, Cell Host and Microbe, Nature Microbiology, the ERJ and the AJRCCM. He continues clinical practice at Tan Tock Seng Hospital, Singapore and currently serves as Vice Dean (Research) at the Lee Kong Chian School of Medicine, NTU Singapore and Deputy Editor at the American Journal of Respiratory and Critical Care Medicine (AJRCCM).

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Dr Meg Coleman graduated from University College London in 2002 and undertook specialist training in Northwest London including a year working and teaching in Malawi. She joined Imperial College Healthcare NHS Trust as a Respiratory Consultant in 2017 where she works as part of the Imperial Respiratory Infection Team. She specialises in looking after patients with complex infections, TB, bronchiectasis and fungal lung disease. She is Head of Service for Respiratory Medicine and leads the Fungal Service and Fungal MDT. She is a member of the BTS SAG for Acute and Complex Respiratory Infections and a clinical advisor on the BTS MDR TB CAS.

Professor Bronwen Connolly is a Professor of Critical Care at Queen's University Belfast, where she is Deputy Director of the Wellcome-Wolfson Institute for Experimental Medicine, and Honorary Senior Fellow at The University of Melbourne, with over 25 years' experience as a physiotherapist. Her research interests focus on acute respiratory and rehabilitation interventions in critical illness, the recovery, long-term outcome, and survivorship of post critical illness patients, and clinical trial methods. Bronwen is currently Co-Chief Investigator of the MARCH trial (NIHR HTA Programme), investigating the effectiveness of mucoactive drugs in acute respiratory failure, as well as co-Investigator on several other nationally funded trials around ventilation management and recovery. Within the critical care community, Bronwen's roles include Intensive Care Society Director of Research and Chair of the UK Critical Care Research Group.

Dr Aurélie Crabbé is an associate professor in microbiology at the Laboratory of Pharmaceutical Microbiology, Ghent University, Belgium. Her work centers on the role of the lung microbiome role in chronic respiratory diseases - particularly cystic fibrosis, COPD, and bronchiectasis - combining patient-driven insights with in vitro data from physiologically relevant 3D models. Hereby, she aims to harness the lung microbiome to combat infection and dampen excessive host inflammation, paving the way for biotherapeutics targeting chronic respiratory conditions.

Dr Sonya Craig is a Sleep and Respiratory consultant working at University Hospital Aintree, Liverpool (UHLG) where she is lead clinician for sleep medicine. She trained at Cambridge University and the Royal Brompton Hospital, London before completing an MD investigating cardiovascular risk and Obstructive Sleep Apnoea (MOSAIC trial) with Prof. John Stradling in Oxford. Her main research interests are vascular risk in OSA and the delivery of Sleep medicine and care effectively and efficiently within the NHS. She is co-chair of the OSA Alliance, ex-chair of the Sleep Specialist advisory group for BTS and has chaired the digital group for the Optimal Sleep pathway taskforce for NHSE. Her most recent publications are the results of the MERGE study published in Lancet Respiratory and a review of residual sleepiness in OSA (ERR 2022).

Professor Sven-Erik Dahlén (MD, PhD, Fellow of ERS and BPS, born 1952) is senior professor of asthma and allergy research at Karolinska Institutet in Stockholm and the Lung- and Allergy Clinic at Karolinska University Hospital. He has served as Director of the Centre for Allergy Research at Karolinska Institutet 2005-2023. He is recognised for translational research on biological effects of eicosanoids (Leukotrienes, prostaglandins and related compounds), the use of eicosanoid metabolites as biomarkers in asthma, and the role of the mast cell in asthma and allergy. He is leading international and national consortia for phenotyping of severe asthma (PI of BIOAIR & BIOCROSS, Co-PI of U-BIOPRED and 3TR-ABC). He has much experience with both preclinical research and clinical trials for the development of new treatments for asthma and has published about 400 peer reviewed papers. He was ERS Research Director 2007-2011 with responsibility for awards, fellowships, and EU affairs, and chairman of the 2007 ERS Congress in Stockholm. Dahlén has over the years had many appointments for the medical faculty of the Karolinska Institute, including chairmanship of the selection committee for Vice Chancellor and other high positions 2010-2018.

Dr Enya Daynes is a consultant physiotherapist in Leicester specialising in non-pharmacological breathlessness management and pulmonary rehabilitation. She leads the Pulmonary Rehabilitation service and is a member of the leadership team in the Centre for Exercise and Rehabilitation Sciences. Through these roles she has led multi-centre trials in rehabilitation that is embedded and translated into clinical services. She contributed to the British Thoracic Society clinical statement and quality statement, and is the Quality Lead for the Royal College of Physicians Pulmonary Rehabilitation Services Accreditation Scheme.

Dr Alex Dipper is a Respiratory registrar in the South West region. In 2024 she gained her PhD in Translational Health Sciences from the University of Bristol, exploring ways in which current management options for malignant pleural effusion could be

SPEAKERS' BIOGRAPHICAL DETAILS

optimised. She co-ordinated the TACTIC randomised control trial across 11 UK sites, in which medical thoracoscopy, talc pleurodesis and IPC insertion was compared to standard care for patients with malignant pleural effusion. Currently, Alex is in her final year of registrar training working at the Bristol Royal Infirmary.

Professor James Dodd is Professor of Respiratory Medicine at the University of Bristol, UK. He established one of the leading NHS airways services, providing patients with access to specialist asthma and COPD care. At the University of Bristol, James leads the Airways Research Group and co-leads the Respiratory theme within the NIHR Bristol Biomedical Research Centre (BRC), where he oversees programmes in digital health, asthma, and COPD. His interdisciplinary research spans lung health, multimorbidity, digital therapeutics, and AI-enabled predictive modelling. His work on aerosol science informed NHS policy, enabling the safe restoration of respiratory diagnostic services during the COVID-19 pandemic.

James is Clinical Lead for COPD Biologics on the National Respiratory Transformation Programme, working to improve the quality of respiratory care and shape national policy. He also contributes to several UK and European longitudinal studies investigating the early origins of chronic lung disease and sits on the scientific committee of a pan-European collaboration in this field.

Dr Hamish Duff is an ST6 in the South West. He's worked on the BTS Quality Improvement Committee and Specialty Trainee Advisory Group. As well as contributing to national audit and quality improvement projects, he has worked with the QIC on the Learning from Patient Safety Events report looking at Oxygen toxicity and management of Acute Hypercapnic Respiratory Failure.

Orianne Dumas is an epidemiologist and head of the "Integrative Respiratory Epidemiology" team of the Centre for research in Epidemiology and Population Health, at Inserm (French National Institute for Health and Medical Research, Villejuif). Over the past 15 years, she studied exposure to irritants and chemicals in relation to asthma and COPD, with the largest part of her contributions in occupational respiratory diseases. She strongly contributed to the emerging evidence for adverse effects of cleaning products in asthma and COPD, based on French and US studies. She also has strong interest in development of novel methods for occupational exposure assessment.

Professor Heather Elphick is consultant in Paediatric Respiratory and Sleep Medicine at Sheffield Children's Hospital, having trained at Alder Hey and Melbourne Children's Hospitals. She leads the sleep service in Sheffield, managing a comprehensive range of sleep disorders and the 5-bedroomed Sheffield Sleep House. Heather holds an honorary chair with The University of Sheffield and has been invited to speak at national and international conferences in recognition of her collaborative work in the field of technological solutions for children with long-term conditions. She is the theme lead for Long-Term Conditions in Childhood for the NIHR HealthTech Research Centre for Children and Young People.

Heather has led a number of NIHR and MRC-funded research and innovation projects and has collaborated with many national and international higher education and industry partners. Heather is currently leading the NIHR-funded COMFORT-ABLE custom-made non-invasive ventilation mask trial and the PifTik-accredited, NICE-approved Moving On Asthma video resource implementation. She has collaborated on the PLEASANT asthma trial and is a partner in the AIRHUB network for research on indoor air pollution. She is developing and validating several technological solutions for Obstructive Sleep Apnoea in children has contributed to the NICE technology appraisal as a specialist committee member.

Dr Hazel Evans is a Paediatric Respiratory Consultant at Southampton Children's Hospital. She has established expertise in paediatric sleep, ventilation, and respiratory care. She leads the regional long-term ventilation service. Her research focuses on sleep disordered breathing, with current work examining respiratory patterns during healthy preterm infant sleep and potential impacts on neurodevelopmental outcomes. She has published widely on paediatric sleep and ventilation and co-chaired the British Thoracic Society guidelines on sleep diagnostics in children. She also contributed to the latest NICE review of home testing devices for diagnosing obstructive sleep apnoea.

Professor Rosa Faner, PhD, is a Serra Húnter Associated Professor, ICREA academia (2024) at the University of Barcelona (UB), Biomedicine Department, and accredited researcher of the IDIBAPS and CIBER. Fellow of ERS (FERS-2024). Currently her main research motivation relates to the improvement of health care delivery to patients with respiratory diseases through the advancement of our understanding of the mechanisms underlying their heterogeneous clinical

presentations, especially in relation to the lung function trajectories, multiomics and early origins of COPD.

Professor Tom Fardon is a consultant physician in Respiratory and General Internal Medicine at NHS Tayside, Honorary Professor with the University of Dundee, and clinical lead of the Respiratory Specialist Delivery Group within the Centre for Sustainable Delivery. He was the author and Clinical Lead for the Respiratory Care Action Plan for Scotland, chairs the Scottish Asthma PRECISION Group, and the Scottish Asthma Committee within CfSD.

He graduated from Cambridge University in 1999, and was awarded his MD from Cambridge in 2008, winning the Ralph Nobel Prize for his thesis "Optimising Anti-inflammatory Therapy in Atopic Asthma". Professor Fardon's clinical interests include difficult to control asthma, cystic fibrosis, bronchiectasis and chronic bronchial infection.

Dr Marie Fisk is a respiratory, acute and general medicine consultant at Cambridge. She is actively involved in clinical research with a special interest in COPD, care pathways and clinical trials. She holds an NIHR Advanced Fellowship and is a member of the BTS COPD Advisory Group and COPD UK Conference Organising Committee.

Professor David Fishwick is the Chief Medical Adviser for the Health and Safety Executive of GB and also for HSE Northern Ireland. He is a retired Consultant Respiratory Physician in the NHS in Sheffield, an honorary Consultant Respiratory Physician in Manchester and an honorary Professorship in Respiratory Medicine at the University of Sheffield.

He is a Co-Director of the Centre for Workplace Health and health theme co-lead for the Thomas Ashton Institute at the University of Manchester. His main previous clinical and research interests are the prevention and recognition of both occupational lung diseases.

Professor Frits Franssen is the head of the Department of Respiratory Medicine in Maastricht University Medical Center in the Netherlands. He is also member of the Board of Directors of Ciro, a center of expertise for patients with chronic respiratory diseases in Horn, the Netherlands. He is professor of Respiratory Medicine, in particular COPD at Maastricht University. His research focuses on unraveling the pathophysiology, complexity and heterogeneity of COPD and the extrapulmonary characteristics and comorbidities. He was a member of the Lancet COPD Commission and is involved in several ERS and ATS taskforces. He is co-author of >320 peer-reviewed English-language articles, Also, he has his own Podcast for Dutch pulmonologists and trainees on recent developments in the field of COPD. In 2019 he was appointed Fellow of the ERS.

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.

He has a clinical and research interest in exercise physiology, chronic obstructive pulmonary disease and access to services for those with chronic lung disease. Since April 2023 he has been National Clinical Director for Respiratory Disease and Chair of the Adult Specialised Respiratory Clinical Reference Group, NHS England.

Aleksandra Gawlik-Lipinski is a Doctoral Researcher at the University of Leicester and an Honorary Clinical Fellow with the NRAP Children and Young People's Asthma workstream. A nurse with 20 years' healthcare experience, she runs specialist respiratory clinics caring for people with lung conditions of all ages. Her PhD uses linked national data to examine fatal, near-fatal and uncontrolled asthma in children and young people in England, with a focus on ethnic and socioeconomic inequalities. Aleks is Co-Chair of the BTS Statement on the Management of Cough in Children, a NICE Adoption and Impact Reference panel member, an Asthma + Lung UK Council Member, and Vice-Chair of the ARNS Research and Education Sub-Committee.

Professor James E. Gern, MD, is a Professor and Vice Chair for Research in the Department of Pediatrics at the University of Wisconsin-Madison. Dr. Gern's research program focuses on identifying how viral respiratory infections and other environmental and host factors influence exacerbations of childhood asthma. He established the Children's Allergy and Asthma Data Repository (CADRE), which pools and harmonizes data from 13 US birth cohorts. Dr. Gern also leads a laboratory-based program investigating rhinovirus-C types and how allergic inflammation and the airway microbiome influence rhinovirus-epithelial cell interactions and respiratory outcomes in childhood asthma.

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Professor Anthony Gordon is the Head of Division of Anaesthetics, Pain Medicine and Intensive Care at Imperial College London. He works as a consultant on the adult intensive care unit at St Mary's Hospital and is an NIHR Senior Investigator. His research focuses on developing precision medicine in sepsis and he has been the Chief Investigator for multiple clinical trials in critical care, including REMAP-CAP in the UK. He is now NIHR Health Technology Assessment (HTA) Programme Director, where he is keen to encourage innovative and novel trial designs.

Dr Janine Gote-Schniering is an SNSF Starting Grant Assistant Professor at the University of Bern, Switzerland, where she leads an interdisciplinary research program on autoimmune and idiopathic interstitial lung diseases. Her research bridges biological scales to uncover the cellular and molecular mechanisms of lung fibrogenesis and translate these discoveries into imaging biomarkers for precision medicine. Following a PhD in Biomedical Imaging at ETH Zurich and postdoctoral training at the University of Zurich and Helmholtz Munich, she develops integrative approaches that combine single-cell and spatial multi-omics, advanced imaging, and functional human tissue models to connect molecular disease mechanisms with patient-level phenotypes.

Dr Neil Greening is a Consultant Physician and Clinical Academic at Glenfield Hospital, Leicester. He is part of a complex COPD service, managing biologics, lung volume reduction, and home oxygen. He leads a programme of research on the consequences and treatment of exacerbations, with a focus of hospitalisation and skeletal muscle dysfunction.

Dr Frances Grudzinska is an academic clinical lecturer in Respiratory Medicine. Frances was awarded a PhD investigating factors influencing outcomes for older adults with community acquired pneumonia.

Frances' research interest is community acquired pneumonia. Her research spans the immunological deficits seen in pneumonia, understanding patient priorities and outcome measures through to establishing who is at risk of adverse outcome following CAP.

Dr Harsha Gunawardena (MBChB MRCP UK PhD), is a Consultant Rheumatologist and Divisional Clinical Director Neurosciences and Musculoskeletal sciences Division at North Bristol NHS Trust. He co-runs a dedicated CTD vasculitis service alongside the Bristol-Interstitial Lung Disease regional service. He has a clinical academic interest in all aspects of CTD and vasculitis, in particular myositis, CTD-ILD and vasculitis. His PhD was on myositis syndromes and this work was the first to describe several novel autoantibody markers. He has published on myositis overlap syndromes, autoantibodies, CTD-ILD reviews and guidelines. He is a lead member of UK Myositis network, Myositis UK patient charity scientific committee, and on the British Society of Rheumatology Myositis and ILD Guidelines Group.

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).

Professor Liangxiu Han is a full Professor of Computer Science and Academic Director of the Centre for Digital Data Research at Manchester Metropolitan University, where she also serves as Faculty Lead for AI, Digital and Cyber Physical Systems and Deputy Director of ManMet Crime and Well-Being Big Data Centre.

Her multidisciplinary research focuses on pioneering AI and machine-learning methods, scalable intelligent architectures, and multimodal data integration to address complex challenges in healthcare and beyond. She has extensive expertise in harnessing medical images, clinical records, sensor and geospatial data for disease detection, prediction and decision support. As PI or Co-I, she has successfully led and contributed to multi-million-pound national and international

programmes funded by UKRI, NIHR, the EU, industry and charities. She also serves on major UK and international research funding and evaluation panels.

Professor Anna Hansell is Professor of Environmental Epidemiology and inaugural Director of the Centre for Environmental Health & Sustainability, University of Leicester, and Director of the National Institute for Health and Care Research (NIHR) Health Protection Research Unit (HPRU) in Chemical Threats and Hazards. She holds an honorary consultant contract with both the UK Health Security Agency (UKHSA) and University Hospitals of Leicester NHS Trust. She leads on national and international studies examining health effects of environmental exposures, with particular research interests in air pollution, environmental noise, and environmental impacts on respiratory disease. She is Chair of the Committee on the Medical Effects of Air Pollution (COMEAP). Her initial career was in respiratory (hospital) medicine and public health, completing a PhD in epidemiology in 2005.

Professor Pia Hardelid is professor of epidemiology at UCL Great Ormond Street Institute of Child Health where she leads the Environmental Child Health Research Group. Her work is primarily focused on the impact of the physical environment on children's health and development. She is the principal investigator for the Kids' Environment and Health Cohort, a new, whole-nation administrative data birth cohort study funded by Administrative Data Research-UK, and co-director of the HDR-UK Social and Environmental Determinants of Health Driver Programme. She leads the CHILI Hub, which focuses on the child and adolescent health impacts of making nursery and school buildings more energy efficient.

Dr Allie Hare is a consultant physician and specialist in Sleep medicine. She is the President of the British Sleep Society and Hon Treasurer of the British Thoracic Society. She is on the editorial board of the EU Sleep Health Coalition and a founding member of the UK Sleep Health Coalition. She led the national review of outpatient sleep medicine pathways and is currently the NHSE Clinical Lead for the Sleep outpatient transformation project.

Dr Shamil Haroon is a Clinical Associate Professor in Public Health at the University of Birmingham and a clinical epidemiologist specialising in respiratory and chronic disease research. His work spans long COVID, asthma, COPD, and multimorbidity, with a strong focus on using large-scale health data to improve early diagnosis, prevention, and patient outcomes. He has led and contributed to national studies on respiratory disease, primary care case-finding, and the health impacts of environmental exposures. Alongside his academic role, he works part time in general practice and supervises postgraduate researchers. His research aims to inform evidence-based policy and reduce respiratory health inequalities.

Professor Liam Heaney MD FMedSci is Professor of Respiratory Medicine at Queens University Belfast, N Ireland, who trained in Northern Ireland and the Royal Brompton Hospital in London. He is Director of the Northern Ireland Regional Difficult Asthma Service which delivers multi-disciplinary systematic assessment and management of severe asthma. He has chaired the British Thoracic Society Specialist Advisory Group for Asthma, the Evidence Review Group for Difficult Asthma for the British Thoracic Society / Scottish Intercollegiate Guideline on Asthma Management and has been a member of the National Steering Committee for the UK Asthma Guidelines. He co-ordinates the UK Severe Asthma Registry and is UK lead and Member of the Steering Committees for the European Respiratory Society Clinical Research Collaboration, Severe Heterogeneous Asthma Research, Patient centred (SHARP) and International Severe Asthma Registry. He is a Fellow of Academy of Medical Sciences and is Academic Lead for the Medical Research Council UK Refractory Stratification Programme) and was an Invited Commissioner for the Lancet Commission on Asthma 2018 and has published extensively and is widely cited on the clinical assessment and management of difficult to control asthma in adults.

Dr Katherine Hickman qualified from Leeds University in 2000, initially completing a Medicine Rotation and gaining her MRCP before settling into General Practice. She is a GP Partner in Bradford and is Primary Care Clinical Lead for the National Respiratory Audit Programme (NRAP). She is also past Executive Chair of the Primary Care Respiratory Society (PCRS).

Professor Adam Hill is an Honorary Consultant Chest Physician at the Royal Infirmary of Edinburgh, Professor of Respiratory Medicine at the University of Edinburgh, and Dean of Post Graduate Medicine, Public Services Delivery Scotland. He is actively involved in clinical and translational research in respiratory infection. He is the Respiratory Advisor to the Scottish CMO and to the Scottish Government. He

is currently President Elect for the BTS but will be the President of the BTS at the winter meeting.

Dr Barnaby Hirons is a Consultant Respiratory Physician at Croydon University Hospital and a researcher with King's College London. His clinical interests include chronic cough, interstitial lung disease, airways disease, and lifestyle medicine approaches to respiratory health. His research focuses on chronic cough and its relationship with respiratory and neurological disorders, including cerebellar ataxia, neuropathy and vestibular areflexia syndrome (CANVAS).

Professor Ling-Pei Ho is Professor of Respiratory Immunology at the MRC Translational Immune Discovery Unit, University of Oxford, Consultant in Respiratory Medicine and Clinical Lead for Oxford Sarcoidosis Service. Her research group works on the immune mechanisms of lung injury, repair and fibrosis, with a focus on the role of myeloid cells in IPF and fibrotic sarcoidosis. She currently serves on the NIHR Respiratory Translational Research Collaboration (R-TRC) and its ILD Early Clinical Trial work stream, the Sarcoidosis UK Clinical Board, and supports the Research Review Panels for SarcoidosisUK and Action for Pulmonary Fibrosis.

Professor Luke Howard is a Consultant Pulmonologist and Clinical Director of the National Pulmonary Hypertension Service at Hammersmith Hospital, Imperial College Healthcare NHS Trust. His clinical and academic work focuses on pulmonary hypertension, exercise physiology, haemodynamics, and the use of physiological data to guide diagnosis, risk stratification, and treatment in specialist clinics. He has a particular interest in integrating invasive haemodynamics, cardiopulmonary exercise testing, and remote monitoring to improve clinical decision-making and patient follow-up. He leads a major clinical research portfolio and has published extensively in pulmonary vascular disease, exercise physiology, and innovative approaches to physiological assessment in routine and research practice.

Claire Hurlin currently works in West Wales for Hywel Dda University Health Board as the Strategic Head for Community and Chronic Conditions. She leads and co-ordinates a range of activities that promote excellence in chronic conditions management, overseeing diabetes, diabetes prevention, respiratory and epilepsy nursing services as well as the Be Well Service supporting people to self-care. This work is undertaken focusing on chronic conditions management with equitable, value based, accessible care, care closer to home, within communities driving respiratory care and improving patient experience.

At a national level she attends on behalf of the Health Board the Respiratory and Diabetes Clinical Network Groups and is the Clinical Lead for the Home Oxygen Service. This group has developed All Wales documentation for the Home Oxygen Service, and a training package for the performance and interpretation of blood gases. Claire is the first nurse to undertake the role of President Welsh Thoracic Society, and received RCN Lifetime Achievement Award 2024.Claire was joint winner at the RCN Wales awards 2018 for Improving Individual and Population Health, MediWales and NHS Wales's team award winner 2018 for the Virtual Pulmonary Rehabilitation Project (VIPAR). In the dragons den Digital Health Innovation Zone at the International Conference for Integrated Care she was awarded first place for the presentation of the VIPAR model.

Professor John Hurst is Professor of Respiratory Medicine at UCL, with a primary research interest in COPD. His focus is on exacerbation detection and mitigation, imaging and cardiovascular risk. His clinical work from the Royal Free Hospital includes supporting community COPD services, leading the London Alpha-1 Antitrypsin Service, and a joint Respiratory-Immunology service for people living with primary immunodeficiency. He is Pro Vice Provost (International Health) at UCL, and Vice Dean (International) to the Faculty of Medical Sciences. He holds a personal NIHR Global Research Professor award. He is Chair of the European Respiratory Society Publications Committee.

Professor David Jackson is Professor in Respiratory Medicine at King's College London and Clinical Lead for the Severe Asthma and Eosinophilic Lung Disease Service at Guy's and St Thomas' Hospitals in London.

Professor Jackson received his medical degree from UCL in 2001. During his respiratory clinical training in London, he completed an MSc in Allergy followed by a PhD from Imperial College London in 2013. In 2015, following the creation of specialist commissioned severe asthma centres by NHS England, Professor Jackson was appointed as a full-time consultant to lead the regional severe asthma centre at Guy's & St Thomas NHS Trust alongside his asthma research. After four years as a full-time consultant, Professor Jackson took a clinical

reader position at King's in 2019. He was promoted to Professor in 2022. Professor Jackson has published over 100 research articles, authored multiple book chapters, and been an invited speaker at all the major International Respiratory Congresses over recent years. He is committed to furthering education and training in asthma and has created several Guy's-based asthma courses and workshops.

Professor Mariam Jamal-Hanjani is a Clinician Scientist and Group Leader at the UCL Cancer Institute in London. She studied theoretical physics as an undergraduate followed by medicine at UCL and is a practicing thoracic medical oncologist. Her laboratory focusses on the biological processes driving metastatic disease, including tumour-and host-initiated mediators of catabolic states and failure of the adaptive immune system. She is Principal Investigator of the national TRACERx and TRACERx EVO lung cancer evolution studies, and Chief Investigator of the national PEACE research autopsy programme, and the first-in-human LungVax cancer prevention vaccine trial.

Professor David Jayne is Professor of Clinical Autoimmunity at the University of Cambridge, and Honorary Consultant Physician at Addenbrooke's Hospital, Cambridge. in the United Kingdom. He directs a large inflammatory disease clinic with a focus on vasculitis and SLE and a research group conducting clinical trials and translational research. He is current president of the European Vasculitis Society. He was elected to the Académie Nationale de Médicine de France in 2025.

Dr Martin Johnson is Director of the Scottish Pulmonary Vascular Unit (SPVU) at the Golden Jubilee National Hospital in Glasgow. He is also an Honorary Associate Clinical Professor with the University of Glasgow within the Institute of Cardiovascular & Medical Sciences and is Clinical Lead for the pulmonary function laboratories in North West Glasgow including the West of Scotland Heart and Lung Centre. Dr Johnson's research interests include pulmonary hypertension and cardiopulmonary physiology, in particular in application to exercise testing and exercise training.

Professor Sebastian Johnston is Professor of Respiratory Medicine & Allergy at the National Heart and Lung Institute, Imperial College London. He has worked in respiratory virus research for 30+ years. He developed the mouse model of rhinovirus infection and rhinovirus-induced exacerbations of chronic airway diseases (asthma & chronic obstructive pulmonary disease [COPD]) and has used these models to produce a rhinovirus vaccine and in pre-clinical studies to test novel therapies for these conditions. He performs human experimental rhinovirus infection studies in healthy subjects, and in patients with asthma, bronchiectasis and COPD, academically, and commercially at his CRO Virtus Respiratory Research Ltd.

Dr Mark Juniper is a consultant respiratory physician is Swindon, and medical director at Health Innovation West of England. He is lead Clinical Co-ordinator at the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) and an author of the report on Pleural Procedures published in November 2026 which highlights areas for improvement in the care of patients treated with chest drains or having a pleural aspiration. He will present key findings and recommendations from this report.

Dr Renata Jurkowska, Reader and UKRI Future Leaders Fellow, School of Biosciences, Cardiff University, is a molecular biologist with general interests in epigenetics, stem cells, and lung biology. She obtained her M.Sc. degree in Biotechnology at Warsaw University (Poland), completed her PhD, and two postdocs in Germany before joining BioMed X Innovation Center (Germany), where she worked at the interface between academia and industry, leading a biomedical preclinical project in the field of respiratory medicine.

She is currently a Reader at the School of Biosciences at Cardiff University (UK) and, since 2024, also a UKRI Future Leaders Fellow. Her research focuses on understanding epigenetic mechanisms driving the development of lung diseases. Her group employs sequencing-based approaches in combination with cutting-edge epigenetic editing tools and organoid models to advance the biological understanding of lung cell regeneration and accelerate the discovery of epigenetic therapeutics and biomarkers for respiratory diseases. Her research has been funded by the Academy of Medical Sciences Springboard Award, BBSRC, UKRI Future Leaders Fellowship, and Wellcome Discovery Award.

Dr Sonal Kansra is a Consultant in Paediatric Respiratory Medicine at Sheffield Children's Hospital, with clinical interests in asthma, aerodigestive disorders and wider determinants of respiratory health. He contributes to postgraduate education through roles

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with the HERMES Examination Committee and the Respiratory Medicine CSAC. Dr Kansra is President of the British Paediatric Respiratory Society, with a focus on collaborative working to improve respiratory care for children and young people.

Dr Samantha Kon is a Consultant Respiratory Physician at Hillingdon and Harefield Hospitals, UK. Her PhD and research interests focus on the use of simple functional outcome measures and health-related quality-of-life instruments in COPD and pulmonary rehabilitation (PR). Her clinical interests are in COPD (including respiratory failure and home ventilation) and integrated respiratory care, and she leads the Hillingdon Integrated Respiratory Service in Northwest London. She is a member of the London Respiratory Clinical Network Leadership Group and co-leads the London PR Network. Dr Kon is also the Co-chair of the BTS PR Specialist Advisory Group and a member of the National Respiratory Audit Programme PR Advisory Group. She led the development of the BTS professional framework for PR and has contributed to the BTS PR Quality Standards.

Dr Maria Teresa Landi, MD, PhD, is Senior Advisor for Genomic Epidemiology and Senior Investigator in the Division of Cancer Epidemiology and Genetics at the US National Cancer Institute. An internationally recognized leader in cancer genomic epidemiology, she directs the Sherlock-*Lung* and EAGLE studies, major multi-institutional investigations of lung cancer in never-smokers and smokers, respectively. She also founded and leads the MelaNostrum Consortium, an international collaboration focused on melanoma genetics and genomics. Her research integrates epidemiology, multi-omics and deep learning to characterize cancer susceptibility, mutational processes, tumor evolution and clinically relevant molecular subtypes. Dr Landi has authored more than 400 publications, including influential studies in *Nature, Science, Nature Genetics* and *Cancer Discovery*, and has been highlighted by the NIH and featured in international media, including *The New York Times*.

Dr Dawn Lau works within a marvellous Multidisciplinary team of the All Wales Adult Cystic Fibrosis Centre (AWACFC), as well as within the non-CF Bronchiectasis Service as Consultant Chest & CF Physician at the Cardiff & Vale University Health Board. She has taken on the role of Chair of the BTS Cystic Fibrosis Specialty Advisory Group (SAG), working within a mini-MDT of SAG members. Another well-worn hat is involvement in Medical Education, and in this role she leads on the Undergraduate agenda within the Health Board working closely with the Cardiff University School of Medicine.

Professor Y C Gary Lee of the University of Western Australia is a recognized leader in clinical/translational pleural research. His program at the Institute for Respiratory Health consists of 10+ clinicians, allied health professionals and scientists. He has delivered 500+ invited pleural lectures in 34 countries and published 390 papers (H-index 69; citations 18500+). His program has trained 30+ clinical pleural specialists. He leads the FHRI Centre for Innovative Pleural Research and the Australasian Malignant PLeural Effusion trial network. He was the recipient of the Society Research Medal of the Thoracic Society of Australia/NZ and of he Asian Pacific Society of Respirology.

Dr Janice Leung is a respirologist at St. Paul's Hospital, Vancouver, Canada, and an Assistant Professor at the University of British Columbia. She is the Tier 2 Canada Research Chair in Translational Airway Biology and the GlaxoSmithKline Chair in COPD. Her research interests are in novel exposures leading to accelerated airways disease, including HIV, vaping, and cannabis smoking. She is supported by grant funding from the Canadian Institutes of Health Research, the Canadian Cancer Society, and the Heart and Stroke Foundation.

Professor Marc Lipman is Professor of Medicine at University College London and Honorary Consultant in Respiratory and HIV Medicine at the Royal Free Hospital where he leads the mycobacterial clinical service. He chairs the UK Joint TB Committee, and NTM Network UK, the country's largest NTM clinical research group. He is Director of UCL-TB, UCL's multidisciplinary TB research platform from which the UCL WHO Collaborating Centre for TB was developed. Marc's research focuses on improving patient outcomes and enabling high-quality healthcare delivery.

Professor David Lomas is Professor of Medicine at University College London (UCL), Honorary Consultant Respiratory Physician at UCLH and the Royal Free Hospitals and NIHR Emeritus Investigator. He was Vice Provost (Health), Head of UCL Medical School and Director of the UCL Partners AHSC from 2015-2023. He previously chaired the Respiratory Therapy Area Unit Board at GlaxoSmithKline and was Deputy CEO of the Medical Research Council (UK).

David is currently Chair of Trustees, Association of Physicians of Great Britain and Ireland, Vice Chair of Trustees, Asthma and Lung UK and a Board member of the Anti-Doping Laboratory Qatar, Rosetrees Charity and AlphaDetect (USA). His research has focused on α_1-antitrypsin deficiency and COPD.

Professor Christopher D Lucas is the chair of Pulmonary Immunity and Regeneration, an MRC Clinician Scientist, and Honorary Consultant in Respiratory Medicine at the Institute for Regeneration and Repair, University of Edinburgh, Scotland. Upon completion of clinical training and award of a Wellcome Trust Fellowship he moved to the University of Virginia, USA, to study cell death in tissue regeneration and repair in the injured lung. In 2020 he moved back to the University of Edinburgh to establish his own research group, focusing on macrophage-epithelial crosstalk and how this regulates tissue biology and epithelial cell regeneration after lung injury.

Dr Rahul Mahida is an MRC Clinician Scientist at the University of Birmingham and an Honorary Consultant in Respiratory Medicine at University Hospitals Birmingham NHS Foundation Trust. His translational research focuses on the role of inflammatory pathways, extracellular vesicles and immune dysfunction in the pathogenesis of Acute Respiratory Distress Syndrome and Interstitial Lung Diseases, with the aim of developing novel therapeutic strategies.

Professor William Man is a Consultant Chest Physician at Royal Brompton and Harefield Hospitals, Guy's and St Thomas' NHS Foundation Trust, and Professor of Respiratory Medicine at King's College London. A full-time NHS clinician, he works across the spectrum of respiratory care, from primary through to tertiary settings, alongside leading a sustained NIHR-funded programme of respiratory health services research.

Professor Man was Co-Chair of the British Thoracic Society Clinical Statement and Quality Standard on Pulmonary Rehabilitation and currently chairs the European Respiratory Society Rehabilitation and Chronic Care Group. He is also Past President of the Association for Respiratory Technology and Physiology (ARTP).

Professor Stefan Marciniak is Professor of Respiratory Science at the University of Cambridge and an Honorary Consultant Respiratory Physician at Addenbrooke's and Royal Papworth Hospitals. His laboratory at the Cambridge Institute for Medical Research investigates how stresssignalling pathways shape the development and progression of lung disease. Clinically, he specialises in pleural medicine, with a particular focus on the genetics of pneumothorax. He coleads the NIHR National Research Strategy Group in Pleural Disease, helping to shape national research priorities. He also codirects the NHS Rare Disease Collaborative Network for Familial Pneumothorax, chairing its bimonthly national multidisciplinary team meeting.

Dr Anthony Martinelli is a respiratory physician-scientist with interests in airways disease, iron metabolism, and infection. He trained in Cambridge as a Wellcome Clinical PhD Fellow and Clinical Lecturer, where his research focused on using genetic approaches to better understand host cell biology. Alongside his laboratory work, he has led multi-centre studies on pneumothorax, including as part of the INSPIRE network. He has enjoyed serving as a previous representative on the BTS Science and Research Committee, and as a member of the COPD and Biologic Therapies Clinical Statement Group.

Professor Nick Maskell is Professor of Respiratory Medicine University of Bristol and Honorary Consultant Bristol NHS Foundation Trust, Bristol, England.

He leads the Academic Respiratory Unit (ARU) at the University of Bristol and is the current Clinical Director and Respiratory theme lead of the 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 79 with over 300 peer reviewed publications and >31000 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, the 2019 ERS malignant pleural effusion taskforce statement, the 2023 BTS pleural disease guidelines, 2024 ERS pneumothorax guidelines. Nick is also the Chairman of the Board of Trustees for Mesothelioma UK and immediate past President for the British Thoracic Society.

Helen May is an Advanced Nurse Practitioner specialising in Respiratory Medicine at Wythenshawe Hospital, Manchester Foundation Trust. She has been working as a clinician in Respiratory since 2020. Her role within the sleep service began in 2023, when she lead a successful quality improvement project developing the urgent CPAP pathway. Besides working within the sleep service her role also includes leading

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the Long Covid Service in South Manchester. Her experience in chronic fatigue provided motivation to improve the quality of life for patients with chronic fatigue and sleep related breathing disorders. Prior to working in Respiratory Medicine, Helen worked as a nurse in Cardiothoracic Surgery for 25 years.

Professor Danny McAuley is a Consultant and Professor in Intensive Care Medicine at the Regional Intensive Care Unit at the Royal Victoria Hospital and Queen's University of Belfast. He undertook his training in Belfast, Birmingham, London and San Francisco. He is NIHR Scientific Director for research programmes. He has several research interests including Acute Respiratory Distress Syndrome, recovery following critical illness and clinical trials.

Professor Melissa McDonnell is a Consultant Respiratory Physician at Galway University Hospitals and NUIG, Ireland. Her current clinical and research interests include difficult to treat airway infections, particularly bronchiectasis, COPD, non-tuberculosis mycobacterial infections and cystic fibrosis. She has received several young investigator awards for research in bronchiectasis and has over 100 publications in peer-reviewed journals. She is a co-author of national and international guidelines for bronchiectasis and COPD. She currently leads a specialist bronchiectasis clinic for complex patients with difficult to treat infections in Galway and is a Principal Investigator at NUIG for several phase 1-3 clinical trials.

Dr Kirsty McGee is a Postdoctoral Researcher at the Department of Inflammation and Ageing, University of Birmingham. Her research spans innate immune dysfunction and neutrophil biology across infection, critical illness and respiratory disease, with a particular focus on how impaired innate immunity drives poor outcomes in acute and chronic lung disease. She leads laboratory studies investigating the host immune response to infection, integrating mechanistic immunology with clinical cohorts to identify pathways that could improve host defence in acute care.

Dr Ben Messer is a Consultant in Critical Care Medicine and Long-term Ventilation in Newcastle-upon-Tyne and the clinical lead of the North East Assisted Ventilation Service. His main critical care interest is acute non-invasive respiratory support. Within home ventilation, his interests are tracheostomy ventilation, upper airway dysfunction and secretion management in MND, and the respiratory and perioperative care of neuromuscular patients. He was the chair of the multi-professional group which produced the model of care document for complex home mechanical ventilation services published by the British Thoracic Society in 2024.

Dr Eleanor Mishra is Clinical Associate Professor at the University of East Anglia and Respiratory Consultant and Pleural Lead at the Norfolk and Norwich University Hospital. She leads a comprehensive pleural service including day-case therapeutic thoracoscopy, image-guided pleural biopsy, indwelling pleural catheter management and ambulatory pneumothorax care. Her research focuses on malignant pleural effusions and pleural infection. She was Chief Investigator for the NIHR RfPB REPEAT study, which derived and validated the RED score, the first clinical tool to predict time to next pleural procedure in patients with malignant pleural effusion. She is currently Chief Investigator for TAPDROP, a multicentre observational study examining which patients benefit from pleural fluid drainage.

Dr Phil Mitchelmore is a Consultant in Respiratory Medicine at the Royal Devon University Healthcare NHS Trust and a midcareer fellow at the NIHR Exeter BRC. He completed a PhD at the University of Exeter examining Pseudomonas aeruginosa epidemiology and microbial diversity in bronchiectasis. His clinical and research interests centre on chronic lung infection in bronchiectasis and cystic fibrosis. He serves as an RSOB member for CTAP, chairs the South & West CTAP regional network, is clinical consultant to the Citizen Phage Library, and chairs the PHAGEUK respiratory MDT. He collaborates on TrailfinderCF and AIRNET.

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 a MSc in Epidemiology from the LSHTM. 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 BTS Quality Improvement Committee, Co-Chair of the BTS/SIGN Severe Asthma Guideline, 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 Education Committee representative and is a member of the ERS spirometry group. She is passionate about supporting training and education in respiratory physiology and occupational asthma.

Dr Prasad Nagakumar is Paediatric Respiratory Consultant and Deputy Director, Research and Development at Birmingham Children's Hospital, UK and Honorary Associate Professor and Department of Inflammation and Ageing at the University of Birmingham, UK.

Prasad's clinical and research interests focus on understanding the pathophysiology of preschool wheeze, asthma, and other chronic respiratory diseases in children, and the influence of broader social determinants on respiratory health. He is also interested in the applied use of artificial intelligence and digital monitoring in paediatric respiratory care , particularly remote monitoring, and how routinely collected home and clinical data can be used to detect deterioration earlier and personalise management.

Dr Claire Nolan is a Senior Lecturer in Physiotherapy at Brunel University of London with clinical expertise in pulmonary rehabilitation. She completed her PhD in Imperial College London in 2018 on gait speed and prognosis in idiopathic pulmonary fibrosis. Dr Nolan's research interests include increasing engagement in rehabilitation programmes and non-pharmacological management of people living with multiple long-term conditions. She has expertise in mixed-methodologies, feasibility and randomised controlled trials as well as intervention development. She was a member of the committees that wrote the BTS Clinical Statement on Pulmonary Rehabilitation and Quality Standards for Pulmonary Rehabilitation.

Chelliah Paramasivan is a Specialist Respiratory Physiotherapist at Cambridge University Hospitals NHS Foundation Trust, with over 20 years of experience in cardiorespiratory physiotherapy. He leads regional severe asthma physiotherapy services and provides specialist respiratory physiotherapy for breathing pattern dysfunction, large airway collapse, non-invasive ventilation, and breathlessness management.

Chelliah has a particular interest in the use of physiological assessment, including cardiopulmonary exercise testing (CPET), to support assessment and clinical decision-making in patients with breathing pattern dysfunction. He has contributed to NIHR-funded research, clinical trials, and service evaluations involving spirometry, cardiopulmonary exercise testing, and six-minute walk testing. He has presented research abstracts at BTS and ERS conferences and has co-authored publications on the use of CPET in dysfunctional breathing.

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 Clinical Co-lead for the NHSE Chronic Cough Elective Care Recovery workstream, is currently co chairing the BTS quality standards for cough group and NEurocough CRC and previously co-chaired the BTS clinical statement on cough in adults.

Dr Irem Patel is a consultant integrated respiratory physician at Kings College Hospital NHS Trust, Joint Respiratory Clinical Director NHSE (London) and COPD clinical lead for the National Respiratory Audit Program.

Dr Tanya Patrick is a Respiratory Registrar in South London. Her interests are lung cancer early detection through screening and interventional pulmonology. She has completed a PhD at University College London on optimising the evaluation of cardiorespiratory comorbidities in lung cancer screening. She is one of the current British Thoracic Oncology Group Respiratory trainee representatives. She has been sub-investigator for multiple clinical trials of novel COPD therapeutics recruiting through lung cancer screening and is currently leading the RESCAN multi-centre service improvement project to evaluate lung cancer screening eligibility after radical treatment for lung cancer.

Dr Kyle Pattinson is Associate Professor at the University of Oxford and Honorary Consultant Anaesthetist at Oxford University Hospitals. His research focuses on the brain mechanisms of

breathlessness, integrating functional neuroimaging, virtual reality, respiratory physiology, and experimental medicine to understand how breathlessness is perceived and modulated. He has led studies of breathlessness in conditions including COPD, asthma, and long COVID, with particular expertise in respiratory interoception and the neural circuitry of breathing.

Professor 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 programs 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 Manuela Platé leads the Lung Regeneration Group in the UCL Respiratory Department at University College London. Her research investigates the molecular mechanisms underlying idiopathic pulmonary fibrosis and chronic obstructive pulmonary disease, with a particular focus on alveolar regenerative failure. Her group uses advanced human models and technologies—including 3D organoids, precision-cut lung slices and next-generation sequencing—to study alveolar regeneration, epithelial–mesenchymal crosstalk, and the genetic and transcriptional changes occurring in chronic lung disease. Alongside her research, Manuela co-leads the Division's Athena Swan self-assessment team and is Treasurer of the British Association for Lung Research.

Professor Joanna Porter is Professor of Respiratory Medicine at University College London and Clinical Lead for the NHS Interstitial Lung Disease service at University College London Hospitals NHS Foundation Trust. Her clinical and research interests focus on ILD, including autoimmune and inflammatory lung disease, mechanisms of lung injury and repair, and the early detection and progression of fibrosis. Her research investigates interactions between neutrophils and other immune cells within the alveolar space, novel PET imaging approaches to identify biomarkers of disease activity and progression, and the long-term pulmonary sequelae of infections including tuberculosis and COVID-19. She is also actively involved in undergraduate and postgraduate medical education.

Dr Tim Quinnell has worked at Royal Papworth Hospital, Cambridge since 2004 and has led one of the UK's largest sleep and ventilation centres since 2021. He specialises in respiratory and non-respiratory sleep disorders, domiciliary noninvasive ventilation and weaning from invasive ventilation. His MD was on genetic and electrophysiological aspects of narcolepsy. He was Chief Investigator for the NIHR Trial of Mandibular Advancement Devices in OSA and recently completed an NIHR trial of combination therapy in OSA. He is past chair of the BTS Sleep SAG. Dr Quinnell is actively engaged in providing and developing multidisciplinary sleep medicine education.

Dr Charles Reilly is an NIHR Advanced Clinical Academic Fellow, Consultant Respiratory Physiotherapist at King's College Hospital NHS Foundation Trust, and Adjunct Reader in Respiratory Care and Health Services Research at the Cicely Saunders Institute of Palliative Care, Policy & Rehabilitation, King's College London. His research focuses on improving the assessment and management of chronic breathlessness through mechanistic research, health services evaluation, and the development and implementation of accessible, evidence-based non-pharmacological interventions. Since his appointment as Consultant Respiratory Physiotherapist in 2014, Dr Reilly has combined clinical leadership with an internationally recognised programme of respiratory research. He has received several prestigious NIHR awards, including an NIHR Clinical Lectureship, an NIHR Advanced Clinical Academic Fellowship, and the NIHR Rising Research Leaders Prize (2026), recognising his outstanding contributions to patient-centred respiratory research, innovation, and clinical academic leadership.

Professor Robert Rintoul is a physician scientist. He is Professor of Thoracic Oncology in the Department of Oncology, University of Cambridge and Honorary Consultant Respiratory Physician, Royal Papworth Hospital.

He leads the Thoracic Cancer Programme of CRUK Cambridge Cancer Centre and is Director of the Papworth Trials Unit Collaboration. He leads the Clinical Advisory Group of the UK Lung Cancer Coalition (UKLCC) and co-directs the Cambridgeshire and Peterborough lung cancer screening programme.

Prof Rintoul's group focuses on translational clinical trials in biomarker discovery for early detection of primary and second primary lung cancer. He also directs Mesobank UK, the UK national bioresource for malignant mesothelioma (www.mesobank.com). His work is funded by NIHR Cambridge Biomedical Research Centre, Cancer Research UK, Medical Research Council and Asthma and Lung UK.

Dr Hilary Robbins is an epidemiologist at the International Agency for Research on Cancer (IARC/WHO) in Lyon, France. She co-leads the IARC Risk Assessment and Early Detection (RED) Team, a group of 18 scientists, fellows, and staff from 14 countries, and the Lung Cancer Cohort Consortium (LC3, lc3.iarc.who.int). The LC3 gathers data and biospecimens from 26 cohorts worldwide, comprising 3 million participants and 67,000 lung cancer diagnoses during follow-up. Dr. Robbins' research includes studies on lung cancer epidemiology, risk prediction, and biomarkers for early detection.

Professor Jody Rosenblatt is Professor of Cell Biology at the Randall and Cancer Centres at King's College London and on secondment at the Francis Crick Institute. She returned to London in 2019 after running her lab in her hometown of Salt Lake City, Utah at the Huntsman Cancer Institute, where she held the H.A. and Edna Benning Chair and a HHMI Faculty Scholarship. Before this, she did an independent postdoc at the LMCB at UCL following a PhD at UCSF. Here she runs the Cell Extrusion Lab with part of her lab still at King's College London.

Dr Hitasha Rupani is a consultant respiratory physician at University Hospital Southampton (UHS) and the holder of a Research Leaders Programme (RLP) award. In addition to inpatient clinical work, she leads the severe asthma service at UHS.

Her research focuses on improving our understanding and treatment of asthma and asthma attacks. She came to UHS to do her PhD, which she completed in 2015. This investigated the immune responses of people with severe asthma to viral infections and how they vary. As a consultant she has continued asthma research including looking at asthma outcomes after an intense education programme, and trialing different ways of diagnosing asthma. She also set up a multi-centre study also looking at immune responses to COVID-19 vaccines in patients with severe asthma.

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.

Professor Liz Sapey is an academic respiratory physician at the University of Birmingham, where she is Head of the School of Medical Sciences. She is President of the British Association for Lung Research and the Association of Physicians of Great Britain and Ireland. Liz is passionate about research into lung health and disease, and mentoring researchers from all professional and personal backgrounds. She is also founding Director of PIONEER, the HDR UK Health Data Research Hub for Acute Care. PIONEER works with patients, researchers and industry to use health data safely to develop and evaluate innovations that improve patient care.

Professor Esther Irene Schwarz is Professor of Respiratory Medicine at the University of Zurich and Deputy Head of the Department of Pulmonology at University Hospital Zurich, where she also leads the Sleep Disorders Centre and Home Mechanical Ventilation Unit. She is Head of Assembly 4 (Sleep-Disordered Breathing) of the European Respiratory Society. Her clinical and research career includes an Swiss Lung Foundation and a ERS fellowship at the Lane Fox Unit and Sleep Disorders Centre at Guy's and St Thomas' NHS Foundation Trust in London, UK.

Professor Aaron Scott is an Associate Professor in Respiratory Science at the University of Birmingham. His research focuses on how dysfunction of the innate immune system contributes to respiratory disease, with particular interests in macrophage and neutrophil biology, respiratory infection and COPD. His group investigates how environmental exposures, including

cigarette smoke, e-cigarettes and inhaled pollutants, alter lung immune function and susceptibility to disease. His current work aims to understand how these exposures shape immune resilience and recovery following respiratory infection.

Professor Calum Semple OBE grew up in Glasgow and Edinburgh. He is a doctor who specialises in treating children with breathing problems and studying serious infectious diseases. He studied medicine at Oxford University and has a PhD in Clinical Virology from University College London. In 2006, he became a Consultant at Alder Hey Children's Hospital in Liverpool, where he treats children with respiratory conditions. He's also a Professor at the University of Liverpool, focusing on outbreak medicine and child health. He continues these roles on a reduced basis during his time with the Scottish Government, which is part-time.

For over 30 years, Calum has studied serious viral outbreaks. He helped set up ISARIC in 2012, an international organisation that brings together experts to tackle severe outbreaks and pandemics. His research has covered diseases including HIV/AIDS, bronchiolitis, flu, Ebola, Mpox, COVID-19 and Hepatitis. Between 2014 and 2016, he led medical research in Sierra Leone during the Ebola outbreak. For this work, he and his team received the Queen's Ebola Medal. He also won a Commonwealth Award in 2019 for his continued support of Ebola survivors.

Calum has advised governments and the World Health Organization during major health crises, including the 2009 Swine Flu pandemic and the COVID-19 pandemic. He was awarded an OBE in 2020 for his work during COVID-19.

Claire Slinger is currently working as Consultant SLT and Service Lead for Preston Complex Breathlessness/ Airways service, Lancashire Teaching Hospitals Trust.

Professional Advisor to Royal College of Speech and Language Therapists (Adult Respiratory care). Co-author RCSLT Position Paper Upper Airway disorders. Current chair of the British Thoracic Society Cough Specialist advisory group. Claire has worked clinically with laryngeal disorders for 25 years and is currently on the working group for the BTS quality standards for chronic cough and the NHSE elective programme for cough pathway optimisation as a joint clinical lead.

Dr Nicola Smallcombe is a Respiratory Registrar in London and currently undertaking a PhD at King's College London. Her research focuses on COPD exacerbations, particularly exacerbation severity, treatment response and patient-reported outcomes. She is a sub-investigator in several clinical trials of biologic therapies for COPD and a trainee representative on the BTS Science and Research Committee. She has also co-led the RSM Respiratory SCE series and Respiratory Optimise interview course.

Dr Elaine Soon is an MRC Clinician Scientist at the Cambridge Institute for Medical Research in the University of Cambridge and Honorary Consultant at Cambridge University Hospital NHS Trust. She read Medical Sciences at King's College, University of Cambridge before undertaking her PhD in the role of inflammation in BMPR2 mutation-associated pulmonary vascular disease. Her current research focuses on how genetic mutations, inflammation and defects in the integrated stress response lead to pulmonary hypertension and right heart failure; with a special interest in animal models of these factors. From the clinical side she has a particular interest in biomarkers, especially with regard to pulmonary hypertension and inflammation; and in the treatment of pulmonary embolism.

Professor Michael Steiner is Professor of Respiratory Medicine at the University of Leicester and an Honorary Consultant at University Hospitals of Leicester. He also serves as Deputy Chief Medical Officer for the Leicester, Leicestershire and Rutland Integrated Care Board. His clinical work focuses on management of advanced COPD and other chronic respiratory diseases and home mechanical ventilation. His academic interests span pulmonary rehabilitation, nutrition and skeletal muscle function in respiratory disease, integrated care and chronic disease management. He is Editor-in-Chief of Chronic Respiratory Disease.

Dr Iain Stewart is an Advanced Research Fellow at Imperial College London's National Heart and Lung Institute. As analytical lead for the Margaret Turner-Warwick Centre for Fibrosing Lung Disease, his research focuses on using data analytics to understand molecular pathways in progressive lung conditions, implementing advanced statistics upon clinical data to improve patient outcomes. He serves as a Statistical Editor for Thorax, Deputy Editor for BMJ Open Respiratory Research, and is an Honorary Senior Statistician at Guy's and St Thomas' NHS Foundation

Trust. He has published 100 original research articles and leads multimodal work packages across several interdisciplinary collaborations.

David A. Stoltz, M.D., Ph.D., is the Sterba Professor of Medicine and Chief of the Division of Pulmonary, Critical Care, and Occupational Medicine at the University of Iowa. A physician-scientist, he cares for critically ill adults and people with cystic fibrosis (CF), primary ciliary dyskinesia (PCD), and asthma. Since 2008 he has led development of porcine models of airway disease, advancing understanding of CF and PCD lung disease origins and therapies. A recipient of the NIH Director's New Innovator Award, he is PI on an NIH P01 studying PCD and an elected member of the ASCI, AAP, and ACCA.

Ema Swingwood is a Consultant Physiotherapist in Critical Care at University Hospitals Bristol and Weston NHS Foundation Trust. Her area of clinical expertise and interest is ventilation, weaning and complex airway clearance. She has lectured at International conferences over a number of years around these topics, alongside postgraduate lecturing at University College London.

Her PhD work focused on the use of Mechanical Insufflation-Exsufflation in the intubated population which was funded through the NIHR Clinical Academic Research Fellowship pathway. Post-doctoral work continues to examine the role of airway clearance interventions in the ICU population. She is a current Senior Clinical Practitioner Research Award holder (2025-7). Most recently she was a co-applicant on a successful NIHR Health and Care Professions Internship Programme for Southwest Central and lead applicant NIHR Undergraduate Internship Placement award.

She sits on the Intensive Care Society (ICS) Physiotherapy Professional Advisory Group (Deputy Chair) and Education Committee. Alongside these roles, Ema is the current Programme Director for the ICS annual congress (SOA) and Deputy Chair for Professional Affairs. She has been part of the multi-professional authorship for BTS/ICS documents related to Respiratory Support Units, Weaning Centres and Complex Home Ventilation.

Dr Karl Sylvester is Consultant Healthcare Scientist and Head of Joint Respiratory Physiology at both Cambridge University Hospitals and Royal Papworth Hospital NHS Foundation Trusts. He is also clinical lead for the breathlessness pathways at the Cambridgeshire and Peterborough Community Diagnostic Centres. His specialist interest is the performance and interpretation of cardio-pulmonary exercise testing, especially in its identification of breathing pattern disorders. He is faculty lead for the ARTP's annual CPET 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. He is the current President of ARTP and has previously been ARTP Honorary Chair. He also sits on the BTS Science & Research committee representing ARTP.

Dr Hui-leng Tan is a consultant in paediatric respiratory medicine at Chelsea and Westminster Hospital. She completed her paediatric respiratory medicine training at Royal Brompton and Great Ormond Street hospitals and paediatric sleep training at the University of Chicago. She is currently co-chair of the British Paediatric Sleep Society.

Dr Amanda Tatler is the Chairperson of the British Association for Lung Research and an Associate Professor within the Nottingham Respiratory Biomedical Research Centre at the University of Nottingham. Her research interests include the pathological mechanisms driving lung tissue remodelling in chronic lung diseases. She has previously undertaken periods of post-doctoral training at both the University of California San Francisco and Harvard Medical School and has been the recipient of two externally funded fellowships (NC3Rs and Medical Research Foundation). Her research is specifically focused on utilising cell- and tissue-based models to understand airway remodelling and lung fibrosis. She has been researching the underlying cellular and molecular mechanisms driving TGFβ-driven airway and parenchymal remodelling for almost 20 years and has published scientific articles, invited reviews and editorials on the subject. Her research has delineated novel pathways driving remodelling in the lung epithelium and airway smooth muscle layer, contributed to the understanding of bronchoconstriction-driven airway remodelling in asthma, and highlighted unique pathways that help drive the progressive nature of IPF. In recent years, she has pioneered the use of precision-cut lung slices as a model of tissue remodelling and has recently established a novel method for applying cyclical mechanical stretch to lung slices to model biomechanical processes in human lung tissue ex vivo.

SPEAKERS' BIOGRAPHICAL DETAILS

Dr Alexandra Teagle is a Consultant in Respiratory Medicine at University Hospitals Plymouth NHS Trust, with a subspeciality interest in lung cancer. She completed the majority of speciality clinical training in South East Scotland and obtained CCT in 2025. She has a PhD from the University of Edinburgh in optimising immune responses to solid tumours such as lung cancer. Her clinical and research interests are in lung cancer, bronchoscopy and emerging approaches to early-stage diagnosis. She is a member of the BTS lung cancer and mesothelioma specialist advisory group (SAG).

Dr Selina Tsim is a Consultant Respiratory Physician at the Glasgow Pleural Disease Unit, Queen Elizabeth University Hospital in Glasgow, and NHS Research Scotland Senior Research Fellow and Honorary Clinical Senior Lecturer at the School of Cancer Sciences, University of Glasgow. She completed her PhD in imaging and blood biomarkers in pleural malignancy and mesothelioma in 2018 and has been chief investigator and principal investigator in a number of clinical and translational studies in pleural disease, lung cancer and mesothelioma.

Professor Alice Turner is a professor in respiratory medicine at University of Birmingham with clinical work focussing on AATD and COPD, and postgraduate qualifications in medical education, leadership and quality improvement (QI). Her roles related to healthcare improvement have included membership of the BTS QI (2019-22) and standards of care committees (2023-), directorship of the NIHR Midlands Patient safety Research Consortium (PSRC), QI lead at the National Respiratory Audit Programme (NRAP) and more recently clinical lead for NRAP.

Dr Don Urquhart is a Consultant and Honorary Reader in Paediatric Respiratory and Sleep Medicine at the Royal Hospital for Children and Young People and University of Edinburgh. Don is a clinician and researcher in 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 90 peer-reviewed publications and has led several investigator-initiated studies in the fields of exercise in CF (most recently the ExACT-CF study) and also in sleep medicine. Don has been PI on a variety of drug trials, and has contributed to the writing of international exercise testing guidelines.

Professor Ioannis Vogiatzis Professor of Rehabilitation Sciences, FHW APVC for R&KE, Northumbria University Newcastle, has over 30 years' experience in pulmonary rehabilitation and cardiopulmonary exercise testing. He served for three years as Head of Assembly 1 of the European Respiratory Society (ERS) and is currently Director of the ERS Educational Programmes. He has contributed to several joint ERS/ATS statements on pulmonary rehabilitation, skeletal muscle dysfunction, cardiopulmonary exercise testing, respiratory muscle function and physical inactivity in COPD. He co-chaired the ATS/ERS policy statement on enhancing the global implementation, use and delivery of pulmonary rehabilitation and served on the WHO Development Group for COPD Rehabilitation. His research focuses on exercise intolerance in chronic lung disease and interval exercise training in COPD.

Dr Samantha Walker is Director of Research + Innovation (R+I) at Asthma + Lung UK. She leads the R+I Directorate who work towards (1) advancing knowledge about asthma, COPD and other lung conditions through direct grant funding based on unmet patient need; (2) securing more R+I funding at a national and international level to bridge the substantial gap between the burden of respiratory disease and current R+I investment and (3) giving a voice to people who live with a lung condition to drive research that is most relevant and most beneficial to them.

Professor Sarah Walmsley is a Professor of Respiratory Medicine, University of Edinburgh, Honorary Consultant Physician, NHS Lothian, Dean of Research and Director of the Edinburgh Clinical Academic Training Scheme. She undertook her medical training at the University of Edinburgh graduating in 1997, and an MRC training fellowship at the University of Cambridge with award of a PhD in 2004. Her specialist training in Respiratory Medicine was in Sheffield, where she also held a Wellcome Intermediate Fellowship, prior to a move to Edinburgh as a Wellcome Senior Clinical Fellow. She is currently based in the Centre for Inflammation Research in the Institute for Regeneration and Repair in Edinburgh. Her research is focused on understanding the mechanisms of innate immune-mediated lung damage.

Dr Ran Wang, BSc, MBChB, MRes, PhD, MRCP(UK), is a National Institute for Health and Care Research (NIHR) Clinical Lecturer in Respiratory Medicine at the University of Manchester and Manchester NHS

Foundation Trust. Her clinical-translational research aims to improve how asthma is diagnosed and treated, with an emphasis on home-based diagnostic care pathways and precision phenotyping. Dr Wang holds an NIHR Research for Patient Benefit (RfPB) award investigating home asthma diagnostic strategies and support from Asthma + Lung UK to advance novel diagnostic and phenotyping approaches. Her broader interests include applied clinical research, biomedical big data, circadian biology, static modelling and lung physiology.

Dr 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 4 years until June 2023 and is the current President-Elect for the Association of Respiratory Technology and Physiology (ARTP). 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.

Dr Katie Ward is a consultant at the Royal Free Hospital with a special interest in multisystem diseases affecting the lung. She is the respiratory lead for the national scleroderma unit at the Free and involved in both clinical work and research. She is the respiratory consultant for the regional vasculitis and sarcoid unit for North Central London. Prior to working at the Free she was a consultant at Imperial College Healthcare NHS trust, working within vasculitis, renal and rheumatology multidisciplinary teams. She is an honorary senior lecturer in Imperial College London School of Medicine.

Dr Emily Watts is a respiratory physician and Wellcome Trust Clinical Research Career development fellow based in the centre for inflammation research at the University of Edinburgh. Her research interests are in understanding the molecular mechanisms which underpin neutrophilic inflammatory lung disease. Using air pollution as a model of inflammation her fellowship is focused on how neutrophils sense and respond to inflammatory and metabolic cues in the environment and how this drives neutrophil synthetic and inflammatory capacity.

Dr Abi Whitehouse is a paediatric respiratory consultant and clinical academic at Queen Mary University of London and Royal London, where she leads the children's environmental health service. Her research focuses on asthma, air pollution, damp and mould exposure, biomarkers. Dr Whitehouse is passionate about medical education, health equity, and improving outcomes for children with respiratory conditions through evidence-based policy, service evaluation, and implementation research. She regularly contributes to professional development and public engagement in paediatric respiratory medicine.

Professor Tom Wilkinson is Professor of Respiratory Medicine at the University of Southampton, Faculty of Medicine, and Honorary Consultant at University Hospitals Southampton. He trained at the University of Cambridge and the Royal London School of Medicine and completed his PhD at UCL studying disease mechanisms driving exacerbations of COPD.He is lead of the Southampton COPD research group, the Respiratory Theme of the NIHR Southampton Biomedical Research Centre and Medical Director of HQIP. His research seeks to improve outcomes for patients with chronic lung disease. He has published over 200 peer reviewed papers and reviews in this field. He is founder and Chairman of my mhealth limited a global leader in digital therapeutics. In 2023 he was appointed as an NIHR Senior Investigator in recognition of his national contribution to respiratory medicine.

EXHIBITORS' INFORMATION

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.

Contact Details

Email: info@actionpf.org
Website: https://www.actionpf.org/

Aerogen **Stand: 30**

Headquartered in Galway, Ireland, Aerogen is the world leader in acute care aerosol drug delivery. With over 25 years of experience, 300 international patents and associated with over 300 clinical papers and publications, Aerogen technology has been used to treat 30 million patients in 80 countries worldwide. Based on pioneering vibrating mesh technology, the Aerogen Solo vibrating mesh nebuliser has become the gold standard for respiratory patient care across the hospital from the emergency department to intensive care. The company continues to leverage its expertise, research, technologies and partnerships to make the greatest possible difference to patient outcomes.

Contact Details

Email: info@aerogen.com
Website: www.aerogen.com

APR Medtech **Stand: 29**

The Association for Respiratory Technology & Physiology (ARTP) **Stand: J**

The Association for Respiratory Technology & Physiology (ARTP) is 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.

Contact Details

Email: admin@artp.org.uk
Website: 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 'Physiotherapy 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.

Contact Details:

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.
Contact Details:
Email: info@arns.co.uk
Website: https://arns.co.uk/

Asthma + Lung UK **Stand: O**

Asthma + Lung UK is leading the fight for healthy lungs by funding groundbreaking research that transforms how lung conditions are prevented, diagnosed and treated. With the support of our community, we invest in vital research across a wide range of lung diseases, driving breathing breakthroughs that revolutionise treatments and save lives. We are also proud to support the Lung Research Grand Challenges, three bold and ambitious goals designed to catalyse cross-disease thinking and cross-disciplinary collaboration to tackle the greatest challenges in lung health.
Contact Details:
Email: R esearch@asthmaandlung.org.uk
Website: https://www.asthmaandlung.org.uk/

AstraZeneca **Stands: 1 & 4**

AstraZeneca is a global, science-led biopharmaceutical company that focuses on the discovery, development, and commercialisation of prescription medicines in Oncology, Rare Diseases, and BioPharmaceuticals, including Cardiovascular, Renal & Metabolism, and Respiratory & Immunology. AstraZeneca operates in over 100 countries and its medicines are used by millions of patients worldwide.
With a proud 100-year heritage in advancing UK science, today AstraZeneca is the UK's leading biopharmaceutical company. The company is based in five different locations across the UK, with its global headquarters in Cambridge.
For more information, please visit www.astrazeneca.co.uk and follow us on X @AstraZenecaUK

Boehringer Ingelheim **Stand: 9,10,11 & 12**

The British Association for Lung Research (BALR) **Stands: 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).
Contact Details
Email: admin@balr.co.uk
Website: https://www.balr.co.uk

The British Society for Allergy and Clinical Immunology **Stands: N**

The British Society for Allergy & Clinical Immunology (BSACI) is the UK's leading allergy society for healthcare professionals caring for patients with allergy. Our aim is to improve the management of allergies and related diseases of the immune system in the UK, through education, training and research.
Contact Details
Email: info@bsaci.org
Website: www.bsaci.org

British Thoracic Society **Stand: A**

The British Thoracic Society (BTS) is the leading professional society for respiratory medicine in the UK and represents over 4,900 members. BTS supports respiratory professionals to deliver the best-quality healthcare to people with respiratory diseases.
The Society's vision is 'Better Lung Health for all' and BTS aims:

- To influence the provision of the optimum respiratory workforce and research-centred services that promote sustainable solutions and reduce health inequalities
- To educate professionals to advance knowledge and share learning in the prevention, diagnosis and treatment of lung disease
- To support all members of the respiratory team to improve standards of care.

Each year BTS hosts two conferences, the Summer Meeting in June and the Winter Meeting in November. The Summer Meeting focuses on education, training developments and opportunities within respiratory health, while the Winter Meeting focuses on science and research. BTS conferences remain internationally renowned celebrations of respiratory medicine and healthcare, relevant to the UK and globally.
Contact Details
Email: bts@brit-thoracic.org.uk
Phone: +44 (0)207 831 8778
Website: www.brit-thoracic.org.uk

EXHIBITORS' INFORMATION

Celltrion Healthcare UK Ltd **Stand: 5**

Celltrion Healthcare, based in South Korea, is a globally recognised biopharmaceutical company. We specialise in developing biosimilars providing accessible treatment options for patients around the world.
For more information visit our website: https://www.celltrionhealthcare.co.uk

Chiesi **Stand: 2**

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.
Contact Details
Email: info.uk@chiesi.com
Website: https://www.chiesi.uk.com/

Creo Medical **Stand: 14**

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.
Contact Details
Email: customerservice@creomedical.com
Website: www.creomedical.com

Diagenics **Stand: 36**

Diagenics is a UK healthcare company specialising in innovative diagnostic solutions for respiratory and allergy care. We work closely with healthcare professionals across the NHS and private healthcare sector, providing high-quality products and support to help improve diagnosis, disease management and patient care.
Our portfolio includes the SUNVOU FeNO Analysers , an innovative solution for measuring fractional exhaled nitric oxide (FeNO) to support the assessment and management of airway inflammation, alongside our established range of specialist allergy diagnostic products.

Erbe Medical Ltd **Stand: 8**

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, Erbe continues to drive forward pioneering medical technologies that improve patient outcomes.
Contact Details
Email: sales@erbe-uk.com
Website: https://uk.erbe-med.com/uk-en/

Fannin UK **Stand: 18**

Fannin UK is a specialist MedTech company delivering innovative medical devices and clinically focused solutions to support healthcare professionals and improve patient care across the UK. Through our Medi-Globe brand, we offer a specialist portfolio of single-use devices for endoscopic procedures across gastroenterology and pulmonology. Working in partnership with healthcare professionals, we provide reliable, high-quality products designed to support safe and effective procedures, enhance clinical efficiency and contribute to positive patient outcomes.
Contact Details
Website: www.fannin.eu
Social media: https://www.linkedin.com/company/fanninuk/

Fisher & Paykel Healthcare **Stand: 24**

Fisher & Paykel Healthcare is a global designer and manufacturer of respiratory products for use in acute and chronic respiratory care, surgery, and treatment of obstructive sleep apnea (OSA).
Headquartered in New Zealand, we have for more than 50 years crafted humidification technologies to support individual respiratory needs across the healthcare spectrum.

F &P Healthcare and its more than 7000 employees have focused on developing innovative therapies that change clinical practice and help deliver a more sustainable healthcare service the world over. It has worked alongside healthcare professionals, knowing their clinical input is critical to success.
Today, our medical devices are used in the treatment of around 14 million patients in more than 120 countries each year, and the company remains unwavering in its drive to produce medical devices that improve the care, outcomes and quality of life of patients.
Contact Details
Email: UKSalesSupport@fphcare.co.uk
Website: https://www.fphcare.com/en-gb/

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 the decade, as a successful, growing company where people can thrive.
Website: United Kingdom | GSK

HealthNet Homecare **Stand: 17**

Idorsia Pharmaceuticals UK **Stand: 46**
The purpose of Idorsia is to challenge accepted medical paradigms, answering the questions that matter most.
Contact Details
Website: www.idorsia.uk

Insmed **Stand: 19, 21 & 22**
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.
We focus on the discovery and development of drug candidates for the treatment of metabolic diseases and endocrine disorders with unmet medical needs.
We are currently supporting patients with NTM & Bronchiectasis.
Insmed seeks to accelerate the discovery and development of potential therapies in new and uncharted areas. We approach our science through the lens of patient need. We take a collaborative, multidisciplinary approach to discovery, applying adaptable strategies to address complex challenges.

Inspire Medical Systems **Stand: 16**
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.
Contact Details
Email: info@inspiresleep.com
Website: www.inspiresleep.co.uk

Its Interventional **Stand: 7**
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 7 to find out more
Contact Details
Website: https://www.itsinterventional.com

Merck **Stand: 47**
Merck is a science-led organisation dedicated to delivering transformative medicines with the goal of making a meaningful difference in the lives of people affected by cancer. Our pioneering spirit drives us to help address this need by discovering and delivering innovative approaches to cancer care.
We are harnessing this spirit and our strength as a global pharmaceutical company to invest significant resources into our research and development, which is demonstrated through our rapidly evolving pipeline of potential new cancer treatments.

But we know we cannot do this alone and endeavour to accelerate innovation by partnering with some of the leading minds in the cancer community—from industry to academic institutions to leading cancer centres, to pursue integrated innovations that might have the potential to transform cancer care.
Our oncology research efforts aim to leverage our synergistic portfolio to tackle challenging tumour types in gastrointestinal, genitourinary, and thoracic cancers.
Contact Details
Learn more at https://www.merckgroup.com/uk-en

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.
Contact Details
Email: info@mesothelioma.uk.com
Website: www.mesothelioma.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.
Contact Details
Email: NRAPinbox@rcp.ac.uk
Website: https://rcp.ac.uk/improving-care/national-clinical-audits/the-national-respiratory-audit-programme-nrap/

NeilMed Pharmaceuticals **Stand: 6**

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.
Contact Details
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Website: www.neilmed.com

NIOX Group plc **Stand: 40**

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.
Contact Details
Email: sales.uk@niox.com
Website: https://www.niox.com/

NTM Patient Care UK and The NTM Network **Stand: M**

NTM Patient Care UK is the first charity in the UK to support people with NTM and their families. It is run by patients in close association with NTM Network UK who recognized the need for direct patient support; working together with NTM patients they developed NTM Patient Care UK in 2018. Our purpose is to improve the lives of people with NTM infection through information, education and patient forums. We host quarterly Zoom meetings attended by patients and health care providers and a guest speaker presents in their area of expertise. Patients attending can ask questions regarding the presentation as well as sending in general questions before the meeting. Zoom meetings are recorded and sent to all members.
Our website ntmpatientcare.uk lists our flyers and has links to patient resources, including the Patient's Guide to the NTM Standards of Care.

Olympus **Stand: 23**

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 solutions for Respiratory Endoscopy, constantly seeking to improve lung cancer outcomes. Olympus is committed to developing new technologies, products, services and solutions that comply with the toughest industry standards.

Orion Pharma (UK) Ltd **Stand: 15**
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)
Contact Details
Email: uk.medicalinformation@orionpharma.com
Website: https://www.orionpharma.co.uk

Pfizer Ltd **Stand: 27**
Website: www.pfizer.co.uk

Pharming **Stand: 41**
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.
Contact Details
Email: info@pharming.com
Website: https://www.pharming.com/

The Pulmonary Rehabilitation Services Accreditation (PRSAS) **Stand: C**
The Pulmonary Rehabilitation Services Accreditation (PRSAS), developed and delivered by the Royal College of Physicians, accredits pulmonary rehabilitation services across the UK and the Republic of Ireland. Since its launch in 2018, PRSAS has supported services in improving the quality, consistency, and accessibility of care for people with chronic respiratory conditions. Through a structured accreditation process, PRSAS promotes clinical excellence, supports continuous service improvement, and encourages patient-centred care. We work with both integrated respiratory services and standalone pulmonary rehabilitation services across NHS and private organisations.
Contact Details
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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.
Contact Details
For information about any of Rocket Medical's products please contact 01914196949 or homecaresupport@rocketmedical.com or www.rocketmedical.com. customerservices@rocketmedical.com

Sanofi **Stands: 25, 26, 31, 32, 33 & 34**
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.

EXHIBITORS' INFORMATION

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

Contact Details
Email: info@sarcoidosisuk.org
Website: https://www.sarcoidosisuk.org

Stirling Anglian Pharmaceuticals **Stand: 39**

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.
Contact Details
Website: www.stirlinganglianpharmaceuticals.com

Tidal **Stand: 28**

Vitalograph **Stand: 44**

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.
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Early Career Investigator Awards

T1 INVESTIGATING THE INFLAMMATORY PROFILE OF EXACERBATIONS IN PATIENTS WITH SEVERE ASTHMA RECEIVING TEZEPELUMAB: THE PHASE IV TEZEX STUDY

[1,2]F Haris, [2]F Cefaloni, [1,2]J Gates, [2]P Khooshemehri, [1]AD Rosario, [1]AP Valles, [1]E Woods, [2]V Kanabar, [2]E Scourfield, [1]G d'Ancona, [1]J Dhariwal, [1,2]AM Nanzer, [1,2]DJ Jackson. *[1]Guy's Severe Asthma Centre, Guy's & St Thomas' NHS Trust, London, UK, London, UK; [2]School of Immunology & Microbial Sciences, King's College London, UK, London, UK*

10.1136/thorax-2026-BTSabstracts.1

Introduction Tezepelumab, an anti-TSLP monoclonal antibody, provides broad suppression of type 2 inflammation; however, a proportion of patients with severe asthma (SA) continue to experience exacerbations. The mechanisms of these exacerbations remain poorly understood.

Aim The phase IV TezEx study aimed to characterise the triggers and inflammatory profile of asthma exacerbations occurring on tezepelumab.

Methods TezEx is a prospective open-label cohort study which recruited participants initiated on tezepelumab for SA in the UK. Clinical characteristics, T2 biomarkers and sputum inflammatory cell profiles were assessed at exacerbation and compared to stable state. Exacerbations were classified as T2-low (FeNO<25ppb and BEC<0.3×10^9/L) or T2-high (FeNO≥25 ppb and/or BEC≥0.3×10^9/L). The ability of BEC and FeNO to predict exacerbations associated with sputum eosinophilia was assessed.

Results Of the 147 participants recruited, 74 (50%) experienced at least one AE by 1 year, with an overall annualised AE rate of 0.78. 47 AE events in 39 patients were captured and analysed. Compared to the stable state tezepelumab visit, the AE visit was associated with significantly higher mean (SD) ACQ-6: 2.9 (1.1) vs 1.5 (1.2), p<0.0001; lower FEV1: 1.99L (0.59) vs 2.34L (0.71), p<0.0001; higher median (IQR) FeNO: 40 (27–58)ppb vs 32 (15–42)ppb, p=0.0001 and higher median (IQR) sputum eosinophil counts: 7.5% (0.6–15) vs 1.0% (0.8–8.4), p=0.019. The blood eosinophil count was unchanged. Microbial pathogens were identified in 29/47 (62%) AE, with the majority (21/29) respiratory viruses.

Using pre-defined T2 biomarker thresholds, 39/47 (83%) AE could be considered T2 high and 54% had a sputum eosinophil count ≥3%. BEC at AE discriminated sputum eosinophilia with an AUC of 0.885 (95% CI 0.760–1.000; p<0.001); a threshold of BEC≥300 cells/μL had a positive predictive value of 87.5% of identifying sputum eos ≥3%. FeNO showed no discriminatory value for sputum eosinophilia (AUC 0.50), and incorporation of FeNO into a combined predictive model did not improve performance beyond BEC alone (AUC 0.88 vs 0.88).

Conclusion In patients established on tezepelumab, asthma exacerbations are associated with increased T2 biomarkers and airway eosinophilia in the majority of episodes. BEC at exacerbation is highly predictive of an eosinophilic exacerbation.

T2 ACQUIRED MONOCYTE REPROGRAMMING DRIVES DIVERGENT RESPONSES TO MICROBIAL STIMULI IN IDIOPATHIC PULMONARY FIBROSIS

L Pearmain, F Hunter, S Murtuza Baker, E Jokl, K Piper Hanley, ER Mann. *University of Manchester, Manchester, UK*

10.1136/thorax-2026-BTSabstracts.2

Introduction IPF exacerbations accelerate disease progression and mortality. Respiratory infections often trigger exacerbations, which are thought to result from a dysregulated host response to pathogens. However, the immune cells and pathways driving these dysregulated responses are incompletely understood. Monocytes coordinate innate and adaptive immunity and are recruited to the injured lung. We therefore investigated their role in host microbial responses.

Methods Healthy controls (n=20) and treatment-naïve IPF patients (n=70) were recruited at diagnosis. Monocyte responses to bacterial (LPS) and viral (ssRNA40/TLR8) stimuli were assessed using functional stimulation assays. Deep immune profiling (70- parameter), monocyte RNA-seq/ATAC-seq and spatial transcriptomics (n=4) defined monocyte states, tissue localisation and clinical associations in IPF.

Results UMAP cluster analysis of blood monocytes revealed a higher proportion of IPF cells differentiating into monocyte derived dendritic-like cells (mono-DCs) in response to bacterial stimuli (LPS). Further, there was an elevated proportion of monocytes differentiating into monocytic myeloid-derived suppressor cells (M-MDSCs) in response to ssRNA. These populations expressed opposing inflammatory, antigen-presentation and proliferative markers. Divergent fates were unique to IPF patients, with both mono-DCs and M-MDSCs rare in healthy controls.

Phospho-flow demonstrated chronic NF-kB activation in fibrotic monocytes. However, inflammatory responses were attenuated following stimulation, indicating monocyte reprogramming. ATAC-seq/RNA-seq supported regulatory monocyte rewiring with preserved IRF but attenuated NF-kB transcriptional responses to ssRNA.

Serum conditioning of NF-kB/IRF dual monocyte reporter cells showed this phenotype can be re-capitulated by progressive, not stable fibrotic serum. Further, *ex-vivo* stimulation of circulating H1N1 infected mouse monocytes with ssRNA expanded M-MDSCs. These results indicate this re-programming is peripheral and acquired in response to lung injury.

IL-1βhiHLA-DRlo monocytes were associated with 1-year mortality, whilst effector memory T- and B-cell populations were depleted in progressive IPF blood. Spatial transcriptomics identified IL-1βhiHLA-DRlo monocytes within AT2 niches and organised lymphoid structures enriched for regulatory dendritic, T- and B-cell populations, positioning reprogrammed monocytes to coordinate host microbial responses and adaptive immune remodelling.

Discussion Together, these findings support a model in which acquired peripheral monocyte reprogramming alters microbial stimulus interpretation, driving maladaptive immune remodelling and explaining both exacerbation risk and progressive fibrosis in IPF.

T3 FEASIBILITY AND EXTERNAL VALIDATION OF THE RED SCORE IN CLINICAL PRACTICE: A MULTICENTRE SERVICE EVALUATION

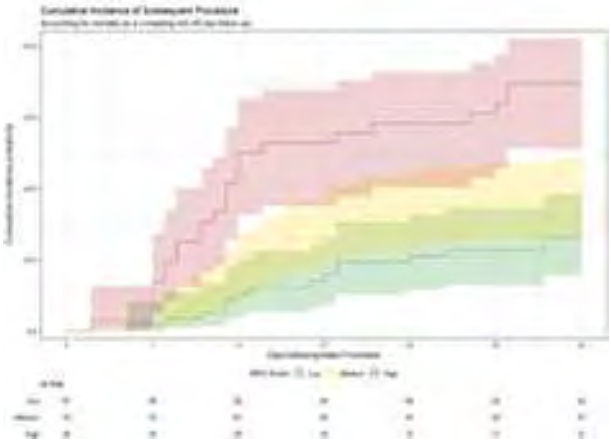
[1]DK Sethi, [2]A Sinha, [3]K Warner, [4]A Aujayeb, [2]M Selvan, [5]S Kuckreja, [6]J Liang, [6]J Zhang, [6]D Jajbhay, [7]J LeCrette, [7]G Tinson, [8]A Kwok, [9]A Bull, [5]R Peries, [3]A Elsheikh, [10]A Clarke, [11]R Bhatnagar, [3]N Rahman, [1]EK Mishra. *[1]Norfolk and Norwich University Hospital, Norwich, UK; [2]East and North Hertfordshire NHS Trust, Stevenage, UK; [3]University of Oxford, Oxford, UK; [4]Northumbria Healthcare NHS Foundation Trust, Newcastle, UK; [5]Princess Alexandra Hospital, Harlow, UK; [6]Guys and St Thomas NHS Trust, London, UK; [7]Bedfordshire University Hospital, Bedford, UK; [8]Liverpool Foundation Trust, Liverpool, UK; [9]Wirral University Teaching Hospital, Liverpool, UK; [10]University of East Anglia, Norwich, UK; [11]University of Bristol, Bristol, UK*

10.1136/thorax-2026-BTSabstracts.3

Introduction The RED score, calculated using respiratory rate, maximum effusion depth on thoracic ultrasound and Visual Analogue Scale for Dyspnoea, was derived in the REPEAT study and demonstrated shown to predict time to subsequent pleural procedures in malignant pleural effusions (MPE). We aimed to externally validate the RED score's predictive validity, real-world feasibility and the diagnostic utility of a high RED score to diagnose MPEs.

Methods This multi-centre service evaluation across nine UK hospitals (2025-2026) included consecutive adults attending pleural clinics for pleural procedures. We performed a competing risks survival analysis (42-day follow-up) to assess the primary endpoint of a subsequent ipsilateral procedure following a non-definitive, index pleural procedure. Feasibility was defined as the proportion of attendances where all three RED score components were successfully documented. The accuracy metrics of a high and low RED score to diagnose MPEs was calculated.

Results The validation cohort included 167 patients (median age 77, IQR 66-84, 62% male). Baseline RED scores were low in 34%, medium in 44%, and high in 22% of patients. Within 42-day follow-up, 69 patients required repeat procedure. A high RED score strongly predicted need for repeat intervention versus low score (sHR 4.22, 95% CI 2.27-7.80, figure 1). By day 14, 50% of high RED score patients required a repeat procedure, versus 9% in the low group. The RED score was successfully calculated in 275/299 (92.0%) presentations, demonstrating high feasibility in routine practice. An MPE was confirmed in 51% of the overall cohort. Using a high RED score as a positive test yielded an 85.3% specificity for MPE, while a low score had a negative predictive value of 81.4%.



Abstract T3 Figure 1 Cumulative incidence of subsequent pleural procedure stratified by baseline RED score. Curves demonstrate probability requiring a repeat pleural intervention within 42-day follow-up period following an index pleural aspiration (diagnostic or therapeutic). Shaded regions represent 95% confidence intervals. Accompanying risk table denotes number remaining at risk (alive and without a subsequent procedure) at 7-day intervals

Conclusion Calculating the RED score in routine practice is highly feasible. Our real-world data externally validates the RED score as a strong predictor of early repeat pleural interventions. A high and low RED score predict and exclude MPE respectively, though the significance of a medium RED score is uncertain. The RED score may help triage patients with undiagnosed effusions to identify those requiring urgent investigation and management.

T4 FENO RESPONSE TO BENRALIZUMAB IS DETERMINED BY BRONCHIAL EPITHELIAL PHENOTYPE

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10.1136/thorax-2026-BTSabstracts.4

Background Fractional exhaled nitric oxide (FeNO) is a type 2 (T2) biomarker thought to reflect IL-13 pathway activation in asthma. Its modulation by the anti-IL5Rα biologic benralizumab in severe eosinophilic asthma (SEA) is heterogeneous and poorly understood. We sought to characterise the bronchial epithelial determinants of FeNO response to benralizumab.

Methods Ten patients with SEA and FeNO >50ppb underwent bronchoscopic sampling before and four months post-benralizumab initiation. Inhaled corticosteroid (ICS) dose remained stable throughout. Patients were classified as FeNO non-responders if FeNO increased or remained >100ppb on treatment. Bulk RNA sequencing on bronchial brushings was analysed using differential gene expression, gene set variation analysis (GSVA), gene ontology enrichment, pathway analysis and cell deconvolution.

Results FeNO responders (n=5) demonstrated significant reductions in FeNO-related ($p=9\times10^{-5}$) and IL-13/T2 epithelial gene signatures (p=0.02) compared to non-responders (n=5). Despite similar baseline FeNO, non-responders showed higher *MUC5B* expression (p=0.002) with cell deconvolution inferring goblet cell and mast cell enrichment, suggesting persistence of mast cell-driven IL-13 signalling following eosinophil depletion. In contrast, neuroendocrine cell enrichment was observed in FeNO responders across both timepoints, supported by predicted *ASCL1*-driven transcriptional programmes, canonical pathway enrichment of neuroendocrine-associated GPCR/cAMP signalling, and acetylcholine and neuropeptide signalling signatures (p<0.05). Non-responders demonstrated significantly elevated interfer-

on-stimulated (p=0.006) and antiviral response (p=0.004) gene signatures by GSVA post-benralizumab, with upstream regulator analysis identifying persistent type I/II interferon, *TLR-MYD88* and NF-κB activation alongside predicted inhibition of corticosteroid-response signatures, suggesting an eosinophil-independent inflammatory programme not suppressed by concurrent ICS.

Conclusion FeNO response to benralizumab appears determined by pre-existing bronchial epithelial cell identity rather than eosinophil depletion in isolation. Our data suggest that a mast cell-dominant epithelium maintains IL-13 signalling independently of eosinophils whilst a neuroendocrine-enriched epithelium with active opioidergic and neuropeptide signalling associates with type 2 inflammation resolution. Non-responders additionally develop a corticosteroid-refractory and interferon-driven inflammatory programmes not present at baseline, identifying a potential therapeutic target population. Planned validation in bronchial biopsies will provide histological corroboration of these transcriptomic findings.

T5 PRECISION MEDICINE IN CHRONIC COUGH: PREDICTING LOW-DOSE MORPHINE RESPONDERS

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10.1136/thorax-2026-BTSabstracts.5

Background Low-dose morphine is highly effective in some chronic cough (CC) patients; others have no response. Identifying patients more likely to benefit may improve outcomes and stratify the patient's therapeutic journey.

Objective To identify clinical predictors of non-response to low-dose morphine in CC and develop a preliminary prediction model.

Methods Data from 111 CC patients (67 from NeuroCough project) treated with morphine were analysed, including 54 responders and 57 non-responders. Between-group comparisons assessed demographic and clinical differences. Multivariable logistic regression was used to identify independent predictors of non-response. Model performance was evaluated using discrimination, calibration, and clinical utility.

Results Never-smokers were more likely to respond than ex-smokers (71.7% [38/53] vs 27.6% [16/58]; $\chi^2 = 21.571$, $p < 0.001$). Responders reported higher Leicester Cough Questionnaire physical (mean 4.0 [SD 1.1] vs 3.6 [1.0]; $p = 0.039$) and social scores (3.2 [1.4] vs 2.7 [1.0]; $p = 0.014$). Cough duration > 15 years and Hull Airway Reflux Questionnaire item 6 (chest tightness) independently predicted non-response ($\chi^2 = 11.267$, $p = 0.004$). The final model showed good calibration (Hosmer–Lemeshow $\chi^2 = 12.727$, $p = 0.122$), moderate discrimination (area under curve 0.70), and 75.0% classification accuracy, with good specificity (77.8%) and negative predictive value (77.8%), indicating particular utility in identifying patients unlikely to benefit from morphine.

Conclusions Simple clinical features can identify a CC patient's likelihood of responding to morphine. These findings support a precision approach to morphine prescribing which may be more widely applicable in CC therapeutics. It provides a basis for prospective validation and refinement of predictive tools in routine care.

T6 INTEGRATED MULTI-OMIC AND FUNCTIONAL PROFILING OF IMMUNE RESPONSES IN PLEURAL INFECTION

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10.1136/thorax-2026-BTSabstracts.6

Introduction Pleural infections are characterised by hyperinflammation and poor clinical outcomes. Treatments include antibiotics and fibrinolytics. Development of novel host-directed therapies requires comprehensive understanding of the neutrophil-dominated inflammatory response. We performed a prospective multi-omic study integrating neutrophil functional assessment with inflammatory and microbiome profiling to investigate whether immune responses are dysregulated in pleural infection.

Methods Observational cohort study of pleural infection, non-infective effusion, and healthy controls. Pleural samples were obtained during clinically-indicated pleural procedures, with paired blood samples. Inflammatory markers were measured via immunoassays and OLINK target48 proteomic panel, and pleural microbiota profiled by 16S rRNA sequencing. Isolated blood neutrophils were assessed for NETosis, *Staphylococcus aureus* killing, and multi-colour flow cytometry. Neutrophil and blood leukocyte transcriptomes were analysed by RNAseq. Two independent UK cohorts were used for validation.

Results 100 participants were recruited (28 pleural infection: age 62.5±15.8[mean±SD], 57.2% male; 41 non-infective effusion, age 71±10.2, 43.9% male; 31 controls, age 65.2±15.2, 35.5% male). 148 participants with pleural infection were included in the validation cohorts (age 59.6±18.8, 64.8% male).

Key neutrophil markers DNA-NE (NETs), calprotectin, azuoricidin-1, plus myeloperoxidase, neutrophil elastase, and DPP-1 activity were increased in pleural fluid from infective vs non-infective effusions(all p≤0.001). Azurocidin-1 was significantly increased in those with organisms detected by 16S sequencing, and across two independent cohorts in patients requiring fibrinolytics, chest drain, or surgery. In serum and pleural fluid, cytokines including IL-17A/F, IL-1β, and OSM were increased in pleural infection.

Blood neutrophil surface markers of activation were altered in pleural infection including increased CD63 (degranulation) and ICAM-1 (reverse migration) and decreased CXCR2(all p≤0.01), which significantly correlated with pleural inflammatory markers.

Severe pleural infection defined by RAPID score had significantly decreased *S. aureus* killing by blood neutrophils and defective PMA-induced NETosis. Neutrophil and blood leukocyte transcriptomes were significantly different in pleural infection; ≥6103 DEGs were identified between patients with infection vs control or non-infective effusion. Differential pathways included NETs, metabolism, and antigen processing.

Conclusion Pleural infection is associated with altered local and systemic neutrophil responses and changes in transcriptome, with bacterial killing and NETs associated with clinical biomarkers and severity of disease, highlighting potential immunomodulatory therapeutic targets.

Empowering the patient to improve self-management

S1 ACCELEROMETRY-BASED BEHAVIOURAL PHENOTYPES AT FIVE MONTHS FOLLOWING HOSPITALISATION FOR COVID-19 AND THEIR ASSOCIATIONS WITH HEALTH OUTCOMES AND RECOVERY AT ONE YEAR

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10.1136/thorax-2026-BTSabstracts.7

Background Persistent functional impairment is common following COVID-19 hospitalisation, but the prognostic value of post-discharge sleep–activity behavioural patterns remains underexplored. We identified accelerometer-derived behavioural phenotypes using five months of data and examined their associations with health outcomes and recovery at one year.

Methods In the UK Post-Hospitalisation COVID-19 (PHOSP-COVID) study, accelerometry data were collected from adults at five months post-discharge using 14-day wrist-worn accelerometers, with health outcomes assessed at one year. Latent class mixture modelling incorporating simultaneous variable selection based on the Integrated Completed Likelihood criterion was applied to accelerometer-derived circadian rhythm indices, physical activity metrics, and sleep parameters to identify behavioural phenotypes. Linear regression examined associations between phenotype membership and Incremental Shuttle Walk Test (ISWT), EQ-5D-5L, Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT), Short Physical Performance Battery (SPPB), and Dyspnoea-12 (D-12). Logistic regression examined associations with patient-perceived recovery (PSQ) at one year. All regression models were adjusted for age, sex, ethnicity, WHO severity, deprivation, body mass index (BMI), comorbidity count, valid accelerometer days, and season of measurement.

Results A total of 1,588 participants with valid accelerometer data were included (mean age 59.5±12.1 years; 38% female). Four behavioural phenotypes were identified: Circadian-Disrupted (n=230), Circadian-Aligned Active (n=498), Highly-Active Circadian-Robust (n=430), and Sedentary Inactive (n=430). The Highly-Active Circadian-Robust phenotype served as the reference group. The Circadian-Disrupted phenotype was associated with worst ISWT (β=−129m, [-206 to -52]), EQ-5D-5L (β=−0.107, [-0.167 to -0.047]), FACIT (β=6.67, 3.59 to 9.75), SPPB (β=−1.03, [-1.55 to -0.51]), and D-12 (β=3.30, 1.22 to 5.37) compared to the Highly Active Circadian-Robust phenotype (all p≤0.001). The Sedentary Inactive phenotype showed the worst PSQ recovery (Odds ratio=0.40, 0.23-0.70, p=0.001) and was associated with worse SPPB (β=−0.61, [-1.02 to -0.19], p=0.004), FACIT (β=4.87, 2.39 to 7.34, p<0.001), and D-12 (β=2.38, 0.72 to 4.04, p=0.005) (figure 1).

Conclusion Following COVID-19 hospitalisation, distinct behavioural phenotypes were independently associated with differential health outcomes at one year. The Circadian-Disrupted phenotype was consistently associated with the worst health and functional outcomes, apart patient-perceived recov-



Abstract S1 Figure 1 (a) Behavioural characteristics across four clusters, including demographics, circadian rhythm, physical activity and sleep features; (b) Associations between behavioural phenotypes and health outcomes and recovery at one year follow-up

ery which was most strongly associated with the Sedentary Inactive phenotype. These findings support early phenotype identification to guide targeted rehabilitation strategies.

S2 HOME-BASED BODY-WEIGHT EXERCISE WITH BLOOD FLOW RESTRICTION IN COPD: A RANDOMISED FEASIBILITY TRIAL

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10.1136/thorax-2026-BTSabstracts.8

Background Pulmonary rehabilitation improves outcomes in COPD, but access and completion remain suboptimal. Blood flow restriction (BFR) during exercise may enhance the benefits of low-intensity exercise, offering a potential home-based treatment.

Aim To evaluate the feasibility, safety, and acceptability of adding BFR to a home-based body-weight exercise programme in stable COPD.

Methods Adults with stable COPD were randomised 1:1 to a 6-week home-based functional body-weight exercise programme with or without BFR in a single-centre, parallel-group, randomised feasibility trial. Two supervised familiarisation sessions preceded the home phase. Primary outcomes were recruitment, retention, adherence, safety and acceptability. Secondary outcomes included cardiopulmonary exercise testing, short physical performance battery (SPPB), quadriceps strength and health-related quality of life.

Results Of 128 eligible patients, 26 consented (20.3%, 95%CI 14.3–28.1%) and 25 were randomised. Retention at 6 weeks was 20 of 25 participants (80.0%, 95%CI 60.9–91.1%). Among 24 participants who commenced training, 18 completed at least 14 of 18 sessions (75.0%, 95%CI 55.1–88.0%). Both groups reported high satisfaction. No BFR-related serious adverse events occurred. Adverse event rates were 10.4 and 8.9 per 100 sessions for BFR and control, respectively. Leg discomfort was higher with BFR, particularly at higher cuff pressures, while dyspnoea was similar between groups. Exploratory outcomes showed directionally gains in SPPB and isokinetic quadriceps torque with BFR.

Conclusions Home-based body-weight exercise with individualised BFR was feasible, acceptable and safe in stable COPD, supporting progression to a multicentre, adequately powered definitive trial.

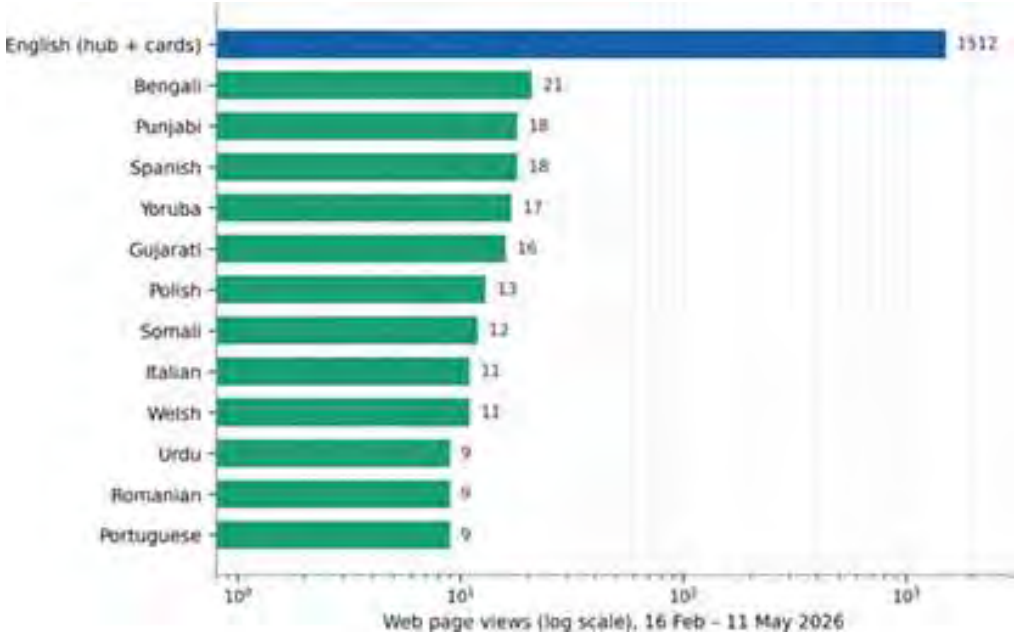
S3 CO-PRODUCING MULTILINGUAL BREATHLESSNESS SELF-MANAGEMENT RESOURCES WITH ETHNICALLY DIVERSE COMMUNITIES: EARLY REACH AND ACCEPTABILITY FOLLOWING NATIONAL DISSEMINATION

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10.1136/thorax-2026-BTSabstracts.9

Introduction and Objectives Breathlessness is among the most common reasons for emergency presentation, yet evidence-based self-management resources are overwhelmingly English-language and rarely designed for varying health literacy. Ethnically diverse communities and those facing health literacy barriers experience a disproportionate burden of severe breathlessness and avoidable emergency attendance but are seldom involved in designing the resources intended to support them. We aimed to co-produce multilingual, multimedia breathlessness self-management resources with ethnically diverse communities, and to examine their early reach and acceptability following national dissemination.

Methods Using a co-production approach, a diverse patient and public involvement (PPI) group, including faith and community network representatives, partnered with a specialist health literacy organisation to adapt evidence-based materials. Resources covering four self-management techniques (controlled breathing, handheld fan use, positioning, and relaxation) were pro-



Abstract S3 Figure 1

duced as short mobile-friendly videos and illustrated guides in 13 languages. Offline distribution routes were embedded for those with limited digital access. Reach was measured through web and video analytics; acceptability was assessed through structured PPI feedback.

Results Over an initial three-month period (February to May 2026), resources received 1,854 web page views from 1,154 users and approximately 640 video views. Uptake was concentrated in English (around 80% of views), with minority-language versions accessed at substantially lower volumes during this early single-site phase (figure 1). PPI contributors rated the resources as clear, culturally relevant, and trustworthy, identifying cultural resonance and plain language, alongside accurate translation, as essential to acceptability. The resources are now hosted across national patient-facing platforms, including a major respiratory charity, extending reach from local to national.

Conclusions Co-production with patients, faith groups, and community organisations is feasible and valued, and produces breathlessness resources judged acceptable by ethnically diverse communities. Early reach data show that passive digital availability alone does not ensure equitable uptake across languages: active embedding within trusted community and national channels is required. This work offers a replicable, equity-focused model for accessible self-management support in respiratory and palliative care.

S4 WHY DON'T MORE PATIENTS MANAGE THEIR OWN INDWELLING PLEURAL CATHETER (IPC)? BARRIERS AND FACILITATORS TO IPC SELF-MANAGEMENT

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10.1136/thorax-2026-BTSabstracts.10

Introduction and Objectives Indwelling pleural catheters (IPCs) are commonly used in the management of recurrent pleural effusions, permitting repeat drainage in a community setting. Current guidelines advocate patient-led drainage to promote independence and self-management, which may both improve quality of life and reduce healthcare utilisation. Despite this, IPC drainage in the UK is predominantly performed by community nurses (CN). This study aimed to identify the barriers, facilitators, and motivators of IPC self-management among patients and family/unpaid carers, and to explore healthcare professional (HCP) attitudes and practices relating to self-management.

Methods Semi-structured interviews were conducted with four groups: healthcare professionals (HCP) involved in IPC services (n=12), CN experienced in IPC management (n=11), and patients (n=17) and family/unpaid carers (n=12) with lived experience of IPC. Thematic analysis was conducted.

Results Twenty-one barriers and facilitators were identified and grouped into four domains: 1) affective, 2) physical/health-related, 3) healthcare system, and 4) social factors.

Key patient and carer barriers included valuing and wishing to retain community nursing support, lack of confidence in IPC management, fear of infection, squeamishness, and IPC as a reminder of underlying disease. Additional barriers included frailty, older age, task difficulty, and limited social support.

System-level barriers included absence of a self-management culture within community services, lack of supporting policies or training frameworks, limited IPC expertise in community services, and reliance on secondary care to initiate or 'grant permission' for self-management. Secondary care barriers included absence of self-management culture, and concerns regarding its teaching, management and infection risk.

Facilitators included positive patient engagement with their IPC, availability of supportive carers, early discussion of self-management within the IPC pathway, and clear communication with community services. Patients were additionally motivated by increased autonomy, flexibility in drainage sched-



Abstract S4 Figure 1

uling, ability to travel, and a desire to reduce pressures on NHS services.

Conclusions For the right patient, IPC self-management may provide substantial benefits, including greater autonomy and improved quality of life. Many of the identified barriers and facilitators appear modifiable/adoptable, highlighting opportunities for targeted service development, education, and pathway redesign to increase access to supported self-management.

S5 IMPLEMENTATION OF A SELF-REFERRAL CHEST X-RAY PATHWAY FOR CANCER DETECTION: EARLY OUTCOMES FROM A PILOT PROGRAMME

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10.1136/thorax-2026-BTSabstracts.11

Introduction and Objectives Delayed presentation and barriers to accessing primary care contribute to late-stage lung cancer diagnosis. Self-referral chest X-ray (CXR) services, established elsewhere in the country have shown promising results. We evaluated the implementation of a self-referral CXR pathway embedded within a Faster Diagnosis (FD) service, assessing uptake, safety and diagnostic yield.

Methods A prospective service evaluation was conducted from March 2025 to May 2026. Patients aged ≥40 meeting NICE NG12 symptom criteria self-referred via a digital platform.

Referrals were triaged by FD nurses to ensure IR(ME)R compliance, including exclusion of recent imaging. CXRs were requested under delegated authority, with:

- direct escalation to cancer pathways for suspicious findings
- GP-led management for non-cancerous significant findings
- discharge with safety-netting for benign results

Outcomes included referral volume, imaging completion, and cancer detection rate.

Results A total of 464 people accessed the service since its inception with 297 patients going on to self-request a CXR and 96.3% meeting criteria to have a CXR on triage with more than 98% attendance rate. Mean age was 57.4 years with about 45% being below the age of 55. 276 CXRs have already been delivered to patients in this service with more orders still pending. 1.45% were diagnosed with lung cancer which is in keeping with the national low-dose CT lung cancer screening conversion rate. 7.25% patients were found to have non-cancerous significant findings requiring further input. Most CXRs were benign, consistent with published data and secondary care input was not necessary.

Conclusions Cancer detection rates are comparable to established services, suggesting effective targeting of symptomatic individuals. Embedding triage within FD services enables robust governance and streamlined escalation while reducing burden on primary care. Follow-up audit is planned in future to see conversion rate in patients with initial normal CXR.

This model, therefore, is safe, feasible with high patient uptake rate and has potential to support improved patient pathways, warranting expansion and further evaluation. CXR remains a valuable tool currently and potentially for those below the national screening age, as reasonable means of providing reassurance about serious lung pathology.

S6 SUPPORTED 'RESCUE PACKS' POST-DISCHARGE FOR PEOPLE LIVING WITH COPD – QUALITATIVE INTERVIEW STUDY

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10.1136/thorax-2026-BTSabstracts.12

Introduction An ability to self-manage is considered an essential skill for a person living with COPD. One important aspect of self-management involves recognising and assessing symp-



Abstract S6 Figure 1

toms, including infections, and deciding if they can be managed at home. While clinical guidelines recommend patient-led self-management using rescue packs (antibiotics and steroids), access to these medications remains inconsistent in the UK, due to varying local prescribing policies and/or clinicians' concerns about medication misuse and abuse. Some evidence suggests that patient-initiated rescue packs to treat a COPD exacerbation, with clinical support, could decrease emergency admissions. It is important to understand the circumstances in which patients would be prepared to assume this responsibility.

Objective This qualitative study explored the perspectives of people living with COPD, who had been hospitalised with exacerbation, on being discharged with rescue packs and supported self-management plans (educational materials and automated telephone/text reminders), provided as part of the RAPID study (NIHR156698).

Method Semi-structured telephone interviews with 26 participants (17 women, 9 men) were conducted approximately three months post-discharge. Transcribed interviews were analysed thematically.

Findings Participants had positive experiences of being discharged with a rescue pack and supported self-management plan. Rescue packs were regarded as the most important element of the discharge intervention. Educational materials and automated reminders were seen as a potentially useful safety net, helping participants decide when to start a rescue pack. Participants robustly contextualised their views through concerns about access to primary care, understanding of their own condition, and perceptions of their responsibility to self-manage.

Conclusion This study demonstrated that tangible, practical support following discharge after COPD exacerbation is highly valued by people living with COPD. For most participants, the rescue pack was the key component of the intervention, but both educational materials and automated reminders had the potential to support the decision of whether to start a rescue pack and what to do next. This study indicates that patients are keen to be in charge of starting emergency medication when they recognise the start of an exacerbation and that they see it as enhancing their ability to self-manage their condition.

From RSV prevention to asthma care in childhood

S7 OPTIMISING THE ASTHMA DIAGNOSTIC PATHWAY IN CHILDREN AND YOUNG PEOPLE – PRELIMINARY RESULTS FROM THE RADICA STUDY

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10.1136/thorax-2026-BTSabstracts.13

Background All guidelines agree that objective evidence is required to support the diagnosis of asthma in children and young people (CYP). However, guidelines differ in tests included, test-order and thresholds used; these differences impact on the diagnostic accuracy. We aimed to establish the optimal diagnostic pathway for asthma in CYP.

Methods Symptomatic and untreated CYP (aged ≥5 and <16 years) with GP-suspected asthma underwent clinical history and physical examination. Fractional exhaled nitric oxide (FeNO), spirometry, bronchodilator reversibility (BDR), skin prick testing (SPT), bronchial challenge testing (BCT, methacholine and/or mannitol) and blood eosinophil counts (BEC) were performed over two visits, with two weeks of peak flow diurnal variability (PEFv) recorded between. All were given a trial of inhaled corticosteroids over 4-8 weeks before FeNO, spirometry, BCT and BEC were repeated. All clinical and test data were used to confirm or exclude asthma (reference standard) by an expert panel consisting at least two asthma specialists. The diagnostic efficiency of individual tests was evaluated at established cut off values; those with specificity of >90% were tested in sequential pathways.

Results Of 193 children, 131 children had a definitive diagnostic outcome (75% asthma); of these 101 completed all tests, allowing inclusion in pathway evaluations. BEC, SPT and PEFv demonstrated poor specificity (<90%) using established cut off values and so were excluded from optimal pathway analysis;

Abstract S7 Table 1 Selected asthma diagnostic pathways in children. Pathway designed as sequential rule in pathway: if Test 1 is positive, asthma is diagnosed; if negative, the pathway proceeds to the next test (Test 2). The algorithm stops once a test yields a positive result. The sequence of tests reflects clinical practicality, for example, although bronchial challenge testing is the most sensitive test, its limited availability places it at the end of all pathways

Pathways	Description	Test 1	Test 2	Test 3	Sensitivity	Specificity	PPV	NPV	% requiring BCT
Established cutoff used: FeNO ≥35ppb, **Spirometry:** airflow obstruction (AFO) defined as FEV_1/FVC< lower limit of normal; **BDR:** ≥12% change in FEV_1 or ≥10% FEV_1 % Predicted; **BCT:** PD_{20} of methacholine <0.2mg or mannitol challenge PD_{15} <635mg									
1	All tests available (2 visits)	FeNO	BDR	BCT	0.81 (0.70-0.90)	0.97 (0.83-1.00)	0.93 (0.91-1.00)	0.70 (0.54-0.83)	61.4%
2	No BCT available (single visit)	FeNO	BDR	-	0.54 (0.42-0.66)	0.97 (0.83-1.00)	0.97 (0.87-1.00)	0.48 (0.36-0.61)	NA
3	No BDR available (single visit)	FeNO	BCT	-	0.79 (0.67-0.88)	0.97 (0.83-1.00)	0.98 (0.90-1.00)	0.67 (0.51-0.80)	71.3%
4	No FeNO available (2 visits)	BDR	BCT	-	0.76 (0.64-0.85)	1.00 (0.89-1.00)	1.00 (0.93-1.00)	0.65 (0.50-0.79)	81.2%
5	Pathway split after baseline spirometry (single visit)	FeNO	Spirometry; If AFO – proceed with BDR; if no AFO, for BCT	-	0.77 (0.66-0.86)	0.97 (0.83-1.00)	0.98 (0.90-1.00)	0.65 (0.50-0.79)	59.4%
6	Pathway split after baseline spirometry (2 visits)	FeNO	Spirometry; If AFO – proceed with BDR; if no AFO, for BCT	If negative BDR- BCT	0.80 (0.69-0.89)	0.97 (0.83-1.00)	0.98 (0.91-1.00)	0.68 (0.52-0.81)	64.4%

the most effective single-visit diagnostic pathway combined FeNO (≥35 ppb) and BCT, achieved a sensitivity of 79% and specificity 97% (table 1, pathway 3). Although alternative pathways excluding FeNO or BDR achieved similarly high diagnostic accuracy, those without BCT demonstrated substantially lower sensitivities (54%) (table 1, pathway 2); where included, BCT was required in most children (59-81%, depending on the pathway used).

Conclusion We propose a new diagnostic pathway for asthma in CYP, using established cut-off values, allowing all tests to be performed in a single clinic visit, that warrants prospective validation. Inclusion of BCT improved pathway sensitivity; however, access to BCT remains limited, even in specialist centres. Reduced sensitivity in pathways excluding BCT highlights the need to improve access to bronchial challenge testing to ensure diagnostic accuracy in this population.

S8 EVALUATING THE CAUSAL EFFECT OF EARLY-LIFE PHENOTYPIC TRAITS ON LUNG FUNCTION TRAJECTORY: A MENDELIAN RANDOMISATION STUDY IN THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN (ALSPAC)

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10.1136/thorax-2026-BTSabstracts.14

Introduction and Objectives Low lung function trajectory between childhood and early adulthood is an important risk factor for COPD. Identifying which childhood phenotypes causally influence the risk of low trajectories could facilitate early disease prevention. In the first study to apply Mendelian randomisation (MR) to the aetiology of adverse lung function trajectories, we investigated whether previously associated childhood phenotypes are causally implicated.

Methods Outcomes were lower FEV_1 trajectory groups from latent profile analysis in participants with ≥2 measurements at ages 8, 15, 24 or 30 in the Avon Longitudinal Study of Parents and Children (% predicted; n=5,165).

Nine phenotypic exposures were selected based on prior associations: pre-term birth, birth weight, pre-school wheeze, childhood aeroallergen sensitisation, body mass index (BMI), eczema, allergic rhinitis, asthma and airways hyperresponsiveness. Exposures observationally associated with lower trajectories in logistic regression were prioritised for MR.

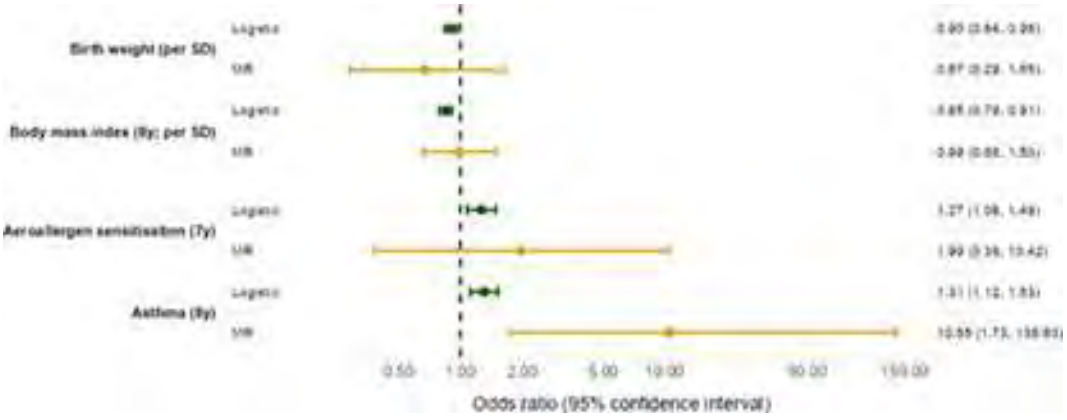
Two-stage residual inclusion MR, applying polygenic risk scores of variants from the largest published genome-wide association studies, estimated odds of lower trajectories by genetic liability to each exposure. Reverse MR investigated reverse causation from childhood FEV_1. Sensitivity analyses interrogated MR assumptions of instrument relevance, independence and exclusion restriction.

Results Of five trajectories identified, two had lower mean FEV_1: 'Below Average' (87.7% predicted) and 'Persistently Low' (72.6% predicted).

Observationally, higher birth weight and childhood BMI were associated with lower odds of these trajectories, and preschool wheeze, childhood aeroallergen sensitisation, asthma and airways hyperresponsiveness with higher odds.

In MR, greater genetic liability to childhood asthma increased odds of lower trajectories (OR 10.55 per SD, 95% CI 1.73, 136.6). MR estimates were directionally concordant with observational analyses but imprecise for birth weight (OR 0.79 per SD; 0.48, 1.31) and aeroallergen sensitisation (OR 1.90 per SD; 0.38, 10.42), and close to null for BMI (OR 1.00 per SD; 0.66, 1.50). In reverse MR, higher genetically predicted childhood FEV_1 was associated with lower BMI (β -0.67 per SD; -1.12, -0.23). There was little evidence of violation of MR assumptions.

Conclusions These findings support a causal role for childhood-onset asthma in high-risk lung function trajectories, suggesting that optimising its diagnosis and management may facilitate COPD prevention.



Abstract S8 Figure 1 Odds ratios by exposure for low lung function trajectories from logistic regression and two-stage residual inclusion Mendelian randomisation (MR)

S9 ASSOCIATION OF PRESCHOOL WHEEZE ENDOTYPES WITH WHEEZE ATTACKS AND ICS TREATMENT

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10.1136/thorax-2026-BTSabstracts.15

Introduction Preschool wheeze episodes are common and impair quality of life. Predicting and preventing attacks remains challenging. One *post-hoc* analysis found that blood eosinophil count (BEC) ≥300 cells/μL was associated with higher probability of response to inhaled corticosteroids (ICS).[1] However, children were recruited from specialist centres limiting generalizability to primary and secondary care. We hypothesised that BEC ≥300 cells/μL, alone or with allergic sensitisation and fractional exhaled nitric oxide (FeNO) ≥10 ppb, was associated with future wheeze attacks and reduced attack odds in children prescribed ICS.

Methods Pragmatic prospective nine-month observational study of 95 preschool children (12–59 months) with ≥1 clinician-confirmed wheeze attack in the preceding year, recruited from primary and secondary care. Baseline assessments included finger-prick BEC, skin prick testing, and tidal-breathing FeNO. The primary outcome was wheeze attacks requiring an unscheduled general practitioner (GP) or emergency department (ED) visit, documented by parental reports, medical records, or oral corticosteroid (OCS) prescriptions. Multivariable logistic regression assessed associations between biomarkers and attack odds. Exploratory analyses examined associations between ICS prescription and attack odds within biomarker-defined subgroups. Empirical Brown's Method combined p values from the three-follow-up methods (significance $p<0.01$).

Results BEC ≥300 cells/μL (n=60) was independently associated with increased wheeze attack odds at three (OR:7.26, 95%CI:2.33–27.36) and nine months (OR:4.27, 95%CI:1.70–11.38). Children with both BEC ≥300 cells/μL and FeNO ≥10 ppb (n=12) had significantly higher three-month attack odds (OR:60.74, 95%CI:2.98–1238.9). At nine months, polysensitised children with BEC ≥300 cells/μL had higher annualised attack frequency than monosensitised or non-sensitised children ($p<0.01$). ICS prescription was associated with reduced three-month wheeze attack odds in children with BEC ≥300 cells/μL (OR:0.11, 95%CI:0.02–0.49) and allergic sensitisation (OR:0.11, 95%CI:0.01–0.65), with the greatest reduction with both combined (OR:0.06, 95%CI:0.002–0.59). Associations were not sustained at nine months.

Conclusion BEC ≥300 cells/μL was associated with increased wheeze attack odds in preschool children, particularly when combined with elevated FeNO. ICS was associated with short-term attack odds reduction in children with BEC ≥300 cells/μL and allergic sensitisation alone and in combination. These findings provide support for biomarker-guided trials in preschool wheeze.

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S10 OUTCOME OF RECURRENT PRESCHOOL WHEEZE: DOES THE SEASON DURING FIRST WHEEZE EPISODE MATTER?

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10.1136/thorax-2026-BTSabstracts.16

Introduction Preschool wheeze (PSW) is heterogeneous, commonly classified as episodic (viral) or multiple-trigger wheeze, but these phenotypes are unstable and overlap (Makrinioti:ERJ:2024). We examined whether the season of a child's first wheeze episode relates to atopy and 12-month outcomes. No interventions were performed; treatment was directed by usual care clinicians.

Methods In a prospective observational cohort study conducted at a UK children's hospital, 100 children aged 1–5 years presenting with recurrent PSW were followed for 12 months, with reviews at 0, 4, 8 and 12 months. Children were grouped by season of first wheeze: spring/summer vs autumn/winter. We compared atopic features, baseline and follow up wheeze attacks, oral corticosteroid (OCS) courses, symptom control (TRACK) and caregiver quality of life (PACQLQ), using Mann–Whitney U and Fisher's exact tests.

Results Groups were comparable for age (median 40.5 [IQR 25–54.5] vs 35 [20.5–53] months; p=0.23), sex (70% vs 67% male; p=0.83), deprivation and baseline disease burden. First wheeze occurred in spring/summer in 40 children and autumn/winter in 60. Eczema was more common in autumn/winter onset children (28/60, 47%) than spring/summer-onset (10/40, 25%; p=0.036), a pattern consistent across autumn (48%) and winter (45%). Atopic features (allergy, hay fever, rhinitis) and baseline ICS use (58% versus 48%) did not differ. Prior year and follow up wheeze attacks were similar (follow up median 3 versus 3; p=0.71), as were unscheduled contacts (p=0.18), hospital admissions (≥1 admission over follow up 40% versus 43%; p=0.82), OCS courses (p=0.78), and the proportion becoming attack free (15% versus 21%; p=0.57). Symptom control and caregiver quality of life were similar at baseline and 12 months.

Conclusions The season of first wheeze episode did not predict attack frequency, healthcare use, symptom control or quality of life over 12 months. These findings underline the heterogeneity of preschool wheeze; future work should integrate objective biomarkers, such as blood eosinophil count, to refine phenotyping and identify children at risk of poor outcomes.

S11 REAL-WORLD OUTCOMES FOLLOWING MART INITIATION IN PAEDIATRIC ASTHMA: A SINGLE-CENTRE SERVICE EVALUATION

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10.1136/thorax-2026-BTSabstracts.17

Introduction Recent BTS/SIGN NICE guidelines recommend maintenance and reliever therapy (MART) to treat asthma.

Whilst there is trial evidence MART reduces asthma attacks, there is little real-world evidence demonstrating effectiveness in children. We have evaluated clinical outcomes and patient/carer experience following MART initiation in referrals to our paediatric respiratory service.

Methods Retrospective data were obtained from 114 consecutive children and young people (CYP) (median age 10 years, 5-17 years; 58% male) referred with poorly controlled asthma despite inhaled corticosteroids. Children were switched to MART in keeping with local regional PIER guidance (https://www.piernetwork.org/asthma-pathways.html). Oral corticosteroid use, emergency department (ED) attendances, asthma-related hospital and PICU admissions were compared for 12 months before and after starting MART. Parents/carers were invited to complete electronic feedback of their experience. Data were analysed using Wilcoxon matched-pairs signed-rank test. Age and deprivation score (based on postcode) were explored as predictors of response. The study was registered as a hospital service evaluation (SEV/0971).

Results 86% CYP used a Turbohaler for MART treatment. Oral corticosteroid use reduced by 41% (154 v 91; $p<0.0001$), ED attendances by 55% (97 v 44; $p<0.0001$), hospital admissions by 60% (30 v 12; $p<0.05$) and PICU admissions by 80% (5 v 1). Positive clinical outcomes occurred in all age groups and were unaffected by deprivation score.

64 parent/carers (56%) provided feedback. 91% reported improved asthma control after starting MART. 100% were confident about using MART with their chosen device. 95% felt confident recognising asthma attacks. 35 still had a salbutamol inhaler; 7 had never used it, 18 had it available for emergencies, 7 used it occasionally for exercise and 3 more regularly. Free-text comments consistently reported a positive experience of MART.

Conclusions Whilst regression to the mean and wider benefits of specialist services might have contributed to positive outcomes, our real-world data suggests switching to MART is associated with significant improvements in children with difficult-to-treat asthma. Some parent/carers with children who had experienced severe asthma attacks continued to have salbutamol available for emergency use, but the majority were able to manage asthma without needing short-acting beta agonists.

S12 SECOND SEASON CLESROVIMAB IN CHILDREN AT INCREASED RISK FOR SEVERE RSV DISEASE: RESULTS FROM THE PHASE 3 SMART TRIAL

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10.1136/thorax-2026-BTSabstracts.18

Introduction SMART (NCT04938830) was a phase 3 trial evaluating clesrovimab, a long-acting monoclonal antibody that binds site IV of the RSV F protein, among infants and children at increased risk of severe RSV disease. Here, we report second season data from SMART.

Methods Participants were randomized 1:1 to receive clesrovimab (105mg on day 1, placebo on day 28) or monthly palivizumab; eligible participants received open-label clesrovimab (210mg) in season 2. Tertiary objectives evaluated a range of RSV-associated disease endpoints through day 180 of season 2. RT-PCR on nasopharyngeal swabs and RSV F sequencing were performed.

Results In season 1,997 participants received clesrovimab or palivizumab, of whom 276 received clesrovimab in season 2. Baseline characteristics were balanced between groups in season 1; by design 99% of participants in season 2 had CHD or CLD. The safety profile of a second season 210mg dose was comparable between children receiving clesrovimab over two consecutive RSV seasons and those who had received palivizumab in their first RSV season. RSV-associated disease incidence rates in season 2 were also comparable by season 1 treatment group. There was an approximately even split of RSV-A and RSV-B infections in season 2, regardless of disease severity. Three participants had RSV-A infections with a variation in the clesrovimab binding site at position 446 (n=2 G446R, n=1 G446E), two had severe MALRI; both received palivizumab in season 1. G446E variants have known reduced clesrovimab susceptibility and reduced viral fitness; characterisation of G446R is ongoing.

Conclusion Clesrovimab was well-tolerated in infants at increased risk for severe RSV disease through two RSV seasons. These findings support the use of clesrovimab in children who remain at risk of severe RSV disease in their second RSV season.

Transforming pleural disease – from prediction to patient centred care

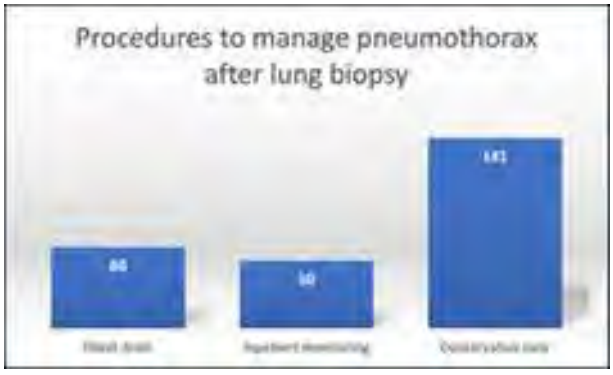
S13 PLUMBING LESS OR MORE. A MIXED-METHOD, MULTICENTRE PROSPECTIVE STUDY TO UNDERSTAND THE MANAGEMENT BASIS OF PNEUMOTHORAX AFTER LUNG BIOPSY IN THE UK (THE PLUMB STUDY)

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10.1136/thorax-2026-BTSabstracts.19

Background Lung biopsy is a key diagnostic test for early-stage lung cancer but pneumothorax complicates 1 in 4 biopsies with a reported 7% drainage rate. With no treatment guidelines, the

treatment of pneumothorax after lung biopsy remains poorly understood despite its direct impact on lung cancer pathways.
Aim To understand the treatment of pneumothorax after lung biopsy and the factors affecting treatment decision-making.
Methods A prospective, mixed-method study was conducted from Dec 2024 till Sep 2025 in 18 centres across the UK. Data was collected on patient demographics, pneumothorax characteristics, treatment and reasons for treatment decision. Semi-structured interviews were conducted with 10 physicians to understand decision-making in the treatment of pneumothorax after lung biopsy. Thematic analysis of transcribed interviews was undertaken to identify and analyse key themes in clinical decision-making.
Results 251 patients were recruited with a median age of 72 (IQR 65.5-77); 129 (51.4%) were females. 208 (82.8%) were current or ex-smokers. Pneumothorax drainage was performed in 60/251 (23.9%); 54/60 required chest drain insertion. 50/251 (20%) needed inpatient monitoring only and 141/251 (56.2%) were treated conservatively (figure 1). Size of pneumothorax was the commonest factor to guide treatment in both initial conservative (156/198;80%) and interventional care (40/53;75.4%). The median time to pneumothorax resolution was 3 days (IQR: 1-10). For 31 patients recruited at the main study site, a mean of 3.5 additional imaging procedures were performed to monitor pneumothorax after lung biopsy. Semi-structured interviews were performed with 8 consultants (3 radiologists, 5 lung cancer/pleural specialists) and 2 pleural fellows. Main themes guiding decision-making were size of pneumothorax with clinical features and logistic factors. Variability in practice and high disease burden emerged as important sub-themes.



Abstract S13 Figure 1

Conclusion PLUMB is the first mixed-method study to prospectively evaluate high intervention rate, size-based decision-making, imaging burden and variability in clinical practice in pneumothorax after lung biopsy. Further studies are needed to assess the impact of this condition on lung cancer diagnostic pathway.

S14 REAL-WORLD MANAGEMENT AND OUTCOMES OF SECONDARY SPONTANEOUS PNEUMOTHORAX: AN INTERNATIONAL MULTICENTRE RETROSPECTIVE ANALYSIS ON PERSISTENT AIR LEAK IN SPONTANEOUS PNEUMOTHORAX (IMPALE)

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10.1136/thorax-2026-BTSabstracts.20

Introduction Secondary spontaneous pneumothorax (SSP) carries a high risk of prolonged air-leak (PAL), recurrence and mortality. There are different approaches to PAL, with variable levels of evidence, and no consensus approach. IMPALE aimed to understand management strategies of PAL in SSP patients both fit and unfit for surgery, evaluate overall adherence to existing guidance and determine efficacy of non-surgical management techniques.
Methods IMPALE was a multicentre, international, retrospective study on patients with SSP with PAL for greater than 48 hours. Data was collected over a 12-month period at participating centres across Europe, Asia, North America and Australia, detailing demographics, interventions, complications, and surgical referral. Final outcomes included length of stay (LOS), time to PAL resolution, mortality and six-month recurrence.
Results IMPALE describes the largest known SSP dataset with 304 patients (75% male, median age 65 years; IQR 53-75, median weight 63.8kg; IQR 53-76). Underlying respiratory disease was present in 271 patients (89%), most frequently COPD(60.1%) followed by ILD (17.3%). Presentations were typically first episodes (n=227, 91.1%), and more often right sided (n=173, 56.9%).

Initial management was almost always intercostal drain insertion (n=296, 97%), with 33% of patients requiring more than one intervention. Non-surgical techniques employed alongside first intervention included thoracic suction (n=176,59.5%), sterile talc administration (n=55,18.6%), autologous blood patch (n=15,7.7%) and ambulatory/Heimlich devices (n=10,5.1%). 155 patients (51%) were referred for consideration of surgical intervention (median timeframe 6 days), with 62(40%) accepted for immediate transfer.

Median LOS was 15 days (IQR9-25) and median time to PAL resolution was 12 days (IQR6-21). Air-leak had resolved at the point of discharge in 234 patients (79%). In-hospital mortality was 12.2%. Recurrence within six months occurred in 11.4% of those with known follow up(n=267).
Conclusion Patients with SSP whose air-leak has not settled by 48 hours typically have prolonged admissions, long durations of air-leak, require more than one intervention and are at substantial risk of mortality. There is varied international practice regarding management, surgical referral and non-surgical techniques. These findings emphasise the comorbidity and risk associated with SSP, alongside the need for ongoing prospective studies to help define optimal management strategy and timing of interventions.

S15 THE RISK OF PNEUMOTHORAX IN CANNABIS SMOKERS: A CASE-CONTROL STUDY

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10.1136/thorax-2026-BTSabstracts.21

Introduction and Objectives Cannabis use is increasing globally, yet robust evidence characterising its respiratory risks remains limited. While cannabis smoking has been associated with paraseptal emphysema and pneumothorax, evidence is restricted to a handful of relatively small studies. We aimed to investigate the potential association between cannabis and pneumothorax and compare this to the effect of tobacco use. This is the first study of its kind in the UK and the world's largest cannabis-pneumothorax case-control study performed to date.

Methods A hospital-based case-control study recruited 450 adults aged 16–50 years attending a specialist pneumothorax service between 2016 and 2026. Participants were matched by sex and age to 450 controls recruited from unrelated outpatient clinics. Cannabis and tobacco smoking history, demographic characteristics and pneumothorax history were collected using an anonymous questionnaire developed in partnership with a patient and public involvement panel. Unconditional logistic regression was used to evaluate the association between cannabis smoking and spontaneous pneumothorax, adjusting for age, sex and smoking history. Odds ratios and 95% confidence intervals were estimated, and potential interactions between cannabis and tobacco exposure were explored.

Results We provide epidemiological data on cannabis use in UK outpatient populations: pneumothorax vs non-respiratory clinics. Among controls, cannabis exposure was reported by 25.5%, whilst 45.9% reported tobacco use. Self-reported cannabis smoking was reported across all ages and was 2.5-fold more prevalent in men. Detailed behavioural data were also collected to characterise the exposure pattern of cannabis smokers in the UK. We compare these data to patients presenting with spontaneous pneumothorax to determine the relative risks associated with cannabis vs tobacco use relative to never-smokers.

Conclusions This study addresses an important evidence gap in respiratory medicine, providing the first UK-specific estimate of pneumothorax risk associated with cannabis exposure. Given increasing cannabis use and the substantial healthcare burden associated with spontaneous pneumothorax, these findings have direct implications for national service provision and respiratory practice internationally.

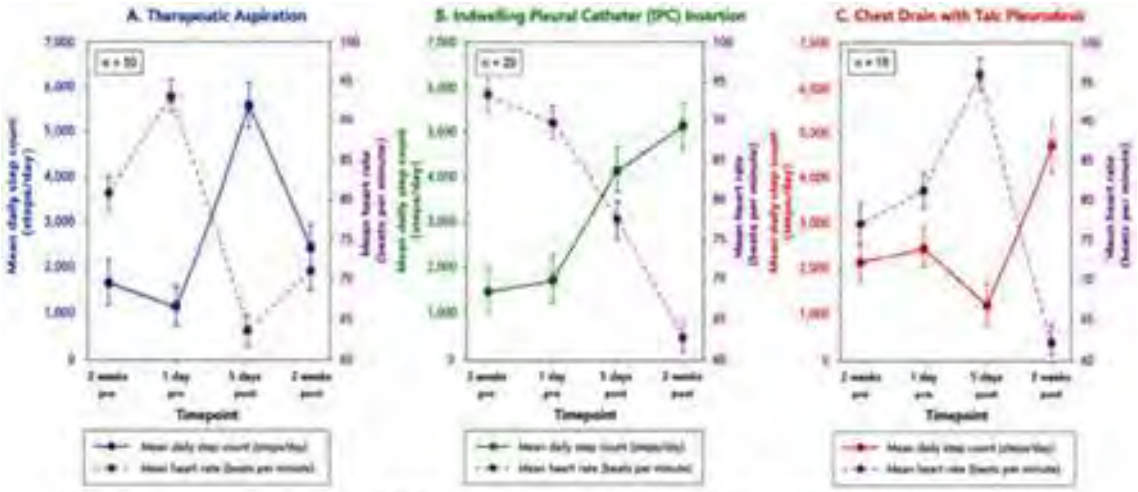
S16 WEARABLE-DERIVED ACTIVITY MONITORING REVEALS DISTINCT RECOVERY TRAJECTORIES FOLLOWING PLEURAL INTERVENTION IN MALIGNANT PLEURAL EFFUSION

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10.1136/thorax-2026-BTSabstracts.22

Background Malignant pleural effusion (MPE) is associated with breathlessness, reduced physical activity, and impaired quality of life. Current assessment of treatment response relies predominantly on intermittent clinical review and patient-reported outcome measures. Wearable devices offer continuous objective monitoring of functional status, but data describing their application in pleural disease are limited. We aimed to evaluate wearable-derived activity and physiological metrics before and after pleural intervention in patients with MPE.

Methods We conducted a mixed prospective and retrospective observational study of patients with MPE managed at a tertiary pleural centre. Patients using consumer wearable devic-



Mean daily step count (solid lines) and average heart rate (dashed lines) at two weeks prior to intervention, one day prior to intervention, five days post-procedure, and two weeks post-procedure following **(A)** therapeutic aspiration, **(B)** indwelling pleural catheter insertion, and **(C)** talc pleurodesis. Therapeutic aspiration produced rapid but transient improvement, IPC insertion was associated with sustained functional recovery, and talc pleurodesis demonstrated an initial decline followed by recovery.

Abstract S16 Figure 1 Wearable-derived activity and physiological trajectories following pleural intervention in malignant pleural effusion

es (Apple Watch, Fitbit or Garmin) consented to share activity and physiological data. Mean daily step count and average heart rate were analysed at four predefined timepoints: two weeks before intervention, one day before intervention, five days after intervention, and two weeks after intervention. Patients were grouped according to intervention: therapeutic aspiration (n=50), indwelling pleural catheter (IPC) insertion (n=20), or chest drain insertion with talc pleurodesis (n=15). Longitudinal changes were analysed using linear mixed-effects models.

Results A total of 85 patients were included. In the therapeutic aspiration cohort, mean daily step count declined prior to intervention before increasing from 1124 to 5139 steps/day at five days post-procedure ($p<0.001$), followed by a subsequent decline by two weeks. IPC insertion was associated with sustained improvement in activity, with mean daily step count increasing from 3254 steps/day pre-procedure to 5508 steps/day at two weeks ($p<0.001$). In contrast, patients undergoing talc pleurodesis demonstrated an initial reduction in activity following intervention, with recovery by two weeks (2400 to 4666 steps/day; $p<0.001$). Heart rate demonstrated reciprocal changes across all groups, improving following intervention (all $p<0.001$).

Conclusion Wearable-derived activity and physiological metrics capture distinct recovery trajectories following pleural intervention in MPE. Therapeutic aspiration produced rapid but transient improvement, IPC insertion was associated with sustained functional recovery, and talc pleurodesis demonstrated an initial procedural burden followed by recovery. These findings support the potential role of wearable technologies as objective, patient-centred outcome measures in pleural disease and future pleural intervention trials. Wearable technologies support NHS ambitions for personalised, digitally enabled care delivered closer to home.

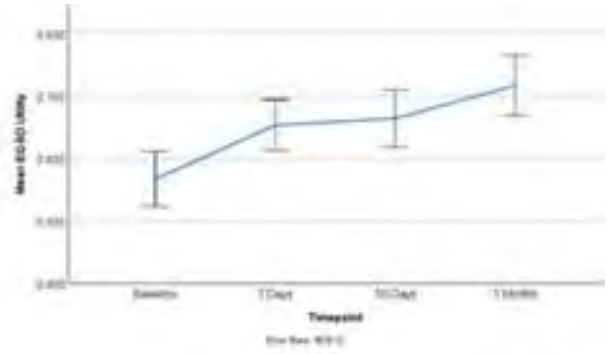
S17 LONGITUDINAL IMPROVEMENT IN HEALTH-RELATED QUALITY OF LIFE FOLLOWING PLEURAL INTERVENTION FOR MALIGNANT PLEURAL EFFUSION

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10.1136/thorax-2026-BTSabstracts.23

Introduction Malignant pleural effusions (MPE) have a substantial symptom burden, chiefly dyspnoea. Pleural interventions are increasingly evaluated for cost-effectiveness. However, utility-based health-related quality of life (HRQoL) data used for quality-adjusted life year (QALY) assessments by the National Institute for Health and Care Excellence are limited.[1]

Methods A secondary analysis of prospective multi-centre observational cohort data (REPEAT study) in patients with known or suspected MPE was performed; only confirmed MPE cases were included. HRQoL was assessed using the EuroQol 5-Dimension 5-Level (EQ-5D-5L) questionnaire at baseline, 7 days, 30 days and 3 months following therapeutic aspiration and subsequent pleural interventions. Utility values were derived from the UK EQ-5D-5L value set.[2] Baseline HRQoL was compared with UK population norms. Longitudinal utility change was analysed using linear mixed-effects modelling



Abstract S17 Figure 1 Longitudinal change in EQ-5D utility following pleural intervention

adjusted for age, performance status (PS), and additional pleural intervention, with survivors-only sensitivity analysis.

Results 210 patients were included. Mean age was 71.7 years. Baseline EQ-5D-5L utility was 0.566 (SD 0.326). Compared with age-matched UK population norms, this was significantly lower than subgroups 65-74 years (mean difference -0.243, $p<0.001$) and ≥ 75 years (mean difference -0.123, $p<0.001$). Usual activity and mobility were the most impaired EQ-5D domains with moderate-to-extreme problems reported in 61.0% and 65.2% respectively. Poor PS was an independent predictor of lower baseline utility ($\beta=-0.473$, $p<0.001$). Utility significantly improved over time ($p<0.001$), with the largest improvement by day 7 and sustained across follow-up, including in survivors-only sensitivity analysis. Mean end-of-life utility was 0.405 (SD 0.317).

Discussion Patients with MPE have marked baseline HRQoL impairment. Pleural intervention was associated with significant and sustained improvement in utility. These findings provide longitudinal EQ-5D-5L utility values for MPE that may inform future cost-effectiveness and QALY calculations for pleural interventions.

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S18 THE ASSOCIATION OF BASELINE PLATELET COUNT WITH CLINICAL OUTCOMES AND SEPTATION BURDEN IN PLEURAL INFECTION: ANALYSIS OF THE PILOT COHORT

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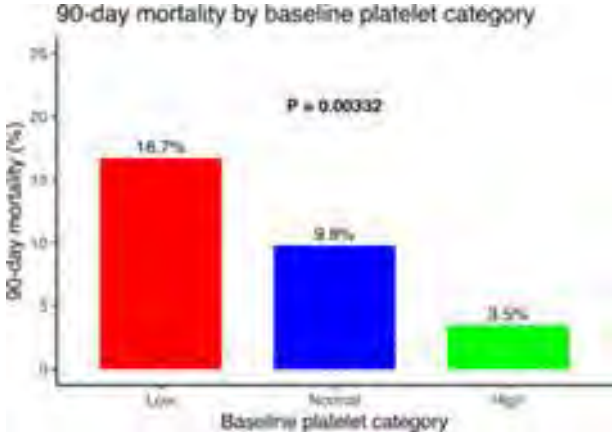
10.1136/thorax-2026-BTSabstracts.24

Introduction Platelets are increasingly recognised as mediators of inflammation and coagulation in infection. In pleural infection, septations and loculations reflect pleural organisation and fibrin deposition that may impair drainage. Although the RAPID score is widely used for risk stratification in pleural infection, platelet count was not evaluated during its derivation. The

relationship between baseline platelet count, clinical outcomes or septation burden in pleural infection therefore remains unclear. We investigated associations between baseline platelet count, 90-day mortality, need for surgery, length of stay and septation burden using data from the largest prospective pleural infection observational study in the world literature (PILOT) PILOT cohort.

Methods Patients enrolled in the PILOT study with available baseline platelet count and 90-day mortality (as the primary outcome of the PILOT study) were analysed. Platelet count was assessed as a continuous variable. Outcomes were 90-day mortality, need for surgery, length of stay, septation presence and septation severity. Logistic regression was used for binary outcomes, linear regression for length of stay and ordinal logistic regression for septation severity. Clinical outcome models were adjusted for RAPID variables. Septation models were adjusted for age, albumin and CRP.

Results Baseline platelet count was available for 510 patients. Median platelet count was 396.5 $\times 10^9$/L. 90-day mortality was 6.9% (35/510). Platelet count was lower in non-survivors than survivors (353.7 vs 445.3 $\times 10^9$/L, p<0.001). Higher baseline platelet count was independently associated with lower 90-day mortality, with each 100x109/L increase associated with a 24% reduction in odds of mortality (adjusted OR 0.76 95% CI 0.58-1.00; p=0.039). Platelet count was not associated with need for surgery, length of stay or septation presence. Higher platelet count was associated with increasing septation severity on adjusted ordinal analysis (p=0.02).



Abstract S18 Figure 1

Conclusion Baseline platelet count was independently associated with 90-day mortality in pleural infection after adjustment for RAPID variables. Lower platelet counts were observed in non-survivors, suggesting platelet count may reflect impaired systemic inflammatory or marrow response. Platelet count was not associated with septation presence, although higher platelet counts were associated with greater septation severity. Further studies should evaluate whether platelet count could contribute to risk stratification in pleural infection.

COPD in primary care

S19 'RESPIRATORY JUST ISN'T SEXY': CLINICIANS' EXPERIENCES OF SPIROMETRY FOR COPD DIAGNOSIS IN UK PRIMARY CARE

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10.1136/thorax-2026-BTSabstracts.25

Background Access to spirometry testing for COPD diagnosis in primary care varies across the UK, with some areas without access to spirometry at all. New delivery models such as Community Diagnostic Centres have been introduced to mitigate disparities in provision. However, there is little contemporary evidence regarding how spirometry services for COPD diagnosis are delivered and experienced.

Aims/Objectives To gain in-depth insights into clinicians' experiences of current COPD diagnostic pathways, barriers and facilitators to spirometry delivery, and views on potential diagnostic innovations to support COPD diagnosis in UK primary care.

Methods Semi-structured qualitative interviews were conducted with healthcare professionals involved in COPD diagnosis within and from UK primary care. Participants were purposively sampled from respondents to a national survey. Interviews took place October 2025 and January 2026 and were analysed thematically.

Results 28 participants were interviewed, predominantly nurses (n=17) and GPs (n=6) but including pharmacist, physiologist and hospital consultant representation. Participants described respiratory care as an area that *'just isn't sexy'*, resulting in de-prioritisation at an organisational level, inconsistent funding and skilled workforce shortages with insufficient interest in developing a special interest within primary care. Many viewed spirometry as technically complex, emphasising that *'spirometry is only as good as the person doing it'*, with concerns regarding performance, interpretation and quality assurance. Access to diagnosis was perceived to depend heavily on local expertise, creating marked geographical variation in service provision. While clinicians were receptive to diagnostic innovation, they emphasised any alternative would need to be *'fast enough to impress the GPs'* as well as accurate, acceptable to patients and capable of integrating into routine workflows.

Implications Clinicians described spirometry as a challenging diagnostic test for both patients and professionals, affecting access to timely COPD diagnosis. They highlighted the need not simply for additional funding for spirometry provision, but for better ways of delivering diagnostic services capable of identifying patients earlier in their disease journey, when intervention may have greater impact. Clinicians saw considerable potential for diagnostic innovation but emphasised that future technologies must be practical, acceptable to clinicians and patients, and capable of improving accessibility and efficiency without compromising diagnostic confidence.

S20 SPIROMETRY PROVISION FOR COPD DIAGNOSIS ACROSS THE UK: FINDINGS FROM A NATIONAL SURVEY OF HEALTHCARE PROFESSIONALS

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10.1136/thorax-2026-BTSabstracts.26

Background Early diagnosis and treatment of COPD can improve symptoms, reduce exacerbations and slow lung function decline. However, COPD remains substantially underdiagnosed, with almost one in four patients reporting over five years' wait before diagnosis. Spirometry is central to diagnosis, yet access is variable and diagnostic pathways have changed considerably, including new delivery models such as Community Diagnostic Centres (CDCs).
Aims/Objectives To map current UK pathways for diagnosing suspected COPD and identify barriers and facilitators in diagnostic service provision.
Methods A UK-wide online survey of health care professionals involved in COPD diagnosis was conducted between August and December 2025. Participants were recruited through professional networks and conferences.

Survey questions explored local diagnostic pathways and barriers and facilitators to spirometry delivery. Data were analysed descriptively.
Results 233 eligible responses were analysed. Respondents predominantly included nurses (n=101, 43%), GPs (n=73, 31%) and respiratory consultants (n=29, 12%).

Almost all respondents (n=209, 94%) believed there are currently delays to COPD diagnosis. Leading causes of delay include patients delaying assessment (n=196; 93%), presenting symptoms being treated without consideration of COPD (n194; 93%), long wait to see a specialist (n=173; 83%), and long waits for diagnostic testing (n=167; 81%).

Spirometry pathways varied considerably. Almost half of respondents reported spirometry being available in-house within GP practices (n=82, 49%), but other options included hubs, CDCs or secondary care services, all generally associated with longer waiting times. Some described very limited local spirometry provision and waiting times exceeding one year.

Over half of those no longer providing spirometry within GP practices had stopped during the COVID-19 pandemic and not restarted (n=34/60, 57%). The leading barriers to providing spirometry were inadequate funding/financial incentives (n=114; 80%); limited staff capacity (n=103; 60%); and high-level training requirements (n=96; 56%).
Implications Healthcare professionals perceived substantial delays in COPD diagnosis and described marked variation in access to spirometry across the UK. While inadequate funding was the most frequently identified barrier, workforce capacity, training requirements and the lasting impact of COVID-19-related service disruption were also important challenges. Improving access to high-quality COPD diagnosis will require service models that address these interconnected barriers rather than funding alone.

S21 STANDBY RESCUE PACK PRESCRIBING CREATES SUBSTANTIAL UNDER-RECORDING OF COPD EXACERBATIONS IN PRIMARY CARE ELECTRONIC HEALTH RECORDS

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10.1136/thorax-2026-BTSabstracts.27

Introduction Oral corticosteroids (OCS) and/or antibiotics are recommended by NICE for COPD self-management, but it may not be clear from coded electronic health records (EHR), whether a prescription was a standby rescue-pack or acute exacerbation (AECOPD) treatment.
Aim To characterise real-world coding and documentation of COPD rescue packs in a selection of general practices in England and assess their potential implications for clinical care and research.
Methods Retrospective manual EHR review (09.2024-09.2025) in a pilot general practice using Optum/EMIS (data collection in other practices is ongoing). All patients with COPD/AECOPD SNOMED code were included (n=270). OCS/antibiotic prescriptions were linked to consultations, correspondence and other notes. Courses issued ±1 day of an AECOPD were classified as acute treatment, all others as rescue-packs. Statistical analyses used Welch's t-test.
Results 115 patients had at least one OCS/antibiotic prescription for AECOPD in the previous year: 35% had rescue-packs and 65% did not. Rescue-pack-recipients and non-rescue-pack-recipients did not differ in age, sex or disease severity, but rescue-pack-recipients had longer COPD duration (9 vs 6 years, p=0.004). There was a record of patients reporting exacerbations in fewer than 1% of self-treated AECOPD. No rescue-packs were SNOMED-coded, leaving their prescribing absent from structured data. Rescue-pack-recipients received more than double the OCS/antibiotic courses per year than non-rescue-pack recipients (4.75 vs 1.81, difference 2.94; 95% CI 1.98-3.89; p<0.001). In the year preceding annual COPD review, prescribed OCS/antibiotic courses exceeded number of recorded exacerbations by a mean of 2.6 per rescue-pack-recipient, significantly more than in non-rescue-pack recipients (2.59 vs 0.61, difference 1.99; 95% CI 0.92-3.05; p<0.001). 35% of patients in both groups had sub-optimal inhaler adherence.
Conclusion If reproduced, these findings suggest that rescue-pack recipients carry a higher, largely uncoded treatment burden that escapes both structured records and annual reviews, and that COPD studies relying on EHR data alone may be biased. The scale of under-recording may risk leaving frequent exacerbators unidentified, missing opportunities for evidence-based interventions and specialist referrals. A third of rescue-pack recipients had suboptimal inhaler adherence, resorting to potent self-administered OCS/antibiotics while underusing maintenance therapy that could prevent exacerbations. Practices providing rescue-packs should implement robust review systems.

S22 PREVALENCE AND CLINICAL CHARACTERISTICS OF PATIENTS WITH COPD WHO CONTINUE TO EXACERBATE DESPITE TRIPLE THERAPY IN ENGLAND: THE SIRIUS-UK STUDY

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10.1136/thorax-2026-BTSabstracts.28

Introduction and Objectives A significant proportion of patients with COPD continue to experience exacerbations despite triple therapy (EDTT), representing an unmet clinical need. This study estimates the point prevalence of such patients in England and describes their clinical characteristics.

Methods SIRIUS-UK is a retrospective cohort study using linked data from CPRD Aurum, Hospital Episode Statistics, and the Office for National Statistics (protocol #25_005716). Patients aged ≥40 years with COPD who remained on triple therapy (TT, single inhaler ICS/LABA/LAMA or ICS/LABA+LAMA) between 01/02/2015 and 28/02/2019, and experienced a qualifying exacerbation event (QEE: either a second moderate exacerbation [primary care event treated with OCS/antibiotics] within 365 days, or one severe exacerbation [hospitalisation due to COPD]) were classified as EDTT. Point prevalence was calculated as the proportion of COPD patients alive on 28/02/2019 meeting EDTT criteria. Clinical characteristics were assessed in the analytical cohort.

Results Of 290,672 patients with COPD alive on 28/02/2019, 40,082 met the EDTT definition, yielding a point prevalence of 13.8% (95% CI 13.7–13.9). The analytical cohort included 50,091 EDTT patients: 47.3% had recurrent moderate exacerbations, 52.7% had severe exacerbations. Mean age was 72.3 years (SD 10.5); 22.6% were <65 years, 50.3% female, and 31.9% current smokers. Socioeconomic deprivation was notable, with 52.9% in the two most deprived IMD quintiles. Most (99.3%) had primary care contact in the year before QEE, but only 22.2% saw a respiratory outpatient. See table 1 for further details.

Conclusions Approximately one in seven COPD patients in England continue to exacerbate despite triple therapy, highlighting a substantial unmet need. These patients are typically older, more deprived, and clinically complex. Such individuals should be prioritised for advanced therapies and targeted, individualised treatment optimisation to improve outcomes.

Abstract S22 Table 1 Clinical characteristics of the EDTT analytical cohort

Characteristic	Result (n=50,091)*
QEE type	
Recurrent moderate exacerbations, %	47.3%
Severe exacerbation, %	52.7%
Triple therapy within year prior to QEE**	
Multiple inhaler ICS/LABA + LAMA, n (%)	48,973 (97.7%)
Single inhaler ICS/LAMA/LABA, n (%)	1,616 (3.2%)
Symptoms	
CAT score, mean (SD)	19.1 (8.8)
Missing CAT, %	65.4%
Lung function – GOLD grades (n = 37,263)	
GOLD grade 1 (FEV1 % predicted ≥80%), n (%)	3,372 (9.0%)
GOLD grade 2 (FEV1 % predicted ≥50% to <80%), n (%)	16,099 (43.2%)
GOLD grade 3 (FEV1 % predicted ≥30% to <50%), n (%)	13,188 (35.4%)
GOLD grade 4 (FEV1 % predicted <30%), n (%)	4,604 (12.4%)
Missing GOLD, n	12,828
FEV1/FVC, mean (SD)	0.6 (0.2)
Missing FEV1/FVC, n	18,186
Blood eosinophils (n = 34,413)	
Eosinophils, mean (SD), x10^9/L	0.2 (0.2)
Eosinophils, median (IQR), x10^9/L	0.2 (0.1–0.3)
<0.3 x10^9/L, n (%)	23,789 (69.1%)
≥0.3 x10^9/L, n (%)	10,624 (30.9%)
Missing eosinophils, n	15,678
Smoking cessation attendance, n (%)	30,998 (61.9%)

*Percentage denominators are patients with available data and are provided with the characteristic; n=50,091 unless otherwise specified; **Categories are not mutually exclusive because treatment was assessed over the year prior to QEE.

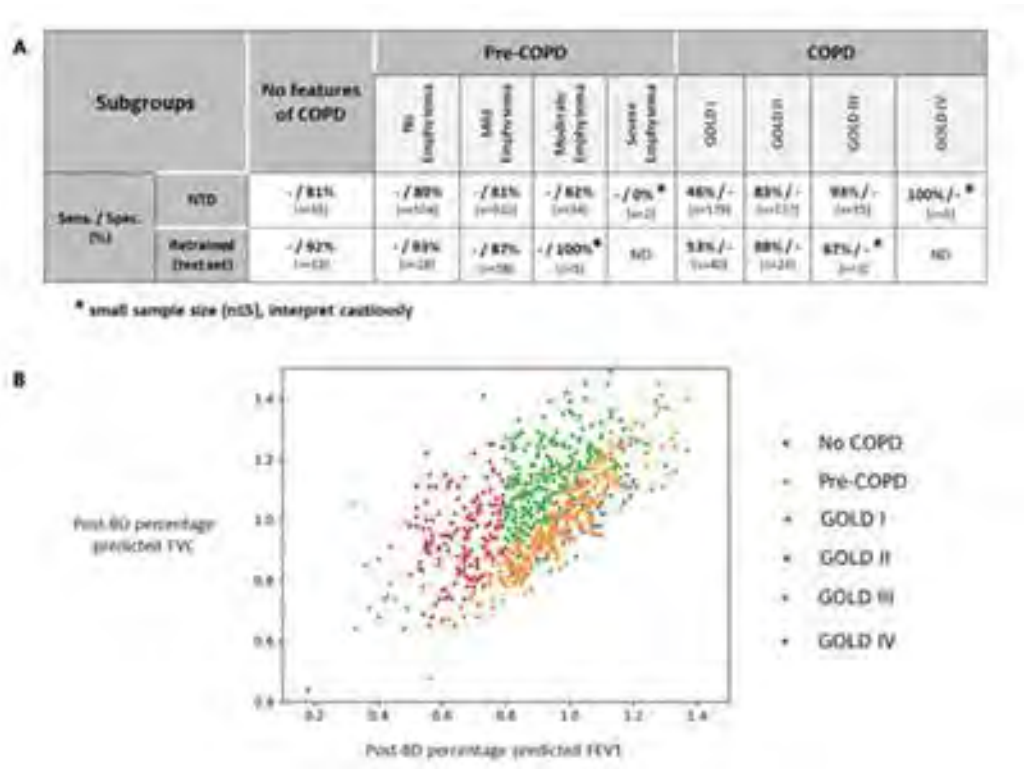
S23 BEYOND-FRONTIER: UTILISING HULL LUNG HEALTH STUDY DATA TO INVESTIGATE THE DIAGNOSTIC ACCURACY OF N-TIDAL DIAGNOSE DIAGNOSTIC ALGORITHMS TO IDENTIFY COPD AND PRE-COPD AMONG PARTICIPANTS IN THE FRONTIER PROGRAMME

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10.1136/thorax-2026-BTSabstracts.29

Introduction Many COPD patients are diagnosed late in disease, with 1 in 8 waiting >10 years for diagnosis. Spirometry is the gold standard COPD diagnostic test; however, capacity is limited by the number of ARTP qualified healthcare professionals, the time taken to carry out spirometry, and acceptability to patients. N-Tidal Diagnose 1 v1.0 (NTD; EU MDR class IIa COPD diagnostic software utilising high-resolution tidal breathing capnography) offers a novel solution which is faster, patient-friendly and more accessible to the healthcare community.

Methods This retrospective cohort study (NCT06788613) recruited consecutive participants from the FRONTIER COPD case-finding programme, who had symptoms and/or emphysema on low-dose CT noted during NHS Lung Cancer Screening. Demographics, clinical characteristics, patient reported outcomes, spirometry and N-Tidal data were collected. Clinicians making the diagnosis of COPD by spirometry (reference standard) were blind to NTD (index test).



Abstract S23 Figure 1 (A) Diagnostic performance (sensitivity for COPD, specificity for No COPD) of NTD and retrained algorithm by subgroup. The test set for the retrained algorithm contained 151 participants after excluding indeterminates (n=55), failed QC (n=2) and those without a pre-bronchodilator capnogram (n=14); (B) Distribution of the participants used for retraining in percentage predicted FEV1 and FVC by subgroup. COPD status is defined using GOLD spirometry criteria and pre-COPD is defined as CAT score of 10 or more and/or emphysema on CT, without airflow imitation on spirometry. ND - no data

NTD diagnostic classifications include: 'highly unlikely', 'unlikely', 'indeterminate', 'likely' or 'highly likely'. Primary analysis was carried out blind to clinical data. Training data were subsequently released for secondary analysis. Revalidation data were retained to investigate performance. NTD classifications of 'likely' or 'highly likely' were considered 'positive'.

Results Data were available for 1,117 participants (54% male). Pre-bronchodilator capnograms were not available for 58 patients (5%). 97.5% of the remaining patients had a NTD result that passed quality control (QC) checks.

NTD diagnostic results (excludes indeterminates, failed QC, and those without pre-bronchodilator capnogram) were available for 61% participants. Sensitivity, specificity, Positive Predictive Value (PPV) and Negative Predictive Value (NPV) in the primary analysis were 63.4%, 82.6%, 76.7%, and 71.4%, respectively. Retrained sensitivity, specificity, PPV and NPV for the test-set were 65.7%, 90.5%, 84.6%, and 76.8%, respectively.

NTD diagnostic performance was higher in GOLD 2–4 and individuals without COPD or emphysema. Performance was lower in GOLD 1, and those with pre-COPD and emphysema; potentially indicating overlapping structural and physiological abnormalities. Supranormal FVC was the primary contributor to obstructive FEV1/FVC in a proportion of GOLD 1 patients.

Conclusions NTD's diagnostic classification appears responsive to COPD features, including airflow limitation and emphysema severity, potentially indicating its ability to provide clinical insights not fully reflected in spirometry.

S24 TARGETED CASE-FINDING AND PROACTIVE CLINICAL MANAGEMENT IN A PRIMARY CARE SETTING REDUCES EXACERBATIONS IN COPD PATIENTS WITH EXACERBATION HISTORY AND SCOPE FOR TREATMENT OPTIMISATION: FINDINGS FROM THE PREVAIL CLUSTER RANDOMISED TRIAL

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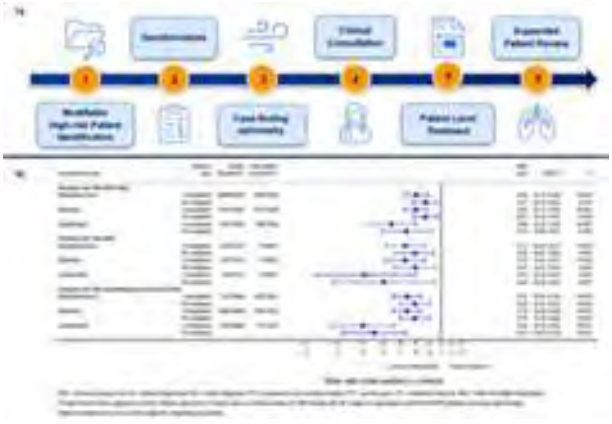
10.1136/thorax-2026-BTSabstracts.30

Introduction Patients with COPD are at high risk of exacerbations. The PREVAIL trial investigated the CONQUEST quality improvement programme (QIP) in patients with newly diagnosed (ND) and already diagnosed (AD) modifiable high-risk (MHR) COPD.

Aims and Objectives To compare COPD exacerbations between CONQUEST patients and usual care.

Methods English general practices were randomised 1:1 to CONQUEST (intervention) or usual care (control). MHR eligibility: 40-80 years, COPD diagnosis or positive smoking history, dual COPD maintenance therapy or less, >=2 moderate or >=1 severe exacerbation in prior 24 months (>=1 in last 12 months). ND patients were identified by CONQUEST spirometry or usual care. CONQUEST provided case-finding, clinical review for assessment and treatment optimisation, and follow-up (figure 1). Exacerbations with subsequent hospitalisation/OCS/antibiotics 8-28 days after initial treatment were defined as complicated. Electronic health records were analysed using propensity score weighted models.

Results 85 intervention and 90 control practices contributed 5,631 patients (mean follow-up: 24 months). Exacerbations and complicated exacerbations reduced by 13% and 26% compared to controls, 21% and 45% in those on monotherapy or less at baseline and 21% and 40% in ND patients (figure 1).



Abstract S24 Figure 1 Intervention and results panel. 1a: Summary of CONQUEST intervention; 1b: Exacerbation rates in MHR patients with COPD

Conclusions The CONQUEST QIP reduced COPD exacerbations in primary care MHR patients, especially those on minimal therapy or newly diagnosed; supporting prompt clinical review and treatment optimisation of at-risk patients.

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Lung cancer – AI & risk

S25 IMAGING-BASED BIOLOGICAL AGE ESTIMATION PREDICTS COMPETING MORTALITY RISK IN LUNG CANCER SCREENING

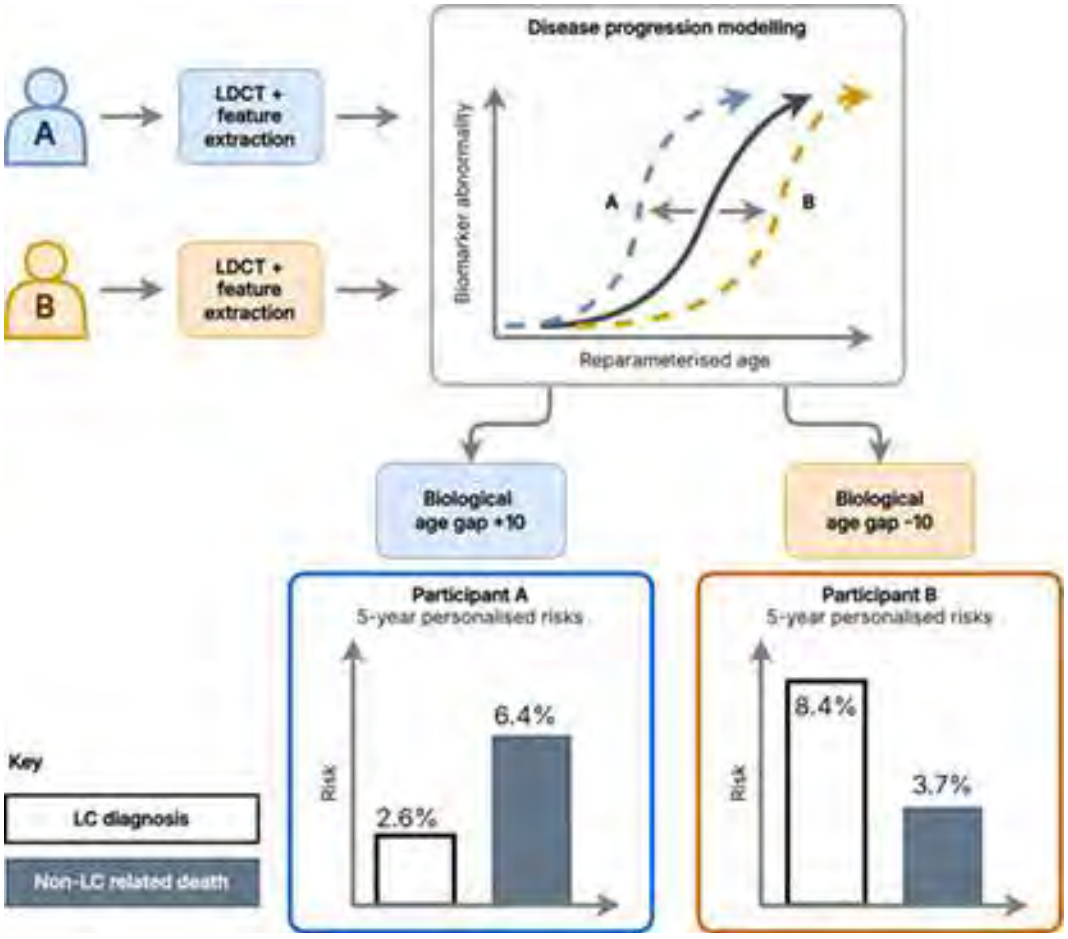
[1]DO Cheng, [1]J McCabe, [2]T Patrick, [1]R Chapman, [1]S Wang, [1]T Yang, [1]A Szmul, [1]M Azimbagirad, [1]S Aslani, [3]ML Mullin, [2]SB Naidu, [2]CR Khaw, [1]ACP So, [2]N Navani, [4]A Nair, [5]JR Hurst, [6]A Altmann, [2]A Pennycuick, [1]DC Alexander, [1]NP Oxtoby, [1]AL Young, [7]M Sperrin, [2]SM Janes, [1]J Jacob. *[1]UCL Hawkes Institute and Department of Computer Science, University College London, London, UK; [2]Lungs for Living Research Centre, University College London, London, UK; [3]Division of Respiratory Medicine, Cumming School of Medicine, University of Calgary, Calgary, Canada; [4]Department of Radiology, University College London Hospitals NHS Trust, London, UK; [5]UCL Respiratory, University College London, London, UK; [6]UCL Hawkes Institute and Department of Medical Physics and Biomedical Engineering, University College London, London, UK; [7]Division of Informatics, Imaging & Data Sciences, University of Manchester, Manchester, UK*

10.1136/thorax-2026-BTSabstracts.31

Background Lung cancer screening (LCS) participants have a high competing risk of non-lung cancer related death, which limits screening benefit. We hypothesized that imaging-based biological age can identify individuals at higher risk of non-lung cancer mortality.

Methods Low dose computed tomography scans from a large LCS trial were retrospectively analyzed. Bone density loss, muscular fat infiltration, vascular calcification, and visceral fat mass were calculated using segmentations from a nnU-Net based deep learning segmentation model. Each participant's age gap, defined as the difference between their estimated biological age and chronological age, was estimated with disease course mapping using a Bayesian mixed-effects model. Disease course map construction used all available longitudinal scan data, whilst individual biological age prediction used baseline scan data only. The performance of age gap as a parameter in competing risk models for non-lung cancer related death versus lung cancer diagnosis was evaluated, following the REMARK reporting guideline.

Findings We included 29,745 scans from 12,478 participants in the final analysis. Median follow-up was 5 years, representing 58,284 person-years at risk. Multivariable survival models of competing risks were constructed, including the estimated age gap. The subdistribution hazard ratio (sHR) for each decade of age gap for non-lung cancer related death was higher (sHR=2.0, 95% CI 1.8–2.3), compared to lung cancer diagno-



Abstract S25 Figure 1

sis (sHR=1.3, 95% CI 1.2–1.5). Addition of age gap improved model concordance of non-lung cancer death compared to a model with clinical variables alone (pooled C-index=0.69 vs 0.73, difference +0.04, 95% CI 0.03–0.04, p<0.001).

Interpretation Biological age estimation using an imaging-based disease progression model improves prediction of non-lung cancer related death in lung cancer screening.

S26 PERSONALISING LUNG CANCER SCREENING ELIGIBILITY REASSESSMENTS BASED ON REAL-WORLD LONGITUDINAL RISK TRAJECTORIES

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10.1136/thorax-2026-BTSabstracts.32

Rationale Risk-based lung cancer screening eligibility is dynamic. Individuals initially ineligible may become screening eligible later in life. The extent and timing of eligibility transitions over time in real-world populations remain poorly characterized, limiting understanding of risk reassessment needs.

Methods This post-hoc analysis of the Rotterdam Study, a prospective population-based cohort, included 14,919 participants followed for a median of 16 years, during which 357 developed lung cancer. Longitudinal changes in screening eligibility were evaluated, defining eligibility as PLCOM2012NoRace risk ≥1.51% between ages 55-74 years. The study aimed to inform personalised risk reassessment strategies.

Results Longitudinal PLCOM2012NoRace risk trajectories showed that 61.8% (3,171/5,129) of individuals who were ever eligible for screening were initially below the 1.51% risk threshold. Among individuals diagnosed with lung cancer who were ever above the threshold, 53.2% (144/271) were initially below the risk threshold, with 43.2% (117/271) crossing the threshold before the age of 75 and a further 10.0% (27/271) at or after 75 years of age. All initially ineligible individuals who reclassified to a risk ≥1.51% and were diagnosed with lung cancer, crossed the threshold before clinical diagnosis, with a median lead time of 7 years (IQR 3-12). Individuals with higher baseline risk were more likely to cross the risk threshold, ranging from <3.0% of those with baseline risk below 0.49%, to up to 57.8% of baseline risks 1-1.50% depending on age-group. Median years to reclassification varied by age group, ranging from 5.5 to 3.2 years between ages 55-59 and 4.4 to 0.9 years for ages ≥60 across risk groups.

Conclusion A substantial proportion of individuals developing lung cancer are ineligible at initial risk assessment but can be timely identified through reassessment. Baseline PLCOM-2012NoRace risk may help identify individuals likely to become high risk and guide risk-stratified reassessment intervals. Individuals with PLCOM2012NoRace risks <1.0% are unlikely to become screening eligible, while those with PLCOM2012NoRace risks 1.0%-1.50% should undergo biennial (ages <60) or annual (ages >60) reassessment.

S27 THE IMPACT OF MENTAL HEALTH DIAGNOSES ON THE UPTAKE OF LUNG CANCER RISK ASSESSMENT AND LOW-DOSE CT SCREENING IN A LUNG CANCER SCREENING IMPLEMENTATION STUDY

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10.1136/thorax-2026-BTSabstracts.33

Background The lung cancer screening (LCS) pathway in England involves inviting people who have ever smoked for a lung cancer risk assessment (usually by telephone) with those at higher risk eligible for low-dose CT (LDCT) screening. Mental health diagnoses, particularly severe mental illness (SMI), are associated with reduced participation in other cancer screening programmes, but there is limited published data investigating the impact of these diagnoses on participation in the LCS in the UK. Here we present data from the Yorkshire Lung Screening Trial, an LCS implementation study, assessing the impact of mental health diagnoses on engagement with screening.

Methods Primary care codes for mental health diagnoses (anxiety, depression, cognitive impairment, memory concerns, SMI) were extracted for invited individuals. Impact on the likelihood of responding to a written invitation for a risk assessment, and on attending LDCT screening if eligible, were analysed using multivariable logistic regression adjusting for relevant factors (age, IMD quintile, smoking status, sex and ethnicity). Data were analysed using Stata v19.5.

Results Of 27,761 individuals invited for risk assessment, 6,930 (25.0%) had at least one mental health diagnosis and 1,512 (5.4%) had a SMI. Overall risk assessment uptake was 52.5%, and uptake of LDCT screening for those eligible was 86.9%; the associations with mental health diagnoses are shown in table 1. The presence of anxiety or SMI did not impact the uptake of risk assessment, whereas uptake was increased in people with depression (OR_{adj} 1.11, 95% CI, 1.02-1.22, p=0.020), cognitive impairment (OR_{adj} 1.18, 95% CI, 1.09-1.28, p<0.001) and memory concerns (OR_{adj} 1.32, 95% CI, 1.06-1.64, p=0.013). SMI was associated with a significant reduction in uptake of LDCT screening for eligible people (OR_{adj} 0.61, 95% CI, 0.46-0.82, p=0.001) but other mental health diagnoses were not associated with altered LDCT uptake.

Conclusions Increased uptake of risk assessment for people with cognitive impairment or memory concerns may reflect system navigation by caregivers. The low uptake of LDCT screening for eligible people with SMI is a concern and suggests that extra support should be offered to this high-risk population to ensure they benefit from the improved outcomes associated with LCS.

S28 RANDOMISED CONTROLLED TRIAL OF PATIENT NAVIGATION FOR LUNG CANCER SCREENING NON RESPONDERS

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10.1136/thorax-2026-BTSabstracts.34

Introduction Inequalities in uptake of lung cancer screening persist internationally, limiting the benefit in populations at highest risk. Patient navigation (PN) provides tailored support to facilitate access to care and has shown efficacy in underserved groups. However, evidence comes exclusively from the US limiting generalisability to other healthcare systems. We evaluated the effect of a PN intervention (relative to usual care) on uptake of telephone-based lung cancer risk assessment, low dose CT (LDCT) completion, and cancer detection in a lung cancer screening implementation trial in the UK.

Abstract S27 Table 1 Relationship of mental health conditions with response to invitation for risk assessment and uptake of LDCT screening if eligible

	Response to invitation for risk assessment			Uptake of LDCT screening if eligible		
MH Condition[2]	**N**	**OR_{adj}[1] (95% CI)**	**p-value**	**N**	**OR_{adj}[1] (95% CI)**	**p-value**
Anxiety[3]	**1,510**	1.11 (0.99-1.24)	0.065	**282**	0.84 (0.59-1.18)	0.314
Cognitive impairment	**3,342**	1.18 (1.09-1.28)	<0.001	**945**	0.98 (0.79-1.21)	0.843
Depression[3]	**2,219**	1.11 (1.02-1.22)	0.020	**459**	0.94 (0.70-1.26)	0.666
Memory concerns[3]	**369**	1.32 (1.06-1.64)	0.013	**97**	0.82 (0.47-1.44)	0.497
Severe mental illness	**1,512**	1.10 (0.98-1.22)	0.101	**340**	0.61 (0.46-0.82)	0.001

[1]Adjusted for age, Index of Multiple deprivation (IMD) quintile, smoking status, sex and ethnicity. [2]Mental health comorbidities derived from the eFIv2 deficits. [3]Recorded up to 5 years before randomisation date

Methods We conducted a two-arm randomised controlled trial nested within the Yorkshire Lung Screening Trial. Participants aged 55-80 with a history of ever smoking who had not responded to postal invitations in two previous biennial screening rounds were randomised to PN (comprising a postal notification of a prearranged, protocolised, telephone appointment with a trained navigator; n=7,335) or usual care (a further postal invitation to screening; n=7,315). Logistic regression compared screening uptake and cancer detection by trial arm on an intention-to-intervene basis, adjusted for clustering by household.

Results Uptake of risk assessment for screening was higher in the PN arm than usual care (40.5% vs. 8.5%; OR: 7.37, 97.5% CI 6.58–8.26, see table 1), as was completion of LDCT screening (10.4% vs. 2.7%; OR: 4.24, 97.5% CI 3.52–5.11). More cancers were also detected in the PN arm (23 vs. 3; OR: 7.67, 95% CI 2.30–25.54), and the cancer yield per LDCT scan was also higher (3.0% vs. 1.5%). A higher proportion of people undergoing risk assessment in the PN arm were from the most deprived quintile (35.2% vs. 30.4%) and were currently smoking (33.9% vs. 28.0%).

Conclusions This is the first trial of PN for lung cancer screening outside the US. The increased cancer yield per LDCT scan in the pathway navigation arm suggests that the additional participants motivated to engage with screening by this intervention are at much higher risk of lung cancer. PN stands to improve the effectiveness and equity of lung cancer screening in the UK, but multi-site testing across different delivery models and navigator staffing profiles is needed.

Abstract S28 Table 1 Statistical comparison of control and intervention arms

	Control arm	Intervention arm
Randomised participants	**7,315**	**7,335**
Underwent initial telephone assessment *	**618 (8.5%)**	**2,970 (40.5%)**
Odds ratio	7.37	
97.5% confidence interval (p-value) adjusted for clustering by household	6.58 – 8.26 (p << 0.0001)	
Underwent LDCT scan *	**195 (2.7%)**	**763 (10.4%)**
Odds ratio	4.24	
97.5% confidence interval (p-value) adjusted for clustering by household	3.52 – 5.11 (p << 0.0001)	
Screen-detected lung cancers	**3 (0.04%)**	**23 (0.31%)**
Odds ratio	7.67	
95% confidence interval (p-value) adjusted for clustering by household	2.30 – 25.54 (p = 0.001)	
Screed-detected lung cancer as a proportion of those undergoing LDCT scans	**1.54%**	**3.01%**

* Co-primary outcomes, confidence intervals were calculated at 97.5% allowing for their related nature

S29 RADIOLOGIST-EXPLAINABLE AI CONCORDANCE AND CONFIDENCE IN EXPLAINABLE AI SUPPORTED CLINICAL IMAGING DECISIONS IN LUNG CANCER EARLY DIAGNOSIS

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10.1136/thorax-2026-BTSabstracts.35

Background Explainable Artificial Intelligence (XAI) may support pulmonary nodule risk stratification, but its clinical value depends on how radiologists use AI outputs during interpretation. Although the impact of AI has mostly been evaluated performance metrics, interactions between clinicians and XAI remain poorly understood. We evaluated whether AI-predicted malignancy risk with morphology-based explanations influenced radiologist decisions and confidence.

Methods Four consultant radiologists independently assessed 100 biopsy-confirmed pulmonary nodules from a multi-centre UK cohort using a counterbalanced design. Each radiologist reader reviewed one list with AI assistance and the other without AI assistance. Reader pairs were assigned opposite conditions so that each case was assessed under both conditions by different readers. XAI support quantified radiological characteristics, such as spiculation, nodule type, to assist radiologists. In the AI-assisted condition, radiologists were shown AI-predicted malignancy risk with explanations simultaneously. Readers recorded a binary malignancy decision and confidence score.

Results Aggregate performance of readers was comparable with and without AI assistance (accuracy 0.78 vs 0.76, sensitivity 0.94 vs 0.93, specificity 0.68 vs 0.64, all $p>0.05$).

Average radiologist confidence was higher for correct than incorrect calls in both conditions (3.69 vs 3.00, $p < 0.01$ with AI, 3.55 vs 2.57, $p < 0.001$ without AI). Specifically, confidence was highest when their decisions agreed with the correct AI output (3.85) but remained high when concurring with an incorrect AI (3.39). However, confidence was low when readers correctly disagree an incorrect AI (2.53) and lowest when disagreeing with a correct AI (1.82).

Discussion AI assistance did not significantly improve aggregate diagnostic performance. In both conditions, radiologist confidence was higher for correct than incorrect cases suggesting that radiologists had some insight into which calls were reliable. However, confidence tracked concordance with the AI more than diagnostic correctness as readers remained confident when agreeing with an incorrect AI. Among discordant reads, confidence was relatively higher when readers correctly overrode an incorrect AI than when they disagreed with a correct one. These data have important implications for radiologist trust in AI decision-support and whether this varies according to case difficulty. The role of AI in 'more challenging' cases requires further study.

Abstract S29 Table 1 Nodule and read characteristics by reader-AI agreement and AI correctness; each nodule was read by two radiologists (200 reads, 100 nodules). N reads = reader-nodule reads, N unique scans = distinct nodules per column (a nodule spans two columns when its readers differed). Bottom row shows the joint stance of both readers toward AI: red, both disagreed; green, both agreed; amber, split (one disagreeing); yellow, split (one agreeing)

	Disagrees x AI incorrect	Disagrees x AI correct	Agrees x AI incorrect	Agrees x AI correct
N reads	19	11	33	137
N unique scans	16	9	23	72
Malignant, n(%)	2 (12%)	3 (33%)	1 (4%)	37 (51%)
Size median (IQR) mm	15.5 (13.0-18.5)	13.0 (7.0-17.0)	14.0 (12.0-19.0)	13.0 (7.0-19.2)
Spiculated, n (%)	0 (0%)	2 (22%)	3 (13%)	16 (22%)
Solid, n (%)	11 (69%)	6 (67%)	17 (74%)	59 (82%)
Part-solid, n (%)	3 (19%)	1 (11%)	5 (22%)	10 (14%)
GGO, n (%)	2 (12%)	2 (22%)	1 (4%)	3 (4%)
Mean confidence	2.53	1.82	3.39	3.85
Reader-AI agreement	6 13	4 7	13 20	7 130

S30 FDG PET/CT FOR THE ASSESSMENT/STAGING OF GROUND-GLASS AND SUBSOLID NODULES: HELP OR HINDRANCE?

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10.1136/thorax-2026-BTSabstracts.36

Introduction and Objectives 18-fluorodeoxyglucose positron emission tomography/computed tomography (PET/CT) is widely used in the staging pathway for lung cancer and accounts for a significant proportion of PET/CT activity within our institution. Current guidance supports PET/CT in the assessment of high-risk pulmonary nodules above established local size thresholds. However, evidence for its utility in subsolid and ground-glass nodules remains limited. Despite this, PET/CT is frequently requested for these lesions in routine clinical practice. We aim to evaluate the clinical value of PET/CT in patients with presumed early-stage lung cancer.

Methods A retrospective analysis is being undertaken of all PET/CT scans performed for suspected lung cancer across South-East Scotland over a five-year period (n=4414). The current data includes cases performed over a 17-month period. Patients with CT identified pre-PET N0M0 disease were analysed, and pulmonary nodule characteristics, including size and morphology, were reviewed. Rates of CT occult nodal and distant metastatic disease detected on PET/CT were assessed. We also identified cases with incidental findings following PET/CT and evaluated their benefit to the patient.

Results A total of 1011 PET/CT scans were performed. 366 patients with confirmed lung cancer (by tissue confirmation or multi-disciplinary team agreement) had radiological pre-PET N0M0 disease. PET/CT demonstrated 57 suspected nodal metastases but 22 of these were later down staged by tissue confirmation. In all cases where nodal metastases were confirmed, the primary lesion was a solid pulmonary nodule with a mean diameter of 34.5mm (range 11mm - 100mm). PET/CT identified 12 patients with distant metastasis. No nodal or distant metastatic disease was identified in patients with subsolid nodules or ground-glass nodules. A total of 95 incidental findings were identified, with only 15 (15.8%) significantly benefiting patient care.

Conclusions PET/CT offers limited additional staging benefit in patients with subsolid or ground-glass pulmonary nodules, while potentially increasing incidental findings and additional investigations. These preliminary findings suggest that more selective utilisation of PET/CT may be appropriate in this cohort. Completion of the full five year analysis will help define thresholds at which PET/CT provides meaningful staging benefit in early lung cancer pathways.

Rewriting the immune landscape of the lung

S31 BROAD-SPECTRUM ANTIBIOTICS INDUCE POLARIZATION OF INFLAMMATORY TYPE 2 IMMUNE RESPONSES IN THE LUNG AND ALTER THE IMMUNE LANDSCAPE IN A MODEL OF ASTHMA

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10.1136/thorax-2026-BTSabstracts.37

Introduction Strong epidemiological evidence links repeated broad-spectrum antibiotic use in humans to the development of asthma, however the cellular and molecular mechanisms as to how this occurs are unclear.

Methods Oral broad-spectrum antibiotics were administered to mice for one week. Lung and gut microbiota were assessed by 16S bacterial sequencing. Pulmonary immune responses were assessed by multi-parameter flow cytometry and RNAsequencing following enzymatic lung digest. House dust mite model to induce type 2 inflammation was used, with sensitisation during antibiotic administration and challenge following antibiotics.

Results Mice administered antibiotics developed spontaneous activation of alveolar and interstitial macrophages in the lung, followed by increased type 2 inflammation as indicated by enhanced numbers of pulmonary eosinophils, Th2 cells and type 2 innate lymphoid cells (ILC2s). RNAsequencing carried out on isolated alveolar macrophages indicated extensive transcriptomic changes including alterations to cellular metabolism pathways, inflammatory signalling and inflammasome activation after antibiotics. Functionally, the metabolic capacity of alveolar macrophages was enhanced. The induction of type 2 inflammation was monocyte-macrophage dependent since mice deficient in monocytes/recruited macrophages (*Ccr2*$^{-/-}$ mice) did not exhibit any change in eosinophils, Th2 cells, or ILC2s after antibiotics. Lung microbiota remained stable after antibiotic use, but the gut microbiota was extensively altered indicating pulmonary immune changes may be influenced indirectly by the gut microbiota. A key candidate is the intestinal metabolite butyrate, a potent anti-inflammatory molecule which is generated from the gut microbiota and can reach peripheral tissues via the bloodstream. Intestinal and circulating butyrate levels were depleted by antibiotics. Administration of butyrate alongside antibiotics prevented alveolar macrophage activation, excess metabolic function and prevented transcriptomic changes. Interstitial macrophages were expanded in all cases during the house dust mite model of asthma, but these were polarised towards type 2 macrophages after antibiotics alongside excess proliferation of ILC2s.
Conclusion Broad-spectrum antibiotic use polarizes type 2 inflammation in the lung and alters the immune landscape in asthma models via monocyte-macrophage dependent mechanisms. Mechanistically, bacterial-derived metabolites from the intestine play a key role in preventing excess type 2 inflammation in the lung. These data have important implications for repeated administration of broad-spectrum antibiotic use in humans.

S32 AN INTACT INTEGRATED STRESS RESPONSE RESTRAINS THE IL-6-PSTAT3 PATHWAY

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Introduction Mutations in GCN2 (General Control Nonderepressible 2) cause deadly forms of pulmonary hypertension such as pulmonary veno-occlusive disease (PVOD). GCN2 is a serine/threonine protein kinase, one of 4 kinases that phosphorylates the α-subunit of the translation initiation factor eIF2, which activates a common adaptive pathway known as the Integrated Stress Response (ISR). We have previously shown that the *Gcn2*$^{-/-}$ mouse develops a mild pulmonary hypertensive phenotype at baseline, which is worsened by LPS exposure. Genetic deletion of IL-6 ameliorates the pulmonary vascular disease in both this and mitomycin-C induced pulmonary vascular disease.
Methods Wild-type and *Gcn2*$^{-/-}$ fibroblasts and mice were used and exposed to specific inhibitors and activators of the Integrated Stress Response.
Results Wild-type mice receiving both Integrated Stress Response InhiBitor (ISRIB) and LPS have an exaggerated IL-6 response in their sera and an increased phospho-Stat3 (pStat3) response in their lungs. The converse is also true, as administration of a novel GCN2 activator, C20, decreased the LPS-induced pStat3 response in wild-type mouse fibroblasts. Another GCN2 activator, NXP800, also ameliorated the pStat3 response in fibroblasts when co-administered with LPS. As NXP800 is currently in phase I trials (Clinicaltrials.gov ID: 50382) and is orally bioavailable, we tested this in a mouse model. NXP800 also ameliorated the pStat3 response in wild-type mouse lungs when co-administered with LPS. In an independent genetic model, we used eIF2α^{AA} fibroblasts, which are unable to mount an ISR due to the mutation of a critical serine at position 51 to alanine. These eIF2α^{AA} and corresponding wild-type fibroblasts were exposed to either vehicle or mitomycin-C for 24 hours. The eIF2α^{AA} fibroblasts responded with increased levels of IL-6 mRNA and protein compared to fibroblasts derived from their wild-type siblings, which phenocopies GCN2 deficiency.
Discussion and Conclusions Mutations in GCN2 cause a hyper-inflammatory phenotype characterised by increased IL-6. Both pharmacological blockade and genetic ablation of the ISR phenocopy this outcome in both fibroblasts and mice, while activation of the ISR via GCN2 blunts the IL-6-induced pStat3 response. We conclude that an intact and functional ISR is required to restrain the IL-6 response.

S33 IMPACT OF CIGARETTE SMOKE EXTRACT AND IL-17A ON T2 AND NON-T2 INFLAMMATORY RESPONSES IN HUMAN AIRWAY SMOOTH MUSCLE CELLS

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10.1136/thorax-2026-BTSabstracts.39

Background Asthmatic airway inflammation can be classified into Type 2 (T2), which is responsive to glucocorticoids, and non-T2, which shows resistance to glucocorticoids. Cigarette smoke (CS), as well as the Th17 cytokine IL-17A, play a role in driving non-T2 inflammatory responses. However, the impact of CS and IL-17A on the T2 inflammatory response in human airway smooth muscle cells (HASMCs) remains unclear. There is evidence indicating that cigarette smoke increases Cyclooxygenase 2 (COX-2) expression and the production of prostaglandin E2 (PGE2) in various cell types, including HASMCs. We hypothesize that CSE and IL-17A alter T2 inflammatory responses through the COX-2/PGE2 pathway.
Methods CSE was prepared by bubbling the smoke from two research cigarettes into 20 ml of cell culture medium. Protein expression was analyzed using Western blotting. The production of T2 inflammatory cytokines was evaluated using ELISA and Bio-Plex assays.
Results CSE inhibited the production of T2 inflammatory cytokines, including IL-4 and IL-13, and the eosinophil chemokines Eotaxin, IP-10 and RANTES in HASMCs, and inhibited IL-17A-induced Th2 cytokines and eosinophilic chemokines. Our results demonstrated that CSE suppresses the T2 inflam-

matory response primarily through a COX-2/PGE2/EP2/EP4/cAMP pathway, which is dependent on oxidative stress.

Conclusion The findings of this study reveal, for the first time, that CSE can alter inflammatory responses in HASMCs and suggests that cigarette smoke promotes the transition from glucocorticoid-sensitive T2 to glucocorticoid-insensitive non-T2 inflammatory responses by inhibiting T2 cytokine production and enhancing the production of the non-T2 chemokine CXCL8. We have identified a key role for the COX-2/PGE2/EP2/EP4/cAMP pathway in cigarette smoke-mediated alterations to inflammatory endotype in HASMCs, which suggests that targeting this pathway holds significant potential for developing innovative treatments for asthmatic smokers.

S34 COMPREHENSIVE CHARACTERIZATION OF A NOVEL PRECLINICAL MOUSE MODEL MIMICKING THE COMPLEX PHENOTYPE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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10.1136/thorax-2026-BTSabstracts.40

Introduction and Objectives Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous condition characterized by progressive and irreversible airflow limitation driven by an excessive inflammatory response to inhaled irritants and pathogens. It encompasses chronic bronchitis and emphysema, often coexisting with varying severity and accompanied by episodic exacerbations typically triggered by bacterial and viral infections. Despite being the third leading cause of death worldwide, current therapies do not halt disease progression. A major limitation in therapeutic development is the lack of animal models that accurately reproduce the complex features of human COPD. This study aimed to characterize a murine model induced by combined noxious stimuli that recapitulates key disease hallmarks within a relatively short timeframe.

Methods Female BALB/c mice were exposed to porcine pancreatic elastase (PPE), *Pseudomonas aeruginosa* lipopolysaccharide (LPS), and polyinosinic:polycytidylic acid (Poly I:C) to induce emphysema, mucus hypersecretion, and mimic bacterial and viral infections. The effect of systemic corticosteroids was evaluated by administering dexamethasone after early disease features had emerged. Comprehensive characterization was performed at molecular, histological, and imaging levels at 2, 3, and 4 weeks. At the final time point, also lung functions and inflammatory infiltrate were assessed.

Results Combined stimuli induced progressive upregulation of the occlusive mucins MUC5AC and MUC5B and promoted mixed inflammatory cell infiltration. Histological analysis showed increased bronchial epithelial thickness and parenchymal destruction over time. Functional impairment, including loss of elastic recoil, airflow limitation, and air trapping, was confirmed by flexiVent and micro-Computed Tomography (micro-CT). Accordingly, transcriptomic and proteomic analyses highlighted that these pathological changes were accompanied by deregulation of distinctive pathways such as upregulation of extracellular matrix (ECM) remodeling and necroptosis, alongside with downregulation of vascular development and epithelial morphogenesis. Dexamethasone partially reduced inflammation but did not significantly improve mucus secretion or emphysematous lesions, consistent with clinical observations in COPD patients.

Conclusions Overall, this murine model reproduced key structural, functional, and molecular features of COPD, supporting its translational relevance. Thereby, it represents a valuable platform for investigating disease mechanisms and evaluating novel therapeutic candidates.

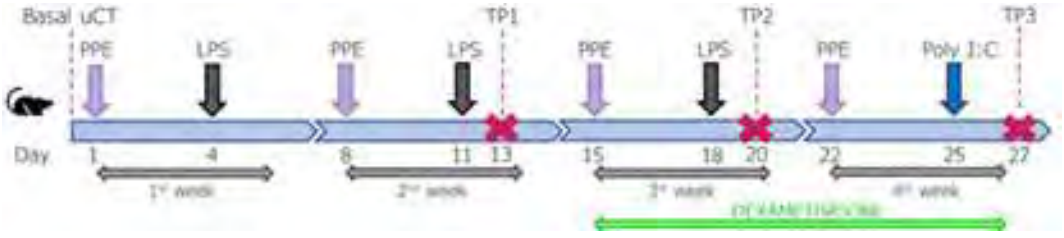
S35 INHIBITION OF EXTRACELLULAR CYCLOPHILIN A SIGNALING ATTENUATES ACID ASPIRATION-INDUCED ACUTE LUNG INJURY

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10.1136/thorax-2026-BTSabstracts.41

Background Acute respiratory distress syndrome (ARDS) presents a major burden in ICU accounting for 10% of all admissions. ARDS carries a high mortality rate up to 35% and survivors often have long term health sequelae. Extracellular Cyclophilin A (eCypA) is an inflammatory mediator particularly known for its role as an inducer of matrix metalloproteinases (MMPs). We have previously shown that eCypA is upregulated in bronchoalveolar lavage fluid (BALF) of patients with ARDS, and promotes inflammation and permeability following ventilator-induced lung injury in mice.[1] In this study we explored whether eCypA plays a role in other aetiologies of lung injury.

Methods Male and female C57BL6 mice were anaesthetised, surgically instrumented and mechanically ventilated with a tidal volume of ~8ml/kg using 100% O_2.2ml/kg of saline or 0.15M hydrochloric acid (pH 1.5) was administered intratracheally to induce lung injury.After 30 minutes, mice were dosed intratra-



Abstract S34 Figure 1

Abstract S35 Table 1 Bronchoalveolar lavage fluid levels of mediators

	Saline + vehicle	HCl + vehicle	HCl + MM284
BALF extracellular Cyclophilin A (ng/ml)	1200±360	38000±8500****	
BALF total protein (mg/ml)	0.50±0.09	2.90±1.26****	1.76±0.36##
BALF sRAGE (pg/ml)	2600±350	88000±12600****	65000±12200###
BALF MMP2 (ng/ml)	20.0±8.1	126±44****	78±23##
BALF CXCL1 (pg/ml)	155±32	1007±636***	611±247
BALF IL-6 (pg/ml)	196±72	2613±1400***	2274±1149

Table legend: Mean±SD. Data analysed by t-test or ANOVA with Dunnett's test. ***p<0.001, ****p<0.0001 vs saline+vehicle; ##p<0.01, ###p<0.001, vs HCl+vehicle. N=7-8/group.

cheally with MM284, an inhibitor of eCypA signaling (1), or vehicle (10% DMSO).Mice were ventilated for a further 2.5 hours following which they were sacrificed and levels of mediators determined in BALF.

Results Acid instillation promoted increased eCypA in BALF, along with increased total protein and soluble RAGE levels, indicating greater permeability and epithelial cell damage. Additionally, levels of IL-6, CXCL1 and MMP2 were also upregulated (table 1). Inhibition of eCypA signaling via administration of MM284 led to significant attenuation of permeability, epithelial injury and MMP2 levels, but not proinflammatory cytokines.

Conclusions Extracellular Cyclophilin A promotes upregulation of MMP2 secretion and epithelial injury/permeability following acid aspiration injury in mice. Inhibition of Cyclophilin A signaling was efficacious after induction of injury, and thus may represent a promising therapeutic target for patients with ARDS.

REFERENCE

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S36 MECHANICAL VENTILATION PROMOTES INFLAMMATORY AND PROFIBROTIC SIGNALING IN THE PLEURAL SPACE FOLLOWING ACUTE LUNG INJURY

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10.1136/thorax-2026-BTSabstracts.42

Background Acute respiratory distress syndrome (ARDS) presents a major burden in ICU. Treatment options are limited primarily to supportive care including mechanical ventilation, although this can lead to worsened inflammation and lung fibrosis. We have recently shown that injurious high tidal volume (V_T) ventilation promotes inflammation in the pleural cavity of mice.[1] Here we explore whether moderate, normally non-injurious V_T, promotes pleural cavity inflammation in mice with pre-existing injury.

Methods Two models of lung injury were induced in male and female C57BL6 mice. i) 30μg lipopolysaccharide (LPS) or saline was instilled intranasally and injury was allowed to develop for 48 hours, before mice were either sacrificed or ventilated using a moderate V_T (20-25ml/kg). ii) 0.15M hydrochloric acid (HCl) or saline was instilled intratracheally following which mice were immediately ventilated at either low (8-10ml/kg) or moderate (15-20ml/kg) V_T. Ventilation was continued for 3 hours before the pleural cavity was lavaged to collect samples. Total protein content was used as a marker of permeability. Proinflammatory molecules measured include interleukin-6 (IL6) and CXCL1. Profibrotic molecules include PAI-1 and MMP9.

Results In the LPS model, single insults (i.e. LPS alone or ventilation alone) had no significant effect on pleural cavity IL6 or CXCL1, while moderate V_T alone tended to increase pleural cavity permeability. However, the combination of pre-injury plus ventilation dramatically enhanced pleural fluid levels of proinflammatory molecules, without further increasing permeability (table 1). Similarly, in the acid model, neither acid+low V_T nor saline+moderate V_T enhanced permeability, proinflammatory or profibrotic molecules. However the combination of acid injury+moderate V_T greatly upregulated permeability, proinflammatory and profibrotic markers in the pleural space.

Abstract S36 Table 1 Pleural lavage fluid proinflammatory and profibrotic molecules

	Total protein (mg/ml)	IL6 (pg/ml)	CXCL1 (pg/ml)	PAI-1 (pg/ml)	MMP9 (pg/ml)
LPS model					
Saline alone	0.40±0.07	16.3±9.0	13.8±9.3		
Saline+moderate V_T	1.45±0.93*	93.7±85.9	157±103		
LPS alone	0.68±0.45	57.3±91.9	37.0±42.3		
LPS+moderate V_T	1.32±0.64	1450±1420*	1118±1399*		
HCl model					
Saline+low V_T	0.64±0.22	12.8±10.1	114±19.0	78.7±45.5	232±120
Saline+moderate V_T	0.87±0.35	55.0±40.1	140±73.0	99.7±45.1	303±124
HCl+low V_T	0.85±0.32	104±54.7	184±99.2	163±101	231±55.8
HCl+moderate V_T	1.08±0.36	452±335*	459±266*	326±115*	404±114*

Table legend: Mean±SD. Data analysed by ANOVA with Sidak's test. *p<0.05 vs corresponding low V_T/non-ventilated groups, N=5-8/group.

Conclusions In clinically relevant models of ARDS, normally non-injurious mechanical ventilation promoted upregulation of proinflammatory and profibrotic molecules in the pleural cavity. While the precise relevance of this remains unclear, these data implicate the pleural cavity as a previously unrecognised source of inflammatory and fibrotic mediators during mechanical ventilation.

REFERENCE

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Beyond Type 2 Asthma

S37 PROINFLAMMATORY NON-T2 IMMUNOLOGY IN THE AIRWAYS OF A T2-LOW SEVERE ASTHMA COHORT

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10.1136/thorax-2026-BTSabstracts.43

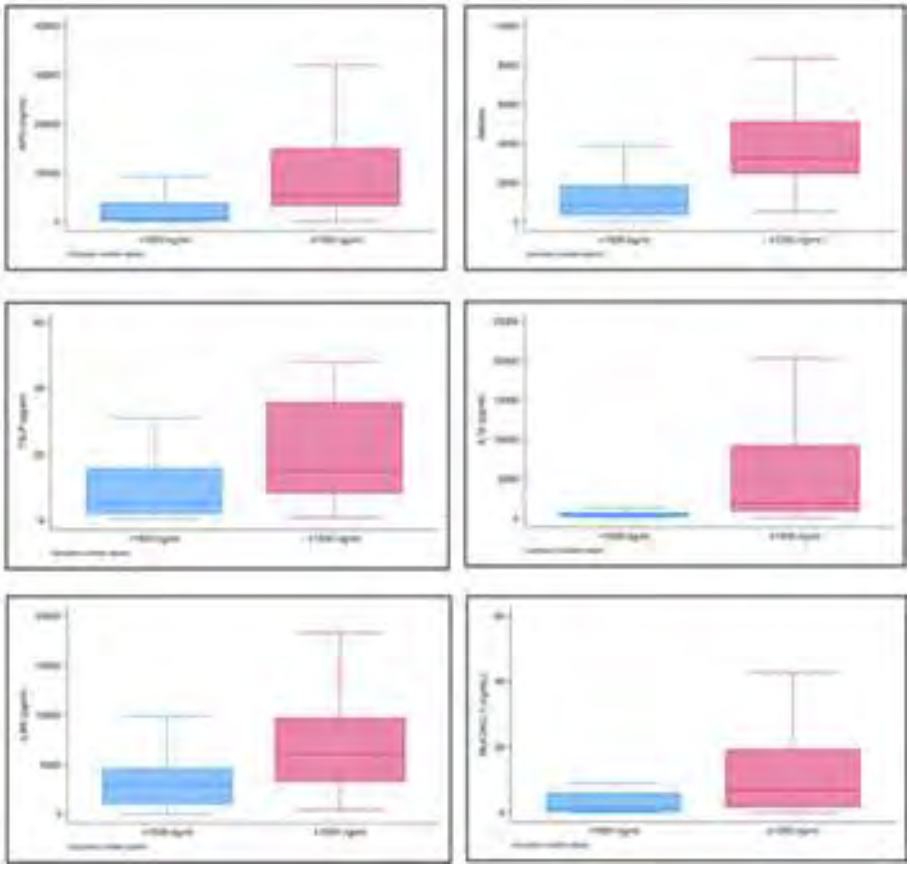
Introduction Asthma is a T2-driven inflammatory disease and therapeutics targeting T2 pathways effectively reduce disease burden. However, suppressing T2 inflammation does not eliminate symptoms or prevent exacerbations in all patients.

Increasing evidence suggests non-T2 mechanisms contribute to poor clinical outcomes. A previous study showed differences in non-T2 inflammatory pathways in a population of T2-high patients treated with mepolizumab. Around a third of patients had increased neutrophil activation, epithelial alarmins, pro-inflammatory cytokines and microbiome shift to increased proteobacteria. These patients had poorer clinical outcomes with anti-IL5 therapy: persistent high symptom burden, poor quality of life and no FEV1 improvement.[1] Using neutrophil elastase (NE) as a biomarker, this study examined a biologic-naive severe asthma cohort with lower T2 inflammation to assess if a similar patient cohort with increased non-T2 inflammation exists.

Methods Analysis was undertaken on sputum samples (n=123) from the 'Biomarker versus symptom-adjusted corticosteroids' trial (RASP Biomarker study).

On sputum proteome analysis, one third of MEX patients exhibited proinflammatory biology associated with neutrophil activation, therefore the upper tertile of NE (1529.3ng/ml (1081,2213) was used as biomarker for proinflammatory biology, with this cohort named 'NE^{HIGH}'.

Sputum samples underwent PCR analysis for pathogen detection, immunoassay for IL-1α, IL-33, IL-1β, IL-6, YKL-40, IL-1RA, TSLP, NE, neutrophil extracellular traps (NET) concentration. Stable isotope dilution liquid chromatography



Abstract S37 Figure 1 Sputum cytokines and proteases in those with low sputum neutrophil elastase (<1500ng/ml) versus those with high sputum neutrophil elastase (≥1500ng/ml)

tandem mass spectrometry quantified mucins (MUC5AC and MUC5B).

Results NE distribution in the RASP biomarker study was similar to the MEX cohort.

As with the MEX cohort, the Biomarker NE^{HIGH} subgroup have longer disease duration, higher symptom burden and a trend towards worse lung function. This subgroup had increased TSLP, IL-1α, IL-1β, IL-6, IL-6r, YKL-40, MPO, NET concentration and MUC5AC-1 (figure 1), as well as increased pathogens when clinically stable.

Conclusion Proinflammatory biology is present in a T2-low severe asthma cohort and is associated with longer disease duration and high symptom burden. Understanding biological drivers of this inflammation could provide an opportunity for targeted therapeutics.

REFERENCE

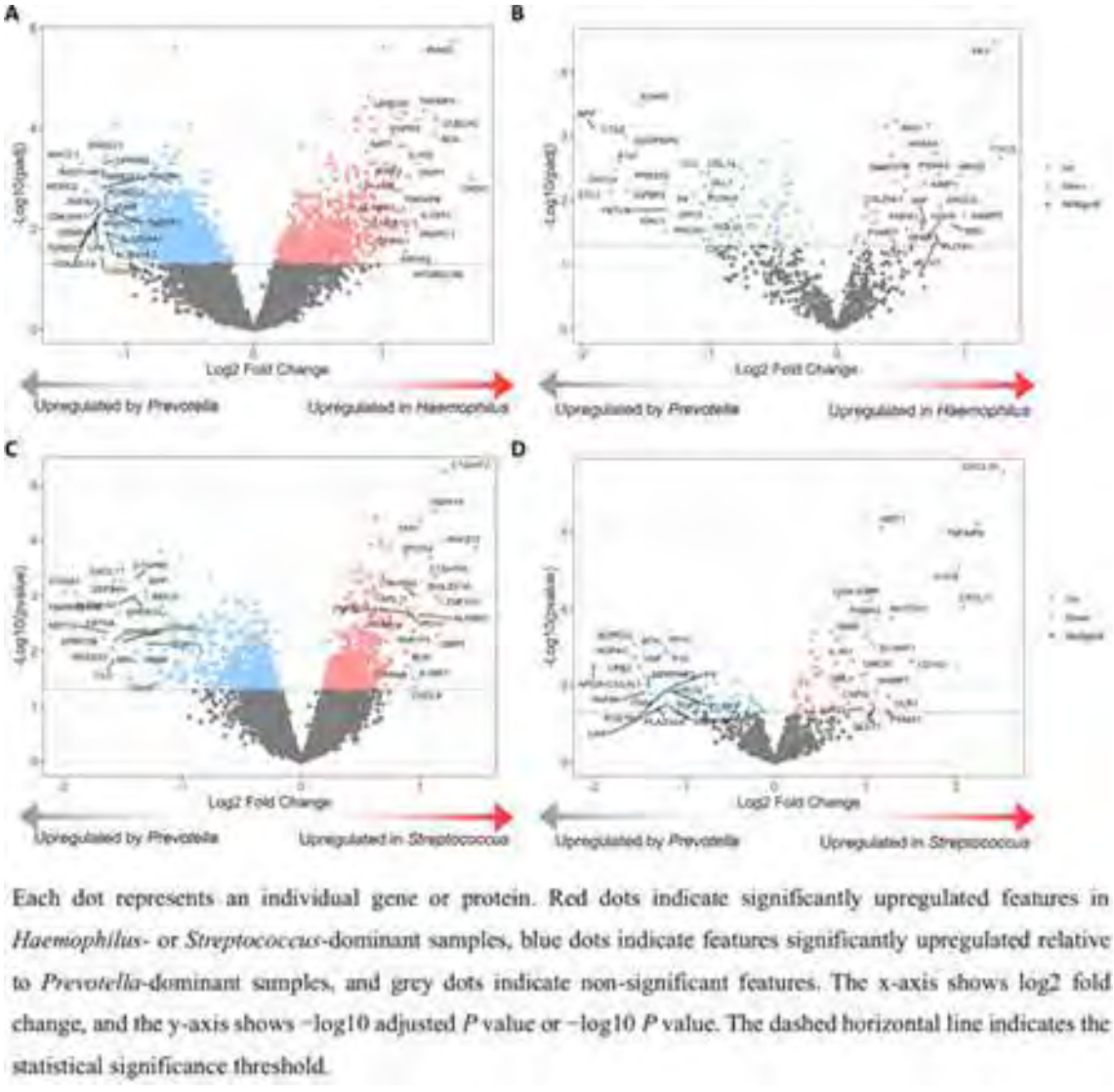
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S38 DISTINCT SPUTUM TRANSCRIPTOMIC AND PROTEOMIC SIGNATURES ASSOCIATED WITH AIRWAY BACTERIA IN SEVERE ASTHMA

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10.1136/thorax-2026-BTSabstracts.44

Background Potentially pathogenic bacteria (PPB) colonisation is associated with asthma exacerbations and disease severity, while *Prevotella* play a potentially protective role in asthma.[1] However, the host molecular responses associated with airway bacteria are not fully understood.



Abstract S38 Figure 1 Differential sputum transcriptomic and proteomic signatures associated with *Prevotella*-, *Haemophilus*-, and *Streptococcus*-dominant severe asthma (A) Sputum transcriptomic differences between *Haemophilus*- and *Prevotella*-dominant severe asthma; (B) Sputum proteomic differences between *Haemophilus*- and *Prevotella*-dominant severe asthma; (C) Sputum transcriptomic differences between *Streptococcus*- and *Prevotella*-dominant severe asthma; (D) Sputum proteomic differences between *Streptococcus*- and *Prevotella*-dominant severe asthma

Methods Clinical characteristics and sputum multi-omics data, including transcriptomics, proteomics, and microbiome profiling, were analysed from 100 adults with severe asthma enrolled in the pan-European U-BIOPRED cohort. Dominant bacterial taxa were defined as accounting for ≥20% of the total sputum microbiome based on 16S rRNA sequencing. Differential expression, functional enrichment, and weighted gene co-expression network analyses (WGCNA) were performed to characterise host molecular signatures associated with microbiome-dominant subgroups.

Results Among patients with severe asthma, sputum samples were most frequently enriched with *Prevotella* (46%), *Haemophilus* (23%), *Streptococcus* (14%), *Neisseria* (12%), *Veillonella* (6%), and *Moraxella* (3%). Patients with *Prevotella*-dominant microbiota demonstrated significantly lower PPB co-colonisation ($p<0.001$), reduced sputum neutrophils ($p<0.001$), better lung function (FEV_1, $p=0.03$), and fewer comorbid allergic rhinitis cases ($p=0.009$). In contrast, *Haemophilus*-dominant severe asthma was associated with longer disease duration ($p=0.02$), more frequent exacerbations ($p=0.049$), elevated C-reactive protein ($p=0.04$), and increased sputum neutrophils ($p=0.04$), but reduced sputum macrophages ($p=0.003$) and lymphocytes ($p=0.02$). Transcriptomic and proteomic analyses in *Haemophilus*-dominant severe asthma demonstrated marked upregulation of neutrophilic and innate immune pathways, including inflammatory mediators (*S100A12, SPP1, TNFAIP6* and *IL1R2*), bacterial sensing and innate immune response genes (*CLEC4E, SIGLEC14,* and *IRAK3*), and epithelial injury- and stress-associated proteins (*CYCS, SRC,* and *AIMP1*). *Streptococcus*-dominant samples exhibited enrichment of Th1/interferon-associated inflammatory responses, characterised by increased expression of *CXCL10, TNFAIP6, IL1R1,* and *PSMA2*.

Conclusions In severe asthma patients, distinct airway bacterial dominance patterns are associated with specific host inflammatory programmes in severe asthma. *Haemophilus*-dominant asthma was characterised by neutrophilic and innate immune inflammation, *Streptococcus*-dominant asthma by Th1/interferon-associated inflammation, whereas *Prevotella*-dominant asthma exhibited a comparatively less inflammatory profile.

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S39 LBP ELEVATION INDICATES GUT BARRIER DYSFUNCTION IN OUTPATIENT AND HOSPITALISED SEVERE ASTHMA EXACERBATIONS

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10.1136/thorax-2026-BTSabstracts.45

Introduction A substantial body of evidence recognises asthma as a systemic inflammatory disease involving extra-pulmonary sites. While research increasingly recognises the link between the gut microbiome and asthma pathophysiology, the role of intestinal barrier dysfunction during asthma exacerbations remains largely unexplored. We aimed to evaluate whether circulating biomarkers of intestinal permeability and microbial translocation including lipopolysaccharide-binding protein (LBP), soluble CD14 (sCD14), and REG3-α change during asthma exacerbations and correlate with clinical severity.

Methods Paired serum samples were analysed from 57 adults across two cohorts during outpatient exacerbations (OE, n = 28) and hospitalised exacerbations (HE, n = 29). Samples were collected during exacerbation and at clinical stability. Serum biomarkers of intestinal permeability and systemic inflammation were compared alongside pulmonary function across both clinical states.

Results In the combined analysis of both cohorts, serum LBP was significantly elevated during exacerbation compared to stable state (14.70 ± 8.07 μg/mL vs. 12.05 ± 5.26 μg/mL, $p = 0.003$). In the hospitalised cohort, median serum LBP was 13.5% higher at exacerbation than at stable state ($p = 0.041$), while in both cohorts sCD14 and REG3-α remained stable. Notably, a significant inverse correlation was identified between Δ LBP and Δ FEV_1 /FVC ratio ($r = -0.38$, $p < 0.01$). Linear mixed modelling identified BMI as a significant driver of overall LBP concentration ($p = 0.013$), independent of exacerbation state.

Conclusion This study provides novel evidence that intestinal barrier dysfunction may be a feature of the systemic pathophysiology of asthma exacerbations. Elevated LBP likely reflects transient systemic lipopolysaccharide exposure, implicating microbial translocation and intestinal permeability as potentially important factors in exacerbations. The correlation observed between LBP and airflow obstruction suggests gut-derived microbial translocation may amplify systemic inflammation and worsen respiratory function during exacerbation. Furthermore, elevated baseline LBP in higher-BMI patients suggests a pre-conditioned state of barrier dysfunction in this cohort. These data highlight the gut-lung axis as a potential therapeutic target for improving asthma control and managing exacerbations.

S40 CHARACTERISTICS AND MECHANISMS OF OBESITY-RELATED ASTHMA: A LARGE-SCALE MULTI-OMICS ANALYSIS

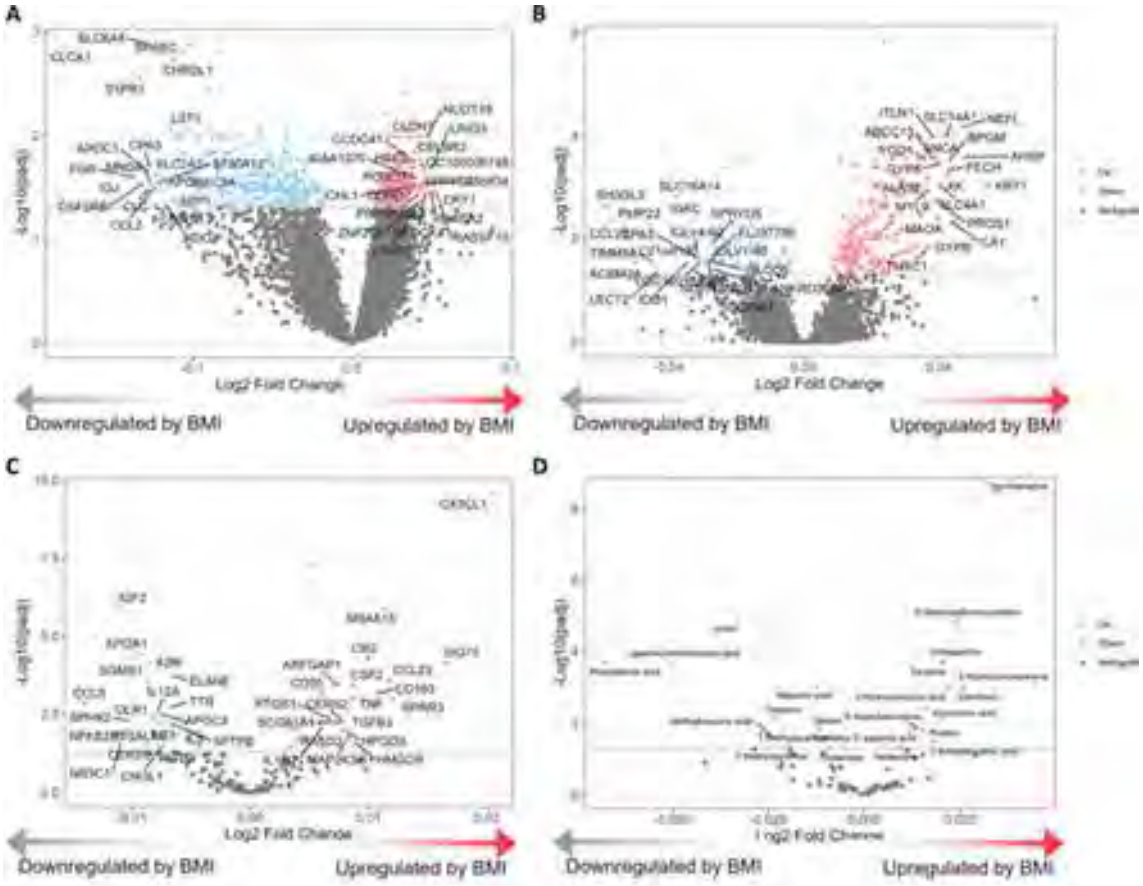
[1]J Shen, [2]N Zounemat-Kermani, [1]B Mappin-Kasirer, [3]C Bloom, [4]SE Dahlen, [1]I Pavord, [5]J Chalmers, [6]P Bradding, [3]I Adcock, [1]T Hinks. *[1]NIHR Oxford Biomedical Research Centre and Respiratory Medicine Unit, Nuffield Department of Medicine, University of Oxford, Oxford, UK; [2]Data Science Institution, Imperial College London, London, UK; [3]National Heart and Lung Institute, Imperial College London, London, UK; [4]Department of Respiratory Medicine, Karolinska University Hospital, Stockholm, Sweden; [5]Division of Molecular and Clinical Medicine, Ninewells Hospital and Medical School, University of Dundee, Dundee, UK; [6]Department of Respiratory Sciences, University of Leicester, Leicester Respiratory NIHR BRC, Glenfield Hospital, Leicester, UK*

10.1136/thorax-2026-BTSabstracts.46

Background Obesity-related asthma (ORA, BMI ≥30) often have more comorbidities, a poor response to corticosteroids, and more hospital admissions than normal-weight asthma. However, the clinical characteristics and mechanisms of ORA remain poorly understood.

Methods Clinical and multi-omics data (bronchial brush transcriptome, blood transcriptome, blood proteome, and urine metabolome) from 379 adult non-smokers with severe asthma in the pan-European U-BIOPRED cohort were analysed. Differential expression and pathway enrichment analyses were performed to identify molecular signatures associated with increasing BMI. Key findings were validated using the RASP-UK and Leicester bronchoscopy cohorts.

Results Among patients with severe asthma, 40% were obese, 33% overweight, 26% normal-weight, and 1% underweight, according to WHO BMI criteria. Compared with normal-weight asthma, ORA tended to have lower total lung capacity (5.7 vs 6.2, p=0.06) and higher ACQ-5 (2.5 vs 2.2, p=0.09), had significantly more diabetes (15% vs 2%, p=0.002), hypertension (43% vs 20%, p<0.001), gastro-oesophageal reflux disease (55% vs 41%, p=0.04) and psychiatric disorders (16% vs 6%, p=0.04), but less allergic rhinitis (46% vs 61%, p=0.03), despite similar medication prescribing patterns. ORA patients had higher blood neutrophils (5.5 vs 4.2, p=0.001), sputum macrophages (37 vs 16, p=0.007), C-reactive protein (4.0 vs 1.2, p<0.001), but lower blood eosinophil counts (0.20 vs 0.24, p=0.01) and periostin (52 vs 46, p=0.006). Bronchial brush transcriptomics showed reduced expression of epithelial and immune-related genes associated with higher BMI, including *CLCA1* and *S1PR1*. Blood transcriptomic analyses demonstrated downregulation of immune-associated genes, including *IGKC, CCL20,* and *PMP22*, while erythrocyte- and metabolic-related genes, including *ITLN1, SLC14A1,* and *BPGM,* were positively associated with BMI. In blood proteomics, increasing BMI was associated with upregulation of inflammatory proteins, particularly CX3CL1, ISG15, CCL23, and TNFα, with concurrent downregulation of protective and metabolic proteins, such as APOA1, IGF2, and SFTPB. Urine metabolomics identified significant BMI-associated alterations in amino acid and kynurenine metabolic pathways, including increased levels of saccharopine, kynurenic acid, and serotonin.



Volcano plots showing associations between body mass index (BMI, modelled as a continuous variable) and molecular features in asthma across (A) bronchial brush transcriptomics, (B) blood transcriptomics, (C) blood proteomics, and (D) urine metabolomics after adjustment for age, sex, and smoking status. Red dots indicate molecules positively associated with BMI, and blue dots indicate molecules negatively associated with BMI, while grey dots represent non-significant features. The x-axis represents log2 fold change, and the y-axis represents −log10(adjusted *P* value). Selected significantly associated genes, proteins, and metabolites are labelled. Positive log2 fold change values indicate upregulation with increasing BMI, whereas negative values indicate downregulation with increasing BMI.

Abstract S40 Figure 1 Differential molecular signatures associated with BMI in severe asthma across multi-omics

Conclusions ORA is a common and distinct clinical phenotype, characterised by greater comorbidity, immune-metabolic dysregulation and epithelial dysfunction, particularly upregulation of systemic CX3CL1, a distinct, asthma-relevant, non-Th2 immunological pathway.

S41 WHEN ASTHMA IS NOT ASTHMA: ALTERNATIVE TREATMENT DECISIONS AND SAFE CORTICOSTEROID WITHDRAWAL IN A COHORT WITH A PRIMARY-CARE DIAGNOSIS OF UNCONTROLLED ASTHMA

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10.1136/thorax-2026-BTSabstracts.47

Background Asthma is often misdiagnosed. Symptoms attributed to asthma may instead arise from an alternative diagnosis. The clinical value of comprehensive evaluation of these 'uncontrolled' patients in specialist centres is yet to be determined.
Hypothesis Comprehensive characterisation of patients referred with possible uncontrolled asthma identifies a substantial proportion with an alternative diagnosis, permits safe withdrawal of ICS, and reveals previously unrecognised co-existing conditions requiring treatment.
Methods Over 26 weeks and 5 visits, symptoms, inflammation and lung function were serially assessed in this prospective cohort study (NCT05357274).[1] Asthma was diagnosed using a multimodal diagnostic template.[2] ICS dosing and treatment of alternative diagnoses were determined by a physician at visits 2-5.
Results Two hundred and twenty participants completed the study. Following complete characterisation, 87 (40%) participants had asthma excluded and received an alternative diagnosis. At follow-up, ICS was successfully reduced or stopped in 66 (30%) participants. Throughout the study, 32 (15%) participants had ICS temporarily reduced or stopped to allow diagnostic features to emerge, with 1 (3%) of these participants having an exacerbation. ICS treatment decisions and treatment for previously unrecognised co-existing or alternative conditions are outlined in figure 1a and 1b.

Conclusion In patients referred to tertiary care with a clinical diagnosis of asthma, structured characterisation reclassified 40% as having an alternative diagnosis, permitted safe withdrawal of ICS, and uncovered a high burden of previously untreated co-existing conditions, particularly untreated upper airway diseases.

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S42 DETERMINING THE EFFECTIVENESS OF AN ONLINE FENO-GUIDED ASTHMA MANAGEMENT INTERVENTION IN PRIMARY CARE (DEFINE): A PRAGMATIC RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2026-BTSabstracts.48

Background The 2024 BTS/SIGN/NICE asthma guideline recommends that fractional exhaled nitric oxide (FeNO) should be used to help monitor treatment at routine asthma check-ups. Meta-analysis of previous trials suggests that FeNO-guided asthma management is more effective than conventional management at reducing acute exacerbations (https://doi.org/10.1016/j.resinv.2024.12.010). However, translating these findings into clinical practice is limited by variation in study population characteristics, intervention design, and definition of acute exacerbations.
Objective To assess whether routine asthma check-ups guided by FeNO are more effective than usual care at reducing acute exacerbations in adolescents and adults.

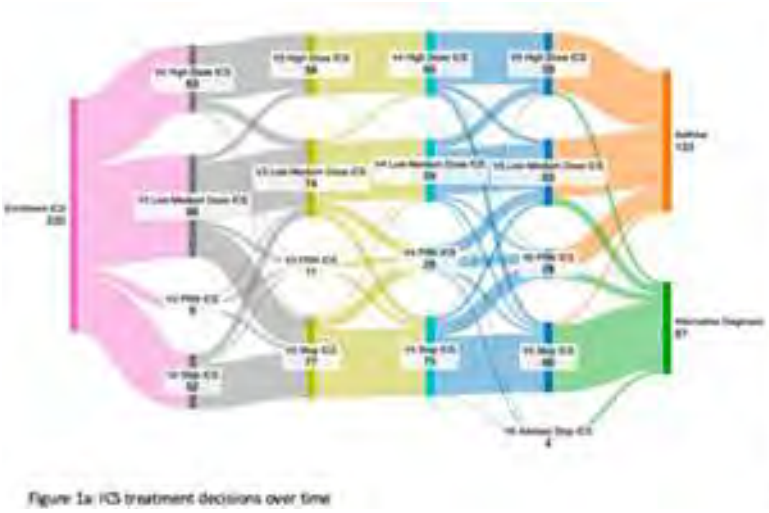


Figure 1a: ICS treatment decisions over time



Figure 1b: Treatment of co-existing and alternative conditions over time

Abstract S41 Figure 1

Methods We conducted a pragmatic randomised controlled trial including patients aged ≥12 years on general practice asthma registers who were prescribed maintenance inhaled corticosteroids or had ≥3 short-acting β_2-agonist prescriptions or ≥1 exacerbation in the last year. We excluded those on maintenance oral corticosteroids (OCS) or biologics. Patients were randomised 1:1 to the FeNO or control arm stratified by prior exacerbations in the last year.

A FeNO-guided clinical decision support system was used during routine asthma check-ups in the FeNO arm. The control arm received usual care. The primary outcome was the proportion of patients who had ≥1 asthma exacerbation requiring OCS, antibiotics, emergency department (ED) attendance or hospitalisation within a year. This study is registered on ISRCTN (ISRCTN16411887).

Results We randomised 2586 eligible patients from 22 August 2022 to 7 June 2024 from 120 general practices in England. Primary outcome data were obtained from 2476 (95.7%). In the FeNO arm 286/1247 (22.9%) had ≥1 primary outcome event vs 236/1229 (19.2%) in the control arm (adjusted odds ratio [OR] 1.27, 95% confidence interval [CI] 1.04-1.55, P=0.019). ED attendances and hospitalisations were similar in both arms (FeNO 17/1247,1.4%; control 19/1229,1.6%) but exacerbations treated with OCS and/or antibiotics were higher in the FeNO arm (283/1247,22.7% vs 236/1229,19.2%). Serious adverse events were similar in both arms and unrelated to the trial (OR 1.07, 95% CI 0.64-1.80).

Conclusion FeNO-guided asthma check-ups led to a significant increase in clinician-diagnosed acute exacerbations treated with OCS or antibiotics. This does not support routine use of FeNO during asthma check-ups.

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Occupational lung disease – focus on silicosis

S43 OCCUPATIONAL CAUSES OF INTERSTITIAL LUNG DISEASE, AND TRENDS IN SILICOSIS, FROM A SUPRA-REGIONAL UK OCCUPATIONAL LUNG DISEASE SERVICE, 2001-25

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10.1136/thorax-2026-BTSabstracts.49

Background Occupational attribution in interstitial lung disease (ILD) has evolved, and activity in supra-regional occupational lung services varies with staffing and referral patterns. UK HSE silica surveillance guidance introduced in 2015, recent pneumoconiosis compensation updates, and access to antifibrotic therapies may also have influenced reporting.

Methods Retrospective review of all cases (n=3165) assessed at a Birmingham supra-regional service, 2001-25, to include all ILD. Diagnoses were asbestosis, silicosis, hypersensitivity pneumonitis (HP), or other pneumoconioses. Annual silicosis counts were compared between 2001-15 and 2016-25 using hypothesis tests for non-parametric data. Negative binomial and multivariate logistic regression were used to model annual silicosis counts, proportions and test post-2015 step effects.

Results Of 646 ILD cases (median annual proportion=20%, IQR 11-40), mean age was 70 (SD=12) and 97% male. Asbestosis, HP and other pneumoconioses (including 60 silicosis) comprised 71%, 10% and 19% respectively, Median annual silicosis counts were higher in 2016-26 than 2001-15 (p=0.004). Silicosis counts increased ~7% per year (IRR=1.07, 95%CI =1.03-1.11; p=0.001), with no post-2015 step effect after year adjustment. Silicosis attribution was higher in 2016-25 after adjustment for asbestosis volume (aOR=3.13, 95%CI =1.3-7.8; p=0.014). Age did not differ by period. Construction accounted for a greater share of silicosis in 2016-26 (39% vs 14%; p=0.05), while metal industries declined (5% vs 46%; p=0.001). Among other non-silicosis pneumoconioses (n=68), cobalt hard metal, welding fume, mixed metal dust, wood dust and aluminium comprised 59%.

Conclusions Silicosis diagnoses increased over time, reflecting temporal trend with industry shift toward construction exposures.

S44 ARTIFICIAL STONE SILICOSIS IN THE UK: WHAT DO WE KNOW?

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10.1136/thorax-2026-BTSabstracts.50

Background Over 6000 cases of silicosis in worktop fabricators (WF) exposed to artificial stone (AS) are now reported globally. The first case was identified in the UK in 2023 in our service[1] and the number of cases continues to rise. No UK case finding programme is currently planned. In 2025, our occupational lung disease service set up a self-referral system so that WF who are concerned about silicosis could contact us directly [www.lungsatwork.org.uk].

Aim To describe the current cases of silicosis in WF from a single UK centre.

Methods We carried out a case note review of cases of silicosis in WF seen in the Royal Brompton Hospital occupational lung disease clinic.

Results Forty-nine cases have been identified to date (table 1) with mean age of 43 years (range 23-65). All cases worked for micro-companies (<15 employees), all had dry processed AS and only two had health surveillance despite the UK guidance. In total, 29% had accelerated disease (defined as <10 years exposure to respirable crystalline silica) suggesting very high exposures. Compared with patients with chronic silicosis (or >10 years exposure) this group were younger, all had been born outside the UK, the majority (79%) had only worked with artificial stone and most had stopped work with silica at the time of diagnosis. Overall, 14% have died (n=4) or are awaiting lung transplant (n=3). To date, 22 WF have been seen in our clinic via the self-referral system and of these 78% (n=17) were subsequently diagnosed with silicosis.

Abstract S44 Table 1 Characteristics of worktop fabricators with silicosis attending the royal brompton hospital

		Years of respirable crystalline silica exposure	
	Total (n=49)	**<10 years (n= 14 (29%))**	**>10 years (n= 35 (71%))**
Age in years (range)	43y (23-65)	30y (23-46)	46y (28-65)
Born in UK	19 (39%)	0	19 (54%)
Median duration of exposure (range)	20y (1-53)	4y (1-10)	22y (11-53)
Artificial stone exposure only	13 (27%)	11 (79%)	2 (6%)
Employment status at referral			
Employed exposed	19 (39%)	2 (14%)	18 (51%)
Employed unexposed	7 (14%)	2 (14%)	5 (14%)
Unemployed	22 (45%)	10 (71%)	12 (34%)
Referral route			
Respiratory	27 (55%)	6 (43%)	21 (60%)
Self	17 (35%)	8 (57%)	9 (26%)
Medicolegal	2 (4%)	0	2 (6%)
Occupational Health	2 (4%)	0	2 (6%)
GP	1 (2%)	0	1 (3%)
Smoking status			
Current	15 (31%)	6 (43%)	9 (26%)
Ex	26 (53%)	5 (38%)	21 (60%)
Never	8 (16%)	3 (21%)	5 (14%)
Radiology			
Simple silicosis	16 (33%)	3 (21%)	13 (37%)
Complicated silicosis	9 (18%)	1 (7%)	8 (23%)
Ground glass nodules	15 (31%)	8 (57%)	7 (20%)
Mediastinal lymphadenopathy only	9 (18%)	2 (14%)	7 (20%)
Active transplant list	3 (6%)	2 (14%)	1 (3%)
Deceased	4 (8%)	2 (14%)	2 (6%)

Conclusions Urgent action in the UK is needed to prevent silicosis in WF. This requires a multifaceted approach which includes a programme of education and ensuring workers are not exposed to high levels of respirable crystalline silica in the workplace. Findings from our clinic support the need for a proactive case finding programme to identify those with early disease enabling interventions to reduce morbidity and mortality in this young workforce.

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S45 WORK OUTCOMES AND SPIROMETRY IN PATIENTS WITH SILICOSIS IN THE NORTH WEST

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10.1136/thorax-2026-BTSabstracts.51

Background Cohorts of patients with silicosis from the UK are poorly described.

Occupational health (OH) surveillance is recommended by Health and Safety Executive (HSE) in those exposed to respirable crystalline silica (RCS) but this is not always carried out in practice at an individual level. RCS has been described as a cause of obstructive lung disease however the prevalence and incidence is unknown.

We aimed to cohort patients from a tertiary occupational lung disease (OLD) service to describe demographics, clinical management, and work outcomes.

Abstract S45 Table 1 Demographics, employment and clinical characteristics of patients with silicosis

Demographics, employment and clinical characteristics		**Patients**	**%**
Age at diagnosis (mean, SD)		56.6 ± 14.8	
Male sex		34	97
SWORD reported silicosis/pneumoconiosis (N=35)		35	100
Silicosis with PMF		8	23
Additional respiratory diagnoses	Asthma	7	20
	Emphysema/bronchitis/COPD	8	23
	Obstructive sleep apnoea	3	9
	Pleural plaques	1	3
	Diffuse pleural thickening	1	3
	History of pneumothorax	2	6
Employed (N=35)	Yes	27	77
	No retired	8	23
Continued RCS exposure (N=27)	Yes	22	81
	No - new role	5	19
Occupational health provisions available with continued RCS exposure (N=22)	Yes	9	41
	No - self employed	7	32
	No - other	1	5
	Data not available	5	23
Occupational health screening with continued RCS exposure (N=22)	Yes	7	32
	No - self employed	7	32
	No - other	3	14
	Data not available	5	23
Aware of others affected if exposed to RCS (N=22)	Not aware of others	2	9
	Self employed	7	32
	Data not available	6	27

Letter explaining mitigation of RCS exposure (N=22)	Yes	17	77
	Data not available	5	23
Spirometry performed (N=35)	Obstructive	22	63
	Normal	11	31
	Not performed	2	6
SWORD reported obstructive lung disease (N=22)		1	5
CT imaging interpreted by radiologist/multidisciplinary team (N=35)		35	100
Tuberculosis screening (N=35)	Yes	34	97
	No	1	3
Autoimmune/renal screening (N=35)	Yes	33	94
	No	2	6
Letter explaining diagnosis (N=35)		35	100
Letter with compensation advice (N=35)		35	100

SD = standard deviation; SWORD = Surveillance of Work-related and Occupational Respiratory Disease;
RCS = respirable crystalline silica

Methods We reviewed clinical records for a sample of silicosis patients seen within the OLD service from 2016-2026. We collected data on demographics, diagnoses, clinical investigations, spirometry and work outcomes.

Results *Demographics:*

35 patients with silicosis were identified and included. 34 (97%) were male and mean age at diagnosis was 56.5 ± 14.8 years. 8 of these patients had progressive massive fibrosis (23%).

Employment outcomes:

27/35 patients (77%) were employed. 22/27 (81%) in employment worked in an RCS exposed environment. Of patients with ongoing RCS exposure, 9/22 (41%) had access to OH, 7/22 (32%) had access to OH screening and 7/22 (32%) were aware of other employees exposed or affected.

Spirometry:

22/35 (63%) patients had airflow obstruction demonstrated on spirometry. 8/35 (23%) of patients had COPD recorded as a comorbidity and 1/35 (5%) had this reported as an occupational diagnosis.

Conclusions Our results suggest that over 3 in 4 patients with silicosis will continue to work in an RCS exposed environment despite their diagnosis and advice about removal from exposure. Most of these patients do not have OH provisions or screening despite HSE recommendations, potentially leading to missed or late diagnosis in other workers. Obstructive lung disease was common with over half demonstrating obstructive spirometry, however less than a quarter had a recorded diagnosis of COPD suggesting it was underreported in our sample. Only 5% had COPD reported as an occupational diagnosis despite its association with RCS exposure. Clinicians should be mindful in recognising that both pneumoconiosis and obstructive lung disease are consequences of occupational RCS exposure.

S46 AIRFLOW OBSTRUCTION AND CRYSTALLINE SILICA EXPOSURE IN A COHORT FROM A SUPRA-REGIONAL NHS OCCUAPTIONAL LUNG DISEASE SERVICE

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10.1136/thorax-2026-BTSabstracts.52

Introduction/Aim Silica exposure may cause airway disease without radiological silicosis. We hypothesised that airflow obstruction (AFO) in silica-exposed patients is not explained by smoking alone. We aimed to assess the frequency and occupational, physiological and radiological correlates of AFO in a UK regional occupational lung disease cohort.

Methods A retrospect study of 53 patients assessed for occupational silica-related disease at an NHS regional service (01/2016-05/2026). AFO was defined as FEV1/FVC below the LLN at diagnosis. Models were adjusted for age, smoking, Index of Multiple Deprivation (IMD) decile and body mass index (BMI).

Results The cohort was mainly male (51/53,96.2%), with median age 61.0 [53.0-69.0] years, exposure duration 32.0 [21.0-40.0] years, radiological silicosis/progressive massive fibrosis in 35/53(66.0%) and baseline silica-related chronic obstructive pulmonary disease in 8/53(15.1%). AFO was present in 23/51(45.1%). Obstructed patients had longer exposure (35.5 [30.0-41.8], 26.0 [17.0-35.0] years;p=0.022). Exposure ≥30 years increased AFO odds (60.7%, 26.3%; adj. OR 6.00,95% CI 1.33-27.17). FEF25-75 z-score was lower in obstructed patients (-1.88[-1.97– -1.28], -0.61 [-0.96-0.29];p<0.001). Dust control (17.4%, 50.0%; adj. OR 0.146,95% CI 0.025-0.844;p=0.032) and respiratory protective equipment use (17.4% vs 57.1%; adj. OR 0.101,95% CI 0.018-0.586;p=0.011) were less common in obstructed patients. AFO occurred in 6/15 neversmokers (40.0%).Mosaic attenuation correlated with lower FEF25-75 (rho=-0.375; p=0.012).

Conclusion AFO was common in this silica-related occupational lung disease cohort, particularly with longer exposure, small airway dysfunction and weaker workplace protection. Baseline silica-related COPD and possible asthma overlap should be considered when interpreting AFO frequency.

S47 UNDERSTANDING THE IMPACT OF OCCUPATIONAL EXPOSURES ON PULMONARY SARCOIDOSIS

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Background Sarcoidosis is a multisystem disorder characterised by granulomatous inflammation, affecting 1/10000 people in the UK. Silicosis is caused by the inhalation of silica dust and

may be indistinct from pulmonary sarcoidosis. Silica and other occupational exposures are associated with an increased risk of sarcoidosis.

Aims To cohort patients with pulmonary sarcoidosis by occupational exposures and describe clinical and radiological features.

Methods We conducted a retrospective observational study of all patients with pulmonary sarcoidosis in a tertiary service up to August 2025. Only male patients were included. Occupational information included: employment status; job title (if known); current/past work in a high-risk silica job; self-reported ever exposure to silica or beryllium. CASCOT coding was used to code major Standard Occupation Classification (SOC2010) code. Cohort demographics, other exposures, clinical, radiological and treatment features were described.

Results Of 205 patients, mean age at diagnosis was 42 (SD 12) years and 76% had white British ethnicity. Ninety-three (45%) were employed. No occupation was recorded in 19% (n=38).

Of 167 with a job title, 32 (19%) had worked in a high-risk job for silica exposure (table 1). Two were subsequently diagnosis with silicosis. The commonest SOC codes in high-risk sarcoidosis were skilled trades (n=21, 66%), process and plant operatives (n=6, 19%), and professional occupations (n=2, 6%). The commonest SOC codes in low-risk sarcoidosis were professional occupations (29, 21%), skilled trades (27, 20%), and elementary occupations (16%).

Abstract S47 Table 1

	High risk (n=32)	Low risk (n=135)	*P* value
Baseline characteristics			
Mean age at diagnosis (years ± S.D)	46 (±13.3)	46 (±13.1)	0.97
Currently employed	15 (47%)	78 (58%)	0.26
Ever-smoker	14 (44%)	51 (37%)	0.53
Extra-thoracic manifestations			
Any extra-thoracic manifestation	8 (25%)	65 (48%)	**0.02**
Splenomegaly	1 (3%)	0	**0.04**
Ocular	0	15 (11%)	**0.048**
Cardiac	0	11 (8%)	0.09
Skin	3 (9%)	26 (19%)	0.18
Hypercalcaemia	1 (3%)	13 (10%)	0.23
Neuro	0 (0%)	5 (4%)	0.27
Liver	3 (9%)	21 (15%)	0.37
Joint	1 (3%)	5 (4%)	0.87
Renal	1 (2%)	4 (3%)	0.96
Treatment			
Steroids, ever	15 (47%)	83 (61%)	0.13
Methotrexate	5 (16%)	37 (27%)	0.16
Mycophenolate	0 (0%)	7 (5%)	0.18
Meets infliximab criteria	0	9 (7%)	0.13
Azathioprine	1 (3%)	9 (7%)	0.44
Hydroxychloroquine	1 (3%)	9 (7%)	0.44
Antifibrotic	2 (6%)	10 (7%)	0.82

One quarter of high-risk patients (n=8, 25%) had extrapulmonary disease. There were more extra thoracic symptoms in low-risk jobs. Medical management was similar between the two groups.

Conclusion One in five workers in a tertiary sarcoidosis cohort did not have an occupational exposure history and a further one in five have worked in jobs with a high risk of silica exposure. Under recognition of occupational exposure may lead to misdiagnosis of silicosis as sarcoidosis with significant implications for patients. Further work is needed to explore causative links between silica and sarcoidosis.

S48 DIVERSE RADIOLOGICAL APPEARANCE OF WELDING-RELATED INTERSTITIAL LUNG DISEASE: EXPERIENCE FROM A SUPRA-REGIONAL NHS OCCUPATIONAL LUNG DISEASE SERVICE

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Background Welding fume exposure is associated with a range of chronic respiratory diseases, although the spectrum of welding-related interstitial lung disease (ILD) remains incompletely characterised. We reviewed diagnosed cases from our NHS occupational lung disease service to describe the radiological phenotypes associated with welding exposure.

Methods A retrospective review was undertaken of our clinical database containing N=3,184 patients assessed for occupational lung disease between January 2000 and April 2026. Cases were identified by searching industry, job title and causative agent for the keyword 'weld$'. Patients with airway disease alone (occupational asthma, COPD, rhinitis, metal fume fever and infective pneumonia) or asbestos-related disease were excluded. Clinical records, physiological data and imaging findings were reviewed to identify cases with interstitial or small airways lung disease attributable to welding fume exposure.

Results Twelve welders with non-malignant parenchymal or small airways disease were identified. All were male and worked predominantly in automotive, metalworking or manufacturing industries. Clinical presentations were heterogeneous and included bronchiolitis (n=3), acute pneumonitis (n=1), siderosis/haemosiderosis (n=4), aluminium-associated disease (n=2), mixed metal pneumoconiosis (n=1) and mixed dust fibrosis related to iron and silica exposure (n=1). Radiological appearances ranged from tree-in-bud nodularity and ground-glass change to diffuse dense micronodules, mixed dust fibrosis and pneumoconiosis-like patterns. Physiological abnormalities varied from normal lung function to severe fixed airflow obstruction and restrictive defects. Several cases demonstrated important co-exposures, including smoking, silica and other metal dusts.

Conclusions Welding-related ILD encompasses a broad spectrum of clinical and radiological phenotypes extending beyond classical siderosis. Recognition of diverse presentations and consideration of co-exposures are essential for accurate diagnosis and management of welding-associated lung disease.

Next generation asthma care

S49 MECHANISTIC INSIGHTS INTO STEROID CHRONOTHERAPY IN ASTHMA

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10.1136/thorax-2026-BTSabstracts.55

Introduction Asthma, a rhythmic disease, demonstrates peak airway narrowing and inflammation overnight. We have previously demonstrated that afternoon dosing (~4pm) (PM) with inhaled corticosteroids (ICS) was most effective in treating asthma compared to morning (~9am, AM) or twice daily (~9 am and ~9pm, BD) dosing.[1] To add to this study, peripheral blood mononuclear cells (PBMCs) were collected and analysed to investigate underpinning mechanisms. We hypothesise that immune cell pathways are more receptive to mid-afternoon steroids compared to morning or twice daily steroid dosing.

Methods RNA was extracted from PBMCs collected at 4am and 4pm, from 10 participants with mild/moderate allergic asthma at baseline (BL, following 2 weeks of ICS washout) and after each ICS treatment period (AM, PM, BD) for sequencing. Spirometry (forced expiratory volume in 1 second, FEV1) and blood eosinophil counts were measured every 6 hours (4am, 10am, 4pm and 10pm). Gene ontology enrichment analysis was performed against an external PBMC background (GSE248695).

Results Overnight FEV1 increased significantly after PM treatment compared to BL (figure 1A), and blood eosinophil counts were suppressed overnight after PM treatment compared to BD (figure 1B). PBMC transcriptomic analysis shows significant time-of-day changes in BL genes at 4pm compared to 4am (figure 2A), with interleukin 1 receptor type 2 (IL1R2) showing a higher expression at 4pm than 4am (figure 2A). Enriched pathways upregulated at BL 4pm include immune-mediated responses (figure 2B). Expression of PBMC clock genes shows changes by time-of day (figure 3).

Conclusions IL1R2 is known to dampen the inflammatory response and is upregulated by steroid.[2] Further work will determine if this significant factor changes with ICS treatment. For the first time in asthma, clock genes will be examined using ClinCirc in 3-hourly blood samples collected at BL, AM, PM and BD. This will advance our understanding of changes in the peripheral blood clock over time, and whether it is impacted by steroid chronotherapy.

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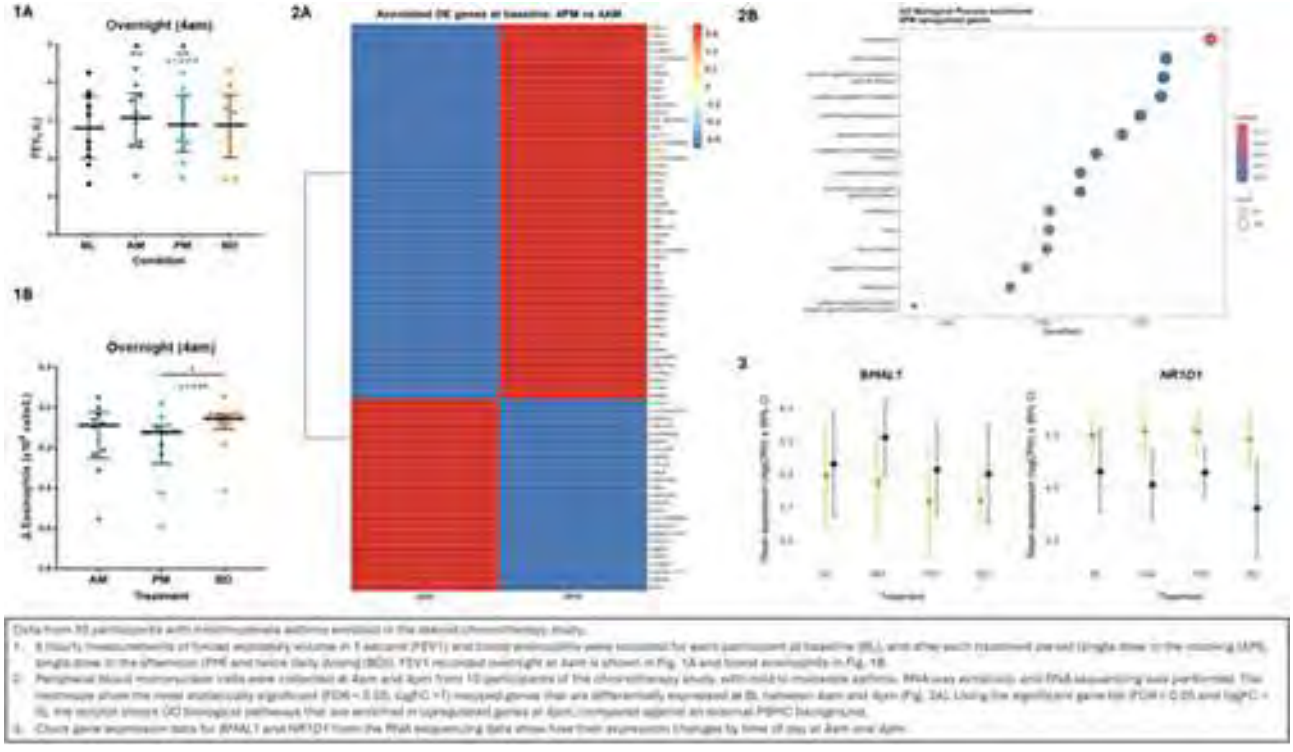
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S50 EXACERBATION TRAJECTORIES IN BIOLOGIC-NAÏVE SEVERE ASTHMA PATIENTS

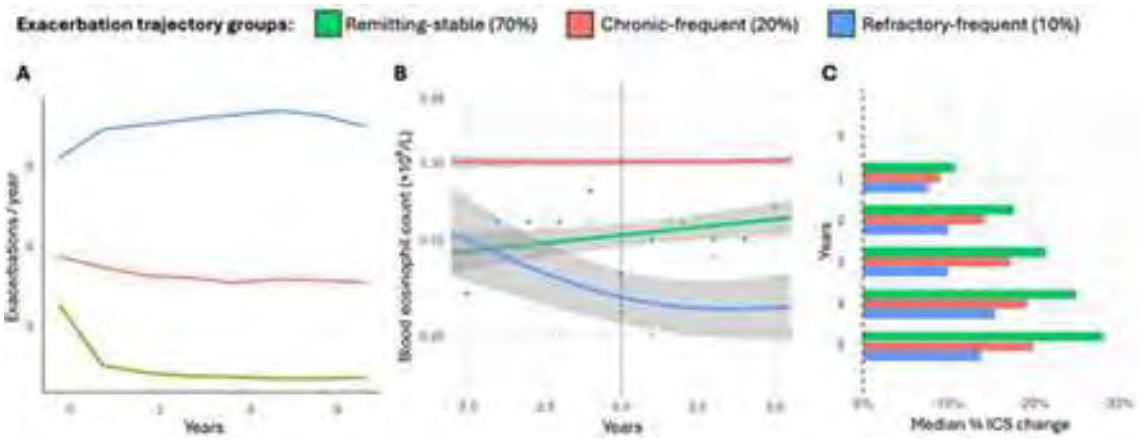
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Background Asthma biologics are highly effective in reducing exacerbations. Improvement in exacerbations after biologic initiation is often attributed to treatment effect. We hypothesised



Abstract S49 Figure 1



Abstract S50 Figure 1 Exacerbation trajectory groups with treatment and biomarker trends around biologic-eligible year (Year 0). (A) Exacerbation trajectories after Year 0: remitting-stable (70%) chronic-frequent (20%), refractory-frequent (10%); (B) Trends in highest annual blood eosinophil count (median per year) by trajectory group. (C) Median percentage change in inhaled corticosteroids (ICS) dose relative to Year 0 by trajectory group (negative values indicate dose reduction)

that a subset of severe asthma patients improves without biologics and determined their clinical characteristics.

Methods Biologic-naïve adults with severe asthma were identified in nationwide UK electronic medical records. Year 0 was defined as the first year patients met severe asthma criteria with ≥3 systemic corticosteroid-treated exacerbations (biologic-eligible). Post-eligibility exacerbation trajectories were identified using longitudinal k-means. Sensitivity analyses included alternative *k* and a lower exacerbation threshold (≥2/year). Covariates included the highest blood eosinophil count (BEC), inhaled corticosteroid (ICS) dose and comorbidities.

Results Among 10,078 biologic-eligible adults (median age 51 years; 71% female), we identified three exacerbation trajectories: (1) *Remitting-stable* (70%, n=7,091), with sustained improvement to <3 exacerbations/year even though ICS reduced after peak exacerbation year; (2) *Chronic-frequent* (20%, n=1,960), with persistently frequent exacerbations (median 5/year); (3) *Refractory-frequent* (10%, n=1,018), with high ongoing oral corticosteroid exposure (median >10 courses/year). Results were consistent across sensitivity analyses.

Remitting-stable patients were least likely to have bronchiectasis but were otherwise similar in demographics and allergic comorbidities. BEC was higher in the chronic-frequent group (Year 0 median 0.30×10^9/L) compared with remitting-stable (0.23×10^9/L) and refractory-frequent groups (0.21×10^9/L, $p<0.001$); this was evident several years before meeting biologic exacerbation criteria (figure 1).

Conclusion Over two-thirds of biologic-eligible patients improved with standard care alone. Chronic-frequent exacerbators had higher blood eosinophil counts, even years before meeting biologic criteria, suggesting a distinct disease phenotype.

S51 EXACERBATION-FREE PROPORTIONS IN PLACEBO ARMS OF ASTHMA BIOLOGIC TRIALS: A META-ANALYSIS AND META-REGRESSION OF PREDICTORS

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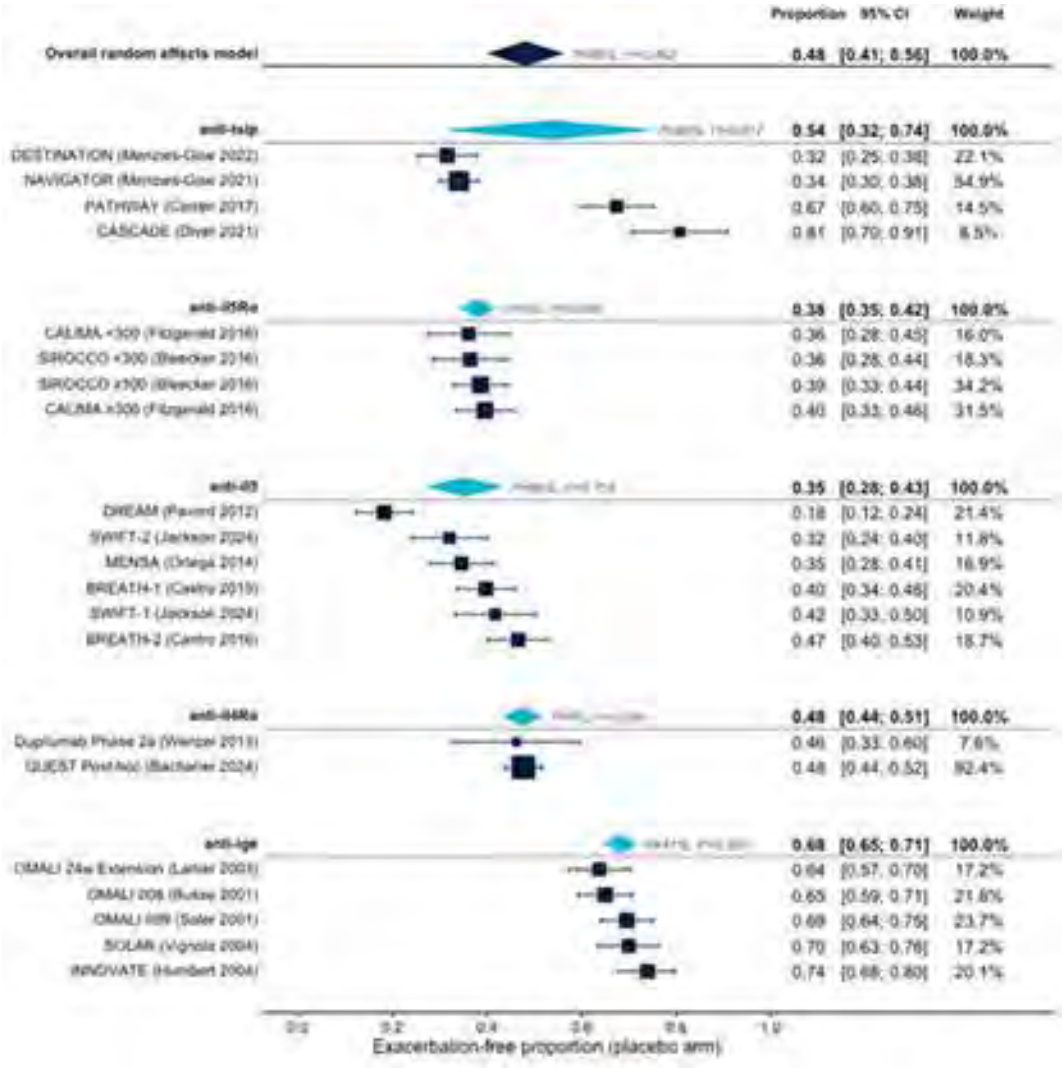
Background Asthma biologic RCTs often show substantial exacerbation reductions in placebo arms, reducing treatment contrast and statistical power. As biologic eligibility criteria evolve, this may increasingly affect trial design. However, the magnitude of placebo-arm improvement, and participant or trial characteristics that predict exacerbation-free outcomes, remain poorly understood.

Aim Determine the proportion of exacerbation-free participants in the placebo arms of biologic asthma RCTs, and their baseline characteristics.

Methods We identified placebo-controlled phase II/III RCTs of anti-IgE, anti-IL5/5Rα, anti-IL4Rα and anti-TSLP. The majority of included trials required a documented history of exacerbations for enrolment. Maintenance oral-corticosteroid-tapering and paediatric-only studies were excluded. Two reviewers independently extracted the exacerbation-free proportion and baseline covariates. Proportions were pooled by random-effects meta-analysis, with heterogeneity summarised by I^2. Subgroups were examined according to drug class, trial phase and blood-eosinophil entry criterion. Univariable meta-regression assessed baseline trial-level predictors, with odds ratios (OR) expressed per 10-unit increase for percentage of predicted FEV_1 and age, and per 1-unit increase for annualised exacerbation rate and ACQ score.

Results 4,647 placebo-treated participants from 19 RCTs (16 phase III, 3 phase II) were included: anti-IgE (5, n=1,161), anti-IL5 (6, n=1,086), anti-TSLP (4, n=833), anti-IL5Rα (4, n=777) and anti-IL4Rα (2, n=690). Median (range) follow-up was 52 (12-104) weeks. Overall, 48% of placebo-treated participants became exacerbation-free, higher in phase II than phase III (66% vs 45%). Anti-IgE trials had the highest proportions: 68% (95% CI 65–71) and anti-IL5 the lowest: 35% (28–43) (figure 1). RCTs with a blood eosinophil count ≥300cells/μL criterion had lower exacerbation-free proportions than those without (37% vs 57%). Higher baseline FEV_1 was associated with higher odds of exacerbation-free outcome (OR 2.14, 95% CI 1.42–3.22), whereas higher annualised exacerbation rate (0.64, 0.42–0.97), ACQ (0.50, 0.29–0.87) and older age (0.48, 0.27–0.84) were associated with lower odds.

Conclusion Approximately half of placebo-treated participants in asthma biologic RCTs became exacerbation-free during the study, diluting the effect size of the treatment. Enriching for



Abstract S51 Figure 1 Exacerbation-free proportions in the placebo arms of asthma biologic trials, stratified by drug class

patients with frequent exacerbations, higher symptom burden and lower FEV_1 may reduce placebo responses and protect trial power.

S52 EFFECT OF TEZEPELUMAB ON LUNG FUNCTION IN PATIENTS WITH OCS-DEPENDENT SEVERE ASTHMA BY BASELINE INFLAMMATORY BIOMARKER LEVELS: RESULTS FROM THE PHASE 3 SUNRISE STUDY

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10.1136/thorax-2026-BTSabstracts.58

Introduction and Objectives Tezepelumab is a human monoclonal antibody that blocks the activity of thymic stromal lymphopoietin (TSLP). In the phase 3 SUNRISE study (NCT05398263), tezepelumab treatment versus placebo led to greater reductions from baseline in daily maintenance oral corticosteroid (OCS) dose at week 28 in patients with severe, OCS-dependent asthma and improved pre-bronchodilator (BD) forced expiratory volume in 1 second (FEV_1) in the overall population, despite tapering of maintenance OCS use. This *post hoc* analysis evaluated the change from baseline in pre-BD FEV_1 at week 28 in SUNRISE patients grouped by high and low blood eosinophil count (BEC), fractional exhaled nitric oxide (FeNO) level or perennial aeroallergen-specific immunoglobulin E status (assessed by fluorescence enzyme immunoassay [FEIA]) at baseline.

Methods SUNRISE was a multicentre, double-blind, placebo-controlled, parallel-group study in patients aged 18–80 years with severe, OCS-dependent asthma. Following optimization of OCS dose, patients were randomized 2:1 to receive tezepelumab 210 mg or placebo subcutaneously every 4 weeks for 28 weeks. The primary endpoint was the categorized percentage reduction from baseline in daily maintenance OCS dose at week 28 while maintaining asthma control. The key second-

Abstract S52 Table 1 Change from baseline in pre-BD FEV1 at week 28, overall and by baseline biomarker subgroups

	Mean (SD) at baseline, L		LS mean (95% CI) change from baseline, L		LS mean (95% CI) difference versus placebo, L
	Tezepelumab 210 mg Q4W (N = 83)	Placebo (N = 39)	Tezepelumab 210 mg Q4W (N = 83)	Placebo (N = 39)	
Overall	1.60 (0.59)	1.53 (0.53)	0.24 (0.13, 0.35)	−0.03 (−0.17, 0.12)	0.26 (0.08, 0.45) Nominal $p = 0.0046$
Baseline BEC, cells/μL					
< 150	n = 14 1.51 (0.63)	n = 12 1.62 (0.63)	n = 14 0.00 (−0.26, 0.26)	n = 12 −0.21 (−0.51, 0.08)	0.21 (−0.19, 0.62)
≥ 150	n = 69 1.62 (0.59)	n = 27 1.49 (0.48)	n = 69 0.30 (0.18, 0.42)	n = 27 0.03 (−0.15, 0.20)	0.28 (0.06, 0.49)
< 300	n = 36 1.58 (0.57)	n = 20 1.56 (0.55)	n = 36 0.13 (−0.04, 0.30)	n = 20 −0.16 (−0.38, 0.06)	0.29 (0.00, 0.57)
≥ 300	n = 47 1.62 (0.62)	n = 19 1.50 (0.52)	n = 47 0.34 (0.20, 0.49)	n = 19 0.07 (−0.13, 0.27)	0.27 (0.02, 0.52)
Baseline FeNO level, ppb					
< 25	n = 31 1.57 (0.61)	n = 12 1.31 (0.46)	n = 31 0.17 (−0.01, 0.35)	n = 12 −0.06 (−0.33, 0.22)	0.23 (−0.10, 0.56)
≥ 25	n = 48 1.64 (0.56)	n = 24 1.68 (0.54)	n = 48 0.30 (0.16, 0.44)	n = 24 −0.01 (−0.18, 0.17)	0.30 (0.08, 0.53)
Baseline perennial aeroallergen-specific IgE status (FEIA)					
Any perennial FEIA positive	n = 37 1.62 (0.59)	n = 21 1.59 (0.60)	n = 37 0.20 (0.04, 0.37)	n = 21 0.02 (−0.18, 0.22)	0.19 (−0.06, 0.44)
All perennial FEIA negative	n = 45 1.59 (0.61)	n = 16 1.39 (0.42)	n = 45 0.28 (0.14, 0.43)	n = 16 −0.08 (−0.31, 0.14)	0.36 (0.10, 0.63)

Analysis performed using ANCOVA adjusting for treatment, visit (subgroup analyses only), baseline pre-BD FEV_1, region and log baseline BEC (plus subgroup variable–treatment interaction for subgroup analyses).

Baseline was defined as the last non-missing measurement on or before the date of randomization.

25 patients (20%) did not complete the study owing to early study termination due to recruitment challenges. Missing data were imputed using a multiple imputation approach assuming missing at random.

ANCOVA, analysis of covariance; BD, bronchodilator; BEC, blood eosinophil count; CI, confidence interval; FEIA, fluorescence enzyme immunoassay; FeNO, fractional exhaled nitric oxide; FEV_1, forced expiratory volume in 1 second; IgE, immunoglobulin E; LS, least-squares; Q4W, every 4 weeks; SD, standard deviation.

ary endpoint was the change from baseline in pre-BD FEV_1 at week 28.

Results Overall, 122 patients received treatment (tezepelumab, n = 83; placebo, n = 39). Tezepelumab treatment versus placebo resulted in a numerical improvement in mean pre-BD FEV_1 from baseline to week 28 in the overall study population (least-squares mean difference: 0.26 L [95% confidence interval: 0.08, 0.45]). Improvements were observed across all baseline BECs and FeNO levels, with greater improvements observed in subgroups with BECs of ≥150 cells/μL or FeNO levels of ≥25 ppb than those with lower BECs and FeNO levels, respectively. Improvements were also observed among both FEIA allergen subgroups evaluated, with greater improvements in FEIA-negative patients than in FEIA-positive patients (table 1).

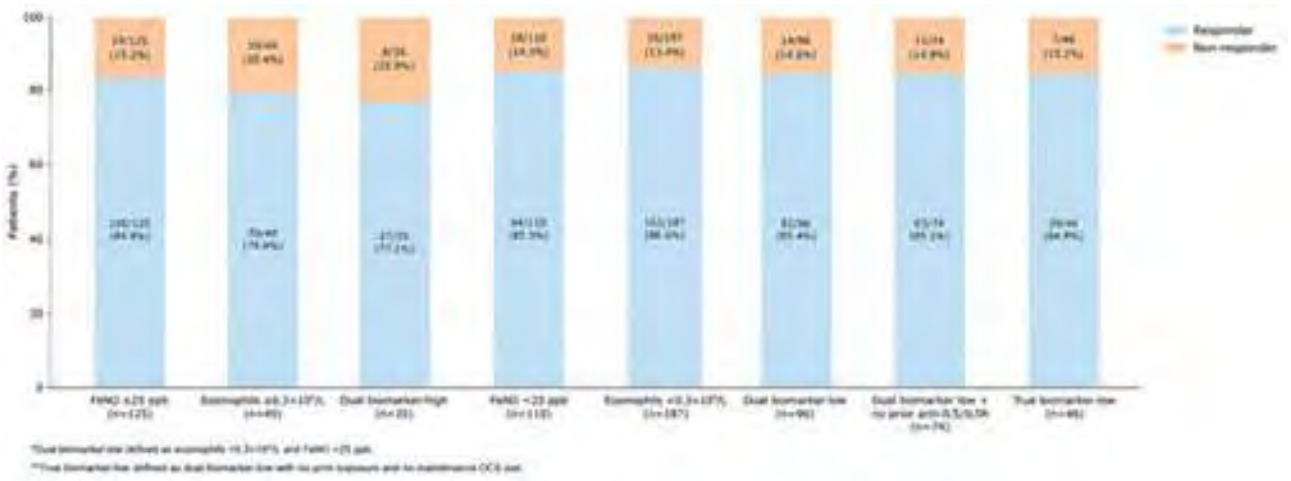
Conclusions Among patients with severe, OCS-dependent asthma, tezepelumab improved lung function versus placebo, despite tapering of maintenance OCS use, overall and across baseline BEC, FeNO level and perennial allergy status subgroups.

S53 CLINICAL RESPONSE TO TEZEPELUMAB IN A REAL-WORLD SETTING IS PRESERVED REGARDLESS OF BASELINE BIOMARKER STATUS OR PRESENCE OF COMORBIDITIES

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10.1136/thorax-2026-BTSabstracts.59

Introduction and Objectives While tezepelumab is approved for the treatment of severe asthma (SA) without specific type 2(T2) biomarker criteria, it is recognised that responses may



Abstract S53 Figure 1 Tezepelumab response across biomarker phenotypes

be attenuated in patients with T2-biomarker low disease. We assessed 12-month real-world outcomes in a heterogeneous SA cohort, including patients with low biomarker levels, prior biologic use and common comorbidities.

Methods We performed a retrospective single-centre analysis of patients treated with tezepelumab for SA. Response was assessed at 12 months using NICE criteria and defined as a ≥50% reduction in exacerbations and/or maintenance oral corticosteroid (mOCS) requirement. Baseline demographics, biomarkers and previous biologic exposure were compared between responders and non-responders. Dual biomarker-low disease was defined as blood eosinophils $<0.3\times10^9$/L and FeNO <25 ppb. Patients within this group with no previous anti-IL5/IL5R exposure and no mOCS use were classified as 'true' biomarker-low. Data analysis was performed using SPSS.

Results Of 236 patients, 201 (85.2%) were responders. Non-responders were older than responders (60.7±12.1 vs 53.9±14.8 years; p=0.010). BMI was similar between groups (31.1±8.6 vs 30.9±6.6; p=0.921). Baseline exacerbation frequency was statistically higher in non-responders, although median exacerbation burden was identical in both groups (4/year; mean 4.69 vs4.23; p=0.035). Baseline ACQ was similar in non-responders and responders (3.42±1.00 vs 3.21±1.12; p=0.315). Baseline mOCS use (34.3% vs 32.3%) and dose was similar between groups (10.58±2.64 vs 9.68±5.81 mg). Mood disorder (25.7% vs 25.9%), breathing pattern disorder (25.7% vs 20.9%), nasal polyps (11.4% vs 10.4%), and previous anti-IL5/IL5R exposure (45.7% vs 34.3%) were similar in non-responders and responders, respectively. Among patients that previously failed to respond adequately to anti-IL5/IL5R biologics, 69/85 (81.2%) responded to tezepelumab. Biomarker phenotype was not associated with outcome (p=0.518, figure 1). Ninety-six (40.9%) patients were dual biomarker-low, of which 82/96 (85.4%) were responders. Within this subgroup, response was high in those without previous anti-IL5/IL5R exposure (63/74, 85.1%) and in true biomarker-low patients (39/46, 84.8%).

Conclusion In this single-centre SA cohort, tezepelumab was associated with high response rates at 12 months that was independent of baseline T2 biomarker status, including dual biomarker low disease. The presence of comorbidities and mOCS use was not associated with reduced response.

S54 TWICE-YEARLY DEPEMOKIMAB DEMONSTRATES EFFICACY IN PATIENTS WITH ASTHMA AND COMORBID CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSWNP): PHASE III SWIFT-1/-2 STUDIES

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Rationale 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 SWIFT1/2, depemokimab significantly reduced exacerbations with sustained suppression of inflammation assessed by blood eosinophil count in patients with type 2 asthma. CRSwNP is a common asthma comorbidity, also often driven by type 2 inflammation. Here we evaluate depemokimab efficacy in reducing annualized exacerbation rate in the CRSwNP subpopulation from SWIFT-1/-2.

Methods Patients were randomized 2:1 to receive depemokimab 100 mg subcutaneously or placebo, plus standard of care, once every 26 weeks over 52 weeks. Annualized exacerbation rate was analyzed in patients with past or current CRSwNP at baseline at Week 52 (pre-specified).

Results At Week 52, among CRSwNP subpopulation, the annualized exacerbation rates were 0.51 (95% confidence interval [CI]: 0.33, 0.77; n=80) for depemokimab and 1.70 (95% CI: 1.02, 2.82; n=33) for placebo, with the depemokimab versus placebo rate ratio of 0.30 (95% CI: 0.15, 0.58; p<0.001). The exacerbation rate was reduced by 70% (95% CI: 42%, 85%) compared with a 54% reduction observed in the overall population of the SWIFT-1/-2 studies.

Conclusions Twice-yearly depemokimab, compared with placebo, reduced clinically significant asthma exacerbations in patients with type 2 asthma and comorbid CRSwNP. This reduction was greater than the exacerbation rate reduction observed in the overall asthma population of the SWIFT-1/-2 studies.

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Bronchiectasis: biology, biomarkers and better care

S55 MOLECULAR DETECTION REVEALS PATHOGEN-SPECIFIC INFLAMMATORY SIGNATURES IN BRONCHIECTASIS

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10.1136/thorax-2026-BTSabstracts.61

Introduction Infections in patients with bronchiectasis are linked to increased inflammation and exacerbation risk. We aimed to characterise infections in patients by Biofire® sputum PCR and determine whether pathogens are associated with distinct sputum inflammatory profiles.

Method Sputum from 489 stable patients with CT-confirmed bronchiectasis in the pan-European EMBARC-BRIDGE study were analysed by the BioFire® FilmArray® Pneumonia Plus Panel. *Pseudomonas aeruginosa* (n=43) and *Haemophilus influenzae* (n=35) BioFire® results were compared to available culture data. 45 inflammatory markers were measured by Olink® Target 48 (n=184).

Results 72% (351/489) of patients had at least 1 bacterial infection, of which 55% (267/489) had at least one high bacterial yield infection of $\geq 10^6$ copies/mL. The most common bacteria detected were *H. influenzae* (32%) followed by *P. aeruginosa* (24%), *Staphylococcus aureus* (13%), *Streptococcus pneumoniae* (8.4%), *Escherichia coli* (6.1%) and *Moraxella catarrhalis* (5.7%). At least 1 virus was detected in 27% (133/489) of sputum samples. AMR genes were found in 3% (13/476) of samples.

Molecular methods detected more infections than culture. 21% (9/43) of *P. aeruginosa* infections had no *P. aeruginosa* detected by culture at the time of sampling or in the previous 10 years, and 49% (17/35) of *H. influenzae* infections were missed.

Sputum inflammatory markers differed between infections. 24 inflammatory markers were significantly changed after FDR correction ($p<0.05$) in association with *P. aeruginosa* infections, including a decrease in CXCL10, CXCL9, CCL13, CCL19 and CCL11, and increased levels of CSF1, HGF, OSM, MMP1 and IL1β. 12 markers were associated with *H. influenzae* infections, including an increase in IL1β, TNF, IL10, CSF and IL18. There were no significantly altered markers linked to *S. aureus*, *E. coli* or *M. catarrhalis* infections. IFNγ was increased in patients with viral infections.

Conclusion Infections are common in patients with bronchiectasis, with a substantial proportion of *P. aeruginosa* and *H. influenzae* infections missed by standard culture. Distinct pathogens are associated with pathogen-specific inflammatory signatures, with *P. aeruginosa* linked to suppression of chemokines involved in chemotaxis and lymphoid trafficking and activation of pro-inflammatory pathways, while *H. influenzae* is associated with an IL-1β and TNF-driven inflammatory response.

S56 BRONCHOALVEOLAR LAVAGE PROTEOMICS IN BRONCHIECTASIS AND PROTRACTED BACTERIAL BRONCHITIS PROVIDES NOVEL INSIGHT INTO AIRWAY INFLAMMATION

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10.1136/thorax-2026-BTSabstracts.62

Introduction Proteases, particularly cathepsin C/DPP-1, have been successfully targeted in bronchiectasis, however the role of other cathepsins and neutrophil proteins are not fully understood. Bronchoalveolar lavage (BAL) proteomic analysis was used aiming to completely profile neutrophil responses in potentially early bronchiectasis and established disease.

Methods Label-free LC-Tandem MS was performed on BAL from 62 individuals. Differential expression was analysed by FDR-adjusted linear models, followed by pathway analysis.

Results 62 individuals were included (bronchiectasis n=42, mean age±SD 63.7±11.3 years, 59.5% female; controls n=10, 65.8±3.16 years, 50% female; protracted bacterial bronchitis (PBB) n=10, 60.3±5.31 years, 70% female).

Significant differences were observed in BAL between controls and bronchiectasis patients, with 201 differentially abundant proteins (58 increased in bronchiectasis and 143 decreased). Interestingly, comparing bronchiectasis and PBB as late and potential early disease populations, there were fewer differences, with 28 differentially abundant proteins (15 increased in bronchiectasis and 13 decreased).

Compared with controls, bronchiectasis patients had reduced abundance of GSTA1, PARK7, PRDX5, SOD1, and GSTP1 (FDR-adjusted $p<0.05$), related to protease inhibition and oxidative stress defences. Bronchiectasis patients showed enrichment of immune activity pathways, including neutrophil-mediated immunity, leukocyte migration, cell killing, ROS metabolism, and defence against bacteria/fungi, with cathepsin Z, cathepsin G, neutrophil elastase, myeloperoxidase, and calprotectin (S100A8/A9) all increased (FDR-adjusted $p<0.05$). All these proteins were increased in bronchiectasis vs PBB,

except for cathepsin Z which was not significantly different, and higher relative abundance of epithelial-derived proteins including ICAM-1 and Surfactant protein D were identified in PBB.

In bronchiectasis, cathepsin Z expression was positively associated with 31 proteins, including MUC5AC, MUC16, LYZ and VIM, and negatively associated with 11 proteins, including PKM and HPX (FDR-adjusted $p<0.05$).

Conclusion In bronchiectasis we identified increased neutrophilic inflammation compared with control and with PBB as a potential early disease cohort. Amongst these pro-inflammatory signatures, cathepsin Z was identified as a previously unstudied differentially expressed protein in Bronchiectasis vs Controls and vs PBB. Cathepsin Z is expressed in several immune cell types and type 1 alveolar cells, with both pro- and anti-inflammatory roles, warranting further investigation to validate these results quantitatively and understand cathepsin Z function in disease.

S57 CROSS-COMPARTMENT INFLAMMATORY PROGRAMMES IDENTIFIED BY MULTI-OMIC INTEGRATION ARE ASSOCIATED WITH DISEASE SEVERITY AND INFECTION IN BRONCHIECTASIS: RESULTS FROM EMBARC-BRIDGE

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10.1136/thorax-2026-BTSabstracts.63

Background The molecular mechanisms underlying bronchiectasis severity remain incompletely understood. Inflammatory signalling spans both the airways and systemic circulation, requiring a multi-compartment approach to identify disease-relevant immune programmes.

Methods Adults with CT-confirmed bronchiectasis from the EMBARC-BRIDGE observational study and matched healthy controls were enrolled. RNA-stabilised peripheral blood samples underwent mRNA sequencing, DESeq2 was used to identify differentially expressed genes, and weighted gene co-expression network analysis (WGCNA) was employed to investigate gene modules associated with clinical parameters. CIBERSORTx was used to impute immune cell proportions. Multi-Omics Factor Analysis (MOFA2) integrated blood transcriptomics (top 5,000 most variable genes) with serum Olink proteomics (n=155), sputum Olink proteomics (n=156), a sputum MSD cytokine panel (n=163), and eight neutrophil-related sputum immunoassays (n=250). Associations with clinical variables were tested using multivariable linear models adjusted for age, sex, batch, and site, with FDR correction.

Results Participants with bronchiectasis (n=535; 57% female, age 63.4±15.2 years) were compared with healthy controls (n=52; 53.8% female, age 62.7±15.7 years). mRNAseq analysis demonstrated significant differences in peripheral blood leukocyte transcriptomes in bronchiectasis, including enrichment of pathways relating to microRNA-regulated gene silencing and downregulation of metabolic pathways including oxidative phosphorylation. WGCNA identified gene modules associated with clinical features, including Bronchiectasis Severity Index (BSI), with greater disease severity associated with reduced expression of adaptive immune pathways such as B-cell activation.

Multi-omics integration identified five coordinated latent factors. Factor 2 showed the strongest association with BSI (FDR=0.002), and was also associated with prior *Pseudomonas aeruginosa* infection (FDR<0.001) and CT mucus plugging (FDR=0.026). Leading proteomic contributors included sputum neutrophil proteins (azurocidin-1, proteinase 3, olfactomedin 4, neutrophil elastase) and serum cytokines and chemokines (IL-6, IL-8, CXCL9, oncostatin M). Transcriptomic drivers included microRNAs and adaptive immune genes (MIR181A1, IGF1, COL1A1, IGHD, IGLL5), consistent with severity-stratified differential expression findings.

Conclusions Peripheral blood leukocyte transcriptomics demonstrated significant changes in systemic immune responses in bronchiectasis. Multi-omic integration further identified a coordinated inflammatory signature spanning airway and systemic compartments, associated with bronchiectasis severity, *P. aeruginosa* infection, and mucus burden.

S58 PATIENT-REPORTED SPUTUM COLOR AND ITS ASSOCIATION WITH NEUTROPHIL SERINE PROTEASES AND EXACERBATIONS IN PATIENTS WITH BRONCHIECTASIS: A POST HOC ANALYSIS FROM THE ASPEN TRIAL

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10.1136/thorax-2026-BTSabstracts.64

Introduction and Objectives Sputum purulence reflects the presence and activity of airway neutrophils; sputum color is considered an indicator of bronchiectasis disease activity, severity, and inflammation. Brensocatib, approved in the US, EU, and UK for treatment of non-cystic fibrosis bronchiectasis in patients ≥12 years (EU, UK: with ≥2 pulmonary exacerbations [PEx] in ≤12 months), targets neutrophilic inflammation by reducing neutrophil serine protease (NSP) activation. We report the association of the Bronchiectasis Exacerbation and Symptom Tool (BEST) sputum purulence score with NSP activity (neutrophil elastase [NE], cathepsin G [CatG], and proteinase 3 [PR3]), and the effect of brensocatib by baseline purulence and during PEx in the ASPEN trial (NCT04594369).

Methods Adults with ≥2 PEx in the 12 months pre-screening were randomized 1:1:1 to once-daily brensocatib (10mg or 25mg) or placebo for 52 weeks. Patients completed the BEST diary, which evaluates breathlessness, cough, cold/flu symptoms, sputum volume, sputum purulence, and fatigue, daily from screening through end-of-study. Sputum purulence was assessed on a 6-point scale and aligned post hoc to Murray chart categories: clear/white=mucoid; yellow=mucopurulent; green/dark green/blood=purulent. Sputum for NSP assessments

Abstract S58 Table 1 Sputum neutrophil elastase percentage reductions from baseline at week 52

	Brensocatib 10 mg	Brensocatib 25 mg	Placebo
Mucoid[a]	12.0%	42.8%	0.0%
Mucopurulent[a]	26.8%	32.7%	20.4%
Purulent[a]	58.8%	45.6%	13.6%

[a]n for brensocatib 10 mg, brensocatib 25 mg, and placebo: mucoid: 36, 29, 29; mucopurulent: 33, 43, 33; purulent: 11, 13, 21.

was collected in a subset of patients at screening, before baseline dosing, and at weeks 4, 16, 28, 40, 52, and 56 (4 weeks posttreatment).

Results In a pooled analysis across treatment arms (n=326 patients), 118 (36.2%), 143 (43.9%), and 65 (19.9%) patients with sputum NSP data reported mucoid, mucopurulent, and purulent sputum, respectively, at baseline. Increased sputum purulence was associated with higher sputum NE, CatG, and PR3 activity at baseline and across the treatment period. Greater baseline sputum purulence was associated with greater disease activity. Treatment with brensocatib was associated with greater reductions in NE vs placebo across all purulence categories (table 1), with similar trends for CatG and PR3.

Regardless of PEx severity, sputum purulence increased across treatment arms during PEx; however, brensocatib 10mg and 25mg reduced sputum purulence scores vs placebo during severe PEx.

Conclusions Greater BEST sputum purulence scores reflected increased NSP and disease activity at baseline. Brensocatib treatment was associated with reduced NSP activity across all purulence categories and reduced sputum purulence scores during severe PEx vs placebo.

S59 THE BRACE STUDY: A MULTICENTRE UK AUDIT OF MULTIDISCIPLINARY BRONCHIECTASIS CARE

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10.1136/thorax-2026-BTSabstracts.65

Background Bronchiectasis management requires systematic multidisciplinary assessment including evaluation of aetiology, disease severity and complications. While national guidelines exist, recent contemporary multicentre data on adherence to recommended investigations and care processes are limited. The BRACE (BRonchiectasis Audit of Care Evaluation) study aimed to evaluate real-world care across UK centres and to explore variation between higher- and lower-volume services.

Methods BRACE was a trainee-led INSPIRE multicentre audit conducted across 19 UK centres, including 9 large-volume (>50 cases) and 10 smaller centres. Adults with bronchiectasis were included. Data collected comprised demographics, comorbidities, aetiological testing, microbiology, treatment, exacerbation and hospitalisation frequency, and multidisciplinary care input. Centre-level data on service provision were also recorded.

Results A total of 1,031 patients were included (mean age 69.0 years; 63.3% female). Patients had significant disease burden, with a median of 2 exacerbations per year (IQR 0–4), 22.2% requiring intravenous antibiotics and 8.2% hospitalised in the preceding 12 months. Microbiological burden was substantial, with chronic *Pseudomonas aeruginosa* infection in 18.2% and non-tuberculous mycobacteria in 2.5%; 36.6% were receiving long-term macrolide therapy.

Adherence to guideline-recommended investigations was variable. Although improvements in aetiological testing were observed compared with the 2017 BTS audit, incomplete immunological and microbiological assessment remained common. Marked variation in multidisciplinary care was identified. Larger centres demonstrated greater access to physiotherapy input (77.3% vs 59.9%), more frequent reassessment of airway clearance (46.4% vs 29.0%), and higher use of written self-management plans (29.4% vs 15.8%). Smaller centres reported greater reliance on telephone review (12.5% vs 7.1%).

Notably, overall use of written self-management plans was lower than in the 2017 BTS audit (25.7% vs 40.9%, p<0.001).

Conclusions The BRACE audit demonstrates substantial disease burden and ongoing gaps in delivery of guideline-recommended bronchiectasis care across the UK. Significant variation exists between centres, particularly in access to multidisciplinary services. These findings highlight key priorities for quality improvement, including standardisation of aetiological testing and expansion of multidisciplinary care pathways to optimise outcomes for patients with bronchiectasis.

S60 EFFECT OF BRENSOCATIB ON PATIENT-REPORTED SYMPTOMS IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: A POST HOC ANALYSIS OF QOL-B RSS INDIVIDUAL ITEMS FROM THE ASPEN PHASE 3 TRIAL

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10.1136/thorax-2026-BTSabstracts.66

Introduction and Objectives Brensocatib is an oral, competitive, and reversible dipeptidyl peptidase-1 inhibitor that targets neutrophilic inflammation and is approved in the US, EU, and UK for the treatment of non-cystic fibrosis bronchiectasis (hereafter, bronchiectasis) in patients ≥12 years (EU, UK: with ≥2 pulmonary exacerbations [PEx] in ≤12 months). In the randomized ASPEN trial (NCT04594369), brensocatib 10mg and 25mg significantly reduced exacerbation burden over 52 weeks

Abstract S60 Table 1

Change From Baseline in QOL-B Respiratory Symptom Domain Item Scores at Week 52

Symptoms	Brensocatib 10 mg once daily (n=566)	Brensocatib 25 mg once daily (n=559)	Placebo (n=555)	Brensocatib 10 mg vs placebo	Brensocatib 25 mg vs placebo
	LS mean change (SE)			LS mean difference (95% CI)	
Congestion	0.367 (0.038)	0.521 (0.038)	0.369 (0.040)	-0.002 (-0.110 to 0.107)	0.152 (0.043-0.261)
Cough	0.209 (0.034)	0.355 (0.036)	0.195 (0.034)	0.014 (-0.080 to 0.107)	0.160 (0.063-0.256)
Sputum production	0.307 (0.038)	0.345 (0.039)	0.275 (0.038)	0.032 (-0.073 to 0.136)	0.070 (-0.037 to 0.176)
Sputum color	0.286 (0.035)	0.281 (0.034)	0.153 (0.034)	0.132 (0.036-0.229)	0.128 (0.034-0.221)
Wheezing	0.180 (0.035)	0.255 (0.032)	0.144 (0.032)	0.036 (-0.057 to 0.130)	0.111 (0.022-0.200)
Dyspnea during activity	0.216 (0.038)	0.224 (0.035)	0.096 (0.037)	0.119 (0.014-0.224)	0.127 (0.027-0.227)
Nocturnal cough	0.143 (0.036)	0.142 (0.033)	0.099 (0.033)	0.043 (-0.053 to 0.140)	0.043 (-0.049 to 0.135)
Chest pain	0.055 (0.028)	0.123 (0.026)	0.008 (0.030)	0.047 (-0.034 to 0.127)	0.115 (0.038-0.191)
Dyspnea while talking	0.070 (0.032)	0.106 (0.032)	-0.062 (0.033)	0.132 (0.041-0.222)	0.169 (0.078-0.259)

Change From Baseline in QOL-B RSS by On-Study Pulmonary Exacerbations at Week 52

Patients	Brensocatib 10 mg once daily		Brensocatib 25 mg once daily		Placebo		Brensocatib 10 mg vs placebo	Brensocatib 25 mg vs placebo
	n	LS mean change (SE)	n	LS mean change (SE)	n	LS mean change (SE)	LS mean difference (95% CI)	
No PEx	280	9.360 (1.002)	277	10.377 (1.029)	236	6.261 (1.088)	3.099 (0.184-6.014)	4.115 (1.167-7.063)
Any PEx	286	4.280 (1.142)	282	6.691 (1.082)	319	3.903 (1.014)	0.377 (-2.618 to 3.371)	2.788 (-0.116 to 5.692)
Any severe PEx	61	6.762 (2.807)	62	9.675 (2.469)	74	3.053 (2.113)	3.710 (-3.148 to 10.567)	6.622 (0.249-12.996)

PEx, defined as ≥3 symptoms (increased cough; increased sputum production or changed sputum consistency; increased sputum purulence; increased breathlessness and/or decreased exercise tolerance; fatigue and/or malaise; or hemoptysis) for ≥48 hours resulting in the need for systemic antibiotics, were adjudicated by a blinded independent committee. Severe PEx were those requiring IV antibiotics and/or hospitalization. Analysis was based on a linear repeated measures model including treatment group, visit, treatment-by-visit interaction, sputum sample being classified as positive or negative for *Pseudomonas aeruginosa* at screening visit, the number of pulmonary exacerbations (<3 or ≥3) in the previous 12 months, and stratification region (North America, Europe, Japan, and rest of world) as fixed effects, and baseline value as covariate. Higher scores represent better functioning and HRQOL.
HRQOL, health-related quality of life; LS, least squares; PEx, pulmonary exacerbations; QOL-B RSS, Quality of Life-Bronchiectasis Respiratory Symptom Score.

vs placebo in patients with bronchiectasis; brensocatib 25mg significantly reduced lung function decline and nominally significantly improved patient-reported symptoms as measured by the Quality of Life-Bronchiectasis Respiratory Symptoms Score (QOL-B RSS). In this post hoc analysis of ASPEN, we report results from the analysis of QOL-B RSS by item level and by on-study PEx.

Methods Adult patients with ≥2 PEx in the 12 months before screening were randomized 1:1:1 to once-daily 10mg or 25mg brensocatib or placebo for 52 weeks. QOL-B was completed by patients at baseline and every 2 weeks through end of trial. Nine items from the respiratory symptom domain were scored, assessing key respiratory symptoms of bronchiectasis (congestion, cough, sputum production, sputum color, wheezing, dyspnea during activity, nocturnal cough, chest pain, and dyspnea while talking). Analyses by on-study PEx status (0, any PEx, or any severe PEx) were also conducted.

Results Across QOL-B RSS items, improvements in symptoms from baseline were observed during the 52-week treatment period, particularly with brensocatib 25mg (table 1). At week 52, the greatest differences for brensocatib 25mg vs placebo were observed for cough, congestion, dyspnea, and sputum color (table 1). Patients with on-study PEx exhibited a greater symptom burden (lower QOL-B RSS) overall vs patients without PEx. Improvements in symptoms were observed with brensocatib vs placebo regardless of the number and severity of on-study PEx (table 1).

Conclusions Brensocatib, particularly the 25mg dose, improved respiratory symptoms vs placebo across all QOL-B RSS items, including cough, congestion, dyspnea, and sputum color, the latter a validated marker associated with neutrophilic inflammation. The effect of brensocatib on symptoms was observed regardless of on-study PEx. Results were consistent with overall ASPEN results, supporting clinical benefit of brensocatib on PEx and lung function.

ILD: risk prediction

S61 SPO2 RECOVERY TIME POST 6MWT IMPROVES RISK STRATIFICATION IN INTERSTITIAL LUNG DISEASE

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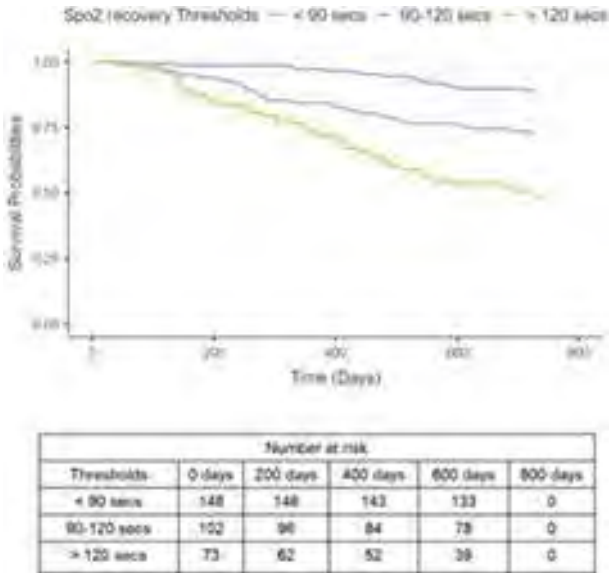
10.1136/thorax-2026-BTSabstracts.67

Introduction Six-minute walk test (6MWT) parameters are widely used to assess prognosis in interstitial lung disease (ILD). While exercise-induced desaturation and six-minute walk distance (6MWD) are established predictors of outcome, the prognostic significance of oxygen saturation (SpO_2) recovery time following exercise remains less well established and is not incorporated into current 6MWT-derived risk scores, including distance-oxygen gender age physiology (DO-GAP) and oxygen flow rate, distance, dyspnoea and saturation (ODDS).

Aims Evaluate the prognostic value and risk stratification value of SpO_2 recovery time following 6MWT and determine whether it provides incremental prognostic information beyond existing 6MWT derived risk models.

Methods Retrospective cohort study of 323 patients with ILD undergoing 6MWT at Royal Papworth Hospital NHS Foundation Trust. SpO_2 recovery time was defined as the time taken for oxygen saturation to return to within 2% of baseline following 6MWT. Associations with 1- and 2-year mortality or lung transplantation were assessed using Receiver operating curve analysis (ROC), Kaplan–Meier survival analysis, and Cox proportional hazards regression. Incremental prognostic value was evaluated by incorporating SpO_2 recovery time into DO-GAP and ODDS models using C-index, Akaike information criterion (AIC), and likelihood ratio testing.

Results SpO_2 recovery demonstrated good discrimination for 1- and 2-year outcomes (AUC 0.75 and 0.73, respectively). At 2 years Kaplan-Meier pairwise log-rank comparisons demonstrated significant differences between all recovery time thresholds (<90 vs 90–120 sec, p<0.001; <90 vs >120 sec, p<0.001; 90–120 vs >120 sec, p=0.001) with the greatest separation was seen in patients with recovery times >120 seconds. (see figure 1). In multivariate survival analyses, TLCO (HR 0.94, 95% CI 0.92–0.96, p<0.001), FVC (HR 0.98, 95% CI 0.97–1.00, p=0.013), and SpO_2 recovery time >120 seconds (HR 2.07, 95% CI 1.02–4.20, p=0.045) were independently associated with survival outcomes. Addition of SpO_2 recovery improved ODDS model performance (p<0.01) and DO-GAP in the whole ILD cohort.



Abstract S61 Figure 1

Conclusions Prolonged SpO_2 recovery time following 6MWT independently predicts adverse outcomes in ILD and may improve risk stratification beyond established 6MWT derived prognostic parameters.

S62 INTERMITTENT HYPOXAEMIA IS ASSOCIATED WITH THE QUALITY-OF-LIFE OF PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS

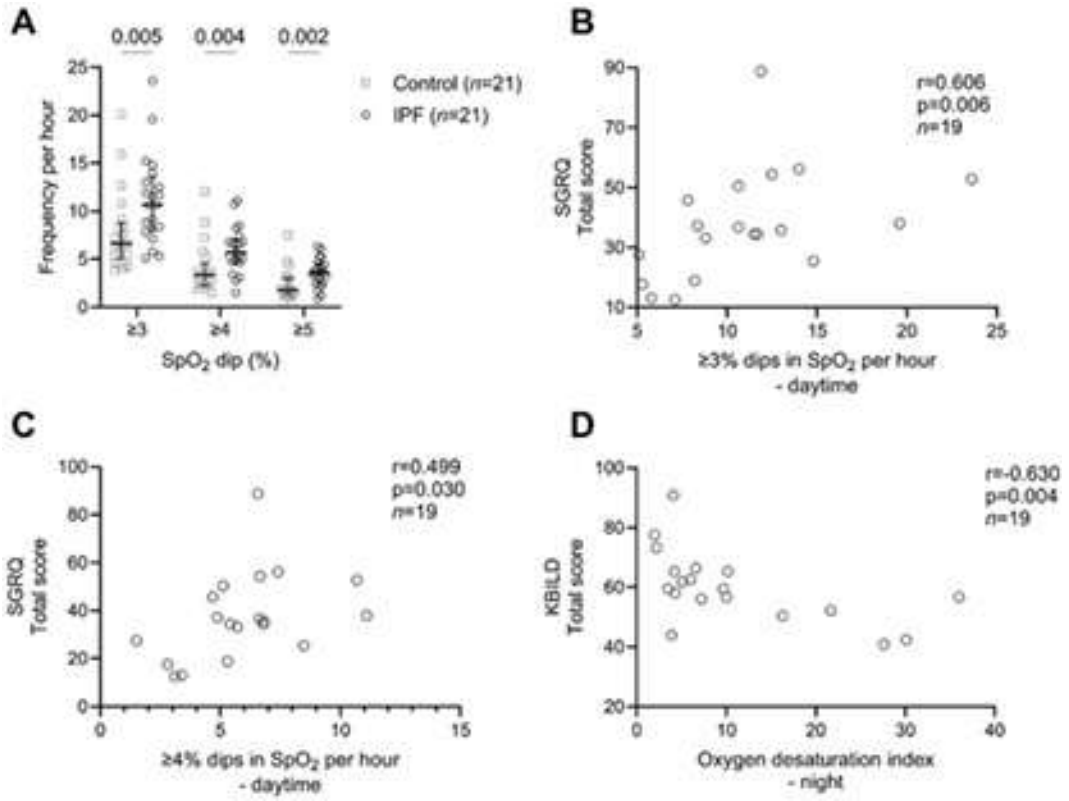
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10.1136/thorax-2026-BTSabstracts.68

Introduction Idiopathic pulmonary fibrosis (IPF) is characterised by progressive lung fibrosis, hypoxaemia and worsening quality-of-life. The impact of intermittent hypoxaemia on quality-of-life is unclear. This study aimed to explore the relationship between intermittent hypoxaemia and patient related outcome measures (PROMs) in patients with IPF.

Methods A prospective observational study (HYBRID, REC:22/WM/0274) was performed at the Queen Elizabeth Hospital Birmingham. Patients with a multidisciplinary team diagnosis of IPF and control participants were recruited. Continuous daytime and overnight oximetry was undertaken on a single day/night using wristwatch oximeters (Checkme O2, Viatom Technology and WatchPAT^{TM}300, Itama Medical Ltd., respectively). IPF participants self-completed the Medical Research Council (MRC) dyspnoea scale, King's Brief Interstitial Lung Disease (KBILD) and St George's Respiratory Questionnaires (SGRQ). Spearman's rank correlations were performed.

Results 23 IPF and 23 age-matched control participants were recruited. IPF patients' mean FVC and TL_{CO} were 78% and 59%, respectively. IPF patients' mean daytime and overnight oxygen saturation (SpO_2) were 94% and 93%, respectively. IPF patients experienced a greater frequency of daytime intermittent hypoxaemia (≥4% dips) than controls (5.7 (4.7–7.1) versus 3.4 (2.3–4.5) p=0.004, figure 1A). Nighttime oxygen desaturation index (ODI) was higher in IPF patients (p=0.003, *n*=19-20). Sub-group analyses revealed that IPF patients with FVC >80% predicted experienced a greater frequency of intermittent hypoxaemia than controls during the daytime (≥5% SpO_2 dips, p=0.038, *n*=10-21) and night (≥4% SpO_2 dips, p=0.034, *n*=9-20). Daytime ≥3% SpO_2 dips was associated with MRC dyspnoea scale (r=0.574, p=0.007), SGRQ (figure 1B) and KBILD breathing and activity score (r=-0.488, p=0.029). Daytime ≥4% SpO_2 dips was associated with SGRQ score (figure 1C). Overnight ODI was associated with total SGRQ (r=0.701, p=0.001) and KBILD scores (figure 1D). Percentage of sleep SpO_2 <90% was associated with SGRQ score (r=0.539, p=0.021).



Abstract S62 Figure 1 (A) - Patients with IPF experience a higher frequency of daytime intermittent hypoxaemia than control participants (p<0.01 by Mann-Whitney tests). (B) - The frequency of ≥3% dips in oxygen saturation (SpO2) per hour during the daytime in patients with IPF was positively associated with total St George's Respiratory Questionnaire (SGRQ) score (r=0.606 (95% CI: 0.195-0.836); p=0.006 by Spearman's rank correlation coefficient, n=19). (C) - The frequency of ≥4% dips in SpO2 per hour during the daytime in patients with IPF was positively associated with total SGRQ score (r=0.499 (95% CI: 0.043, 0.783); p=0.030 by Spearman's rank correlation coefficient, n=19). (D) – Oxygen desaturation index (≥4% dips in SpO2) during the night in patients with IPF was negatively associated with total KBILD score (r =- 0.630 (95% CI: -0.847 - -0.232); p=0.004, n=19)

Conclusions This study identified that intermittent hypoxaemia is common in IPF even those with mild disease. Frequency of episodic hypoxaemia is associated with PROMs. This study highlights the need for further investigation into the relationship between intermittent hypoxaemia and quality-of-life in IPF patients. In particular, curation of clinical trials that determine the benefit and optimal administration of ambulatory/nocturnal oxygen therapy in IPF.

S63 ACUTE RESPIRATORY WORSENING AND SURVIVAL OUTCOMES IN PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS: A POPULATION-BASED COHORT STUDY

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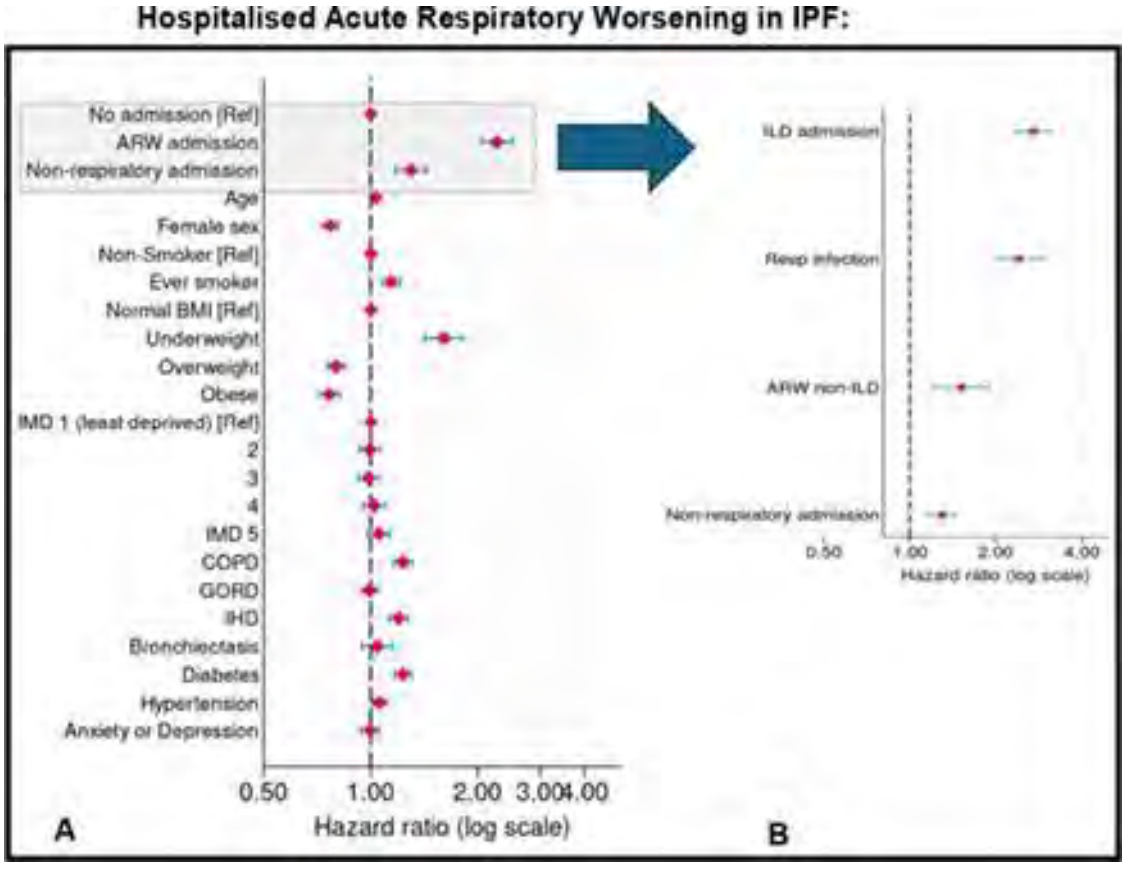
10.1136/thorax-2026-BTSabstracts.69

Introduction Acute exacerbations of idiopathic pulmonary fibrosis (IPF) are associated with abrupt clinical deterioration and increased mortality. Studies have focused on hospitalised exacerbations, with limited understanding of community-managed episodes, particularly those related to respiratory infections.

Using the acute respiratory worsening (ARW) framework, we characterised ARW incidence, assessed association with mortality, identified the highest-risk ARW components, and evaluated mortality risk associated with community-treated infections.

Methods We identified a cohort of adults with IPF using health records from the Clinical Practice Research Datalink linked to Hospital Episode Statistics. The primary exposure was hospitalisation, categorised as ARW or non-respiratory admissions. ARW admissions were further classified into: interstitial lung disease (ILD)-related, respiratory infection, and other respiratory. The outcome was all-cause mortality, obtained from Office for National Statistics death records.

We also assessed community-treated respiratory infections and their relationship with mortality, including episodes preceding hospital admission. Analyses employed a 31-day landmark design with adjusted Cox proportional hazards models controlling for age, sex, socioeconomic status, and comorbidities. Follow-up was censored at subsequent hospital admission.



Abstract S63 Figure 1 Forest plot showing the association between hospital admission and all-cause mortality. (A) displays admission categorised as either acute respiratory worsening (ARW) or non-respiratory admission; (B) is the same model, but the ARW admission reason is more granular: ARW includes ILD admission, respiratory infection or ARW that was non-ILD/infection

Results A total of 13,694 IPF patients were included, with 54,220 hospitalisations in 9,768 patients between 2004 and 2024. Hospitalisation rates were 37.0, 29.1, and 25.8 per 100 patient-years for ILD-related, respiratory infection, and other respiratory admissions respectively.

ARW admission was associated with an approximately three-fold increase in mortality compared with no hospitalisation (HR 2.27, 95% CI 2.05-2.51). By ARW category, ILD-related and respiratory infection admissions showed the strongest associations (HR 2.71, 95% CI 2.35-3.11; HR 2.41, 95% CI 1.99-2.91).

Community-treated respiratory infections were also associated with increased mortality (HR 1.28, 95% CI 1.08-1.52). Among these patients, 888 of 3,535 (25.12%) were hospitalised within 30 days (median 10 days, IQR 4-19).

Mortality rates were highest within 90 days following hospital admission (130.1 per 100 patient-years) and community-treated infections (97.9 per 100 patient-years). Although rates declined by 12 months, they remained elevated compared with non-hospitalised and non-infected patients.

Conclusion ARW episodes in IPF are associated with substantially increased mortality, particularly within 90 days. Respiratory infections confer a mortality risk comparable to ILD-related admissions and warrant close monitoring, even when managed in the community.

S64 EXTERNAL VALIDATION OF MORTALITY PREDICTION MODELS IN IDIOPATHIC PULMONARY FIBROSIS USING REAL-WORLD DATA FROM THE BRITISH THORACIC SOCIETY (BTS) INTERSTITIAL LUNG DISEASE (ILD) REGISTRY

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Introduction and Objectives Idiopathic pulmonary fibrosis (IPF) is the most common progressive interstitial lung disease (ILD) and is associated with substantial morbidity and mortality. Trial-derived prognostic models, including the baseline Gender-Age-Physiology (GAP), longitudinal GAP, and du Bois models, link declining lung function to increased mortality risk but lack external validation in real-world settings. We aimed to validate these models using data from the British Thoracic Society (BTS) ILD Registry.

Methods Patients with IPF were identified from the BTS ILD Registry (February 2013–June 2025). For the baseline GAP model, patients were required to have an observed death or at least 1 follow-up visit. For the longitudinal GAP and du Bois

Abstract S64 Table 1 Baseline characteristics of the analytic cohorts for the 3 mortality prediction models

Characteristics	Baseline GAP model N = 2108	Longitudinal GAP model N = 123	du Bois model N = 131
Age, mean ± SD [median], years	73.8 ± 8.0 [74]	73.5 ± 7.8 [74]	73.5 ± 7.9 [74]
≤60	123 (5.8%)	10 (8.1%)	11 (8.4%)
61–65	177 (8.4%)	12 (9.8%)	13 (9.9%)
66-69	289 (13.7%)	8 (6.5%)	9 (6.9%)
≥70	1519 (72.1%)	93 (75.6%)	98 (74.8%)
Male, n (%)	1743 (82.7%)	108 (87.8%)	114 (87.0%)
ppFVC, %, mean ± SD [median]	81.0 ± 19.7 [79]	86.2 ± 19.1 [83]	85.3 ± 19.3 [82]
≤50	69 (3.3%)	1 (0.8%)	2 (1.5%)
51-65	370 (17.6%)	10 (8.1%)	12 (9.2%)
66-79	693 (32.9%)	43 (35.0%)	46 (35.1%)
≥80	976 (46.3%)	69 (56.1%)	71 (54.2%)
ppDL$_{CO}$, %, mean ± SD [median]	50.7 ± 19.1 [48]	57.5 ± 16.4 [57]	57.5 ± 16.4 [57]
≤25	112 (5.3%)	2 (1.6%)	2 (1.5%)
26-35	335 (15.9%)	11 (8.9%)	11 (8.4%)
36-55	920 (43.6%)	42 (34.1%)	42 (32.1%)
>55	741 (35.2%)	68 (55.3%)	68 (51.9%)
Unknown	0	0	8 (6.1%)
Undergone a 6MWT in past 12 months, n (%)	686 (32.5%)	38 (30.9%)	41 (31.3%)
Distance walked in 6MWT, mean ± SD [median], meters	313.8 ± 144.6 [320]	339.0 ± 100.7 [350]	330.9 ± 115.2 [350]
History of respiratory hospitalizations, n (%)	–	15 (12.2%)	17 (13.0%)
Antifibrotic medications in past 3 months, n (%)	840 (39.8%)	30 (24.4%)	35 (26.7%)
Comorbidities, n (%)			
Hypertension	732 (34.7%)	49 (39.8%)	52 (39.7%)
Ischemic heart disease	458 (21.7%)	27 (22.0%)	27 (20.6%)
Symptoms of gastroesophageal reflux disease	426 (20.2%)	34 (27.6%)	37 (28.2%)
Diabetes	410 (19.4%)	22 (17.9%)	24 (18.3%)
Atrial arrhythmias	88 (4.2%)	10 (8.1%)	10 (7.6%)
BMI categories, n (%)			
Underweight, BMI <18.5	6 (0.3%)	0 (0.0%)	0 (0.0%)
Normal weight, 18.5 ≤ BMI <25	224 (10.6%)	16 (13.0%)	17 (13.0%)
Overweight, 25 ≤ BMI <30	446 (21.2%)	51 (41.5%)	55 (42.0%)
Obesity, ≥30	353 (16.7%)	39 (31.7%)	40 (30.5%)
Missing	1,079 (51.2%)	17 (13.8%)	19 (14.5%)

6MWT, 6-minute walk test; BMI, body mass index, ppDL$_{CO}$, percent of predicted diffusing capacity of the lung for carbon monoxide; SD, standard deviation.

models, patients were required to have a percent predicted forced vital capacity (ppFVC) measurement at 24±6 weeks from the initial visit, along with an observed death or subsequent follow-up. Model performance was evaluated using the integrated calibration index (ICI) and time-dependent area under the curve (AUC). Sensitivity analyses varied the allowable follow-up window for ppFVC assessments.

Results The baseline GAP, longitudinal GAP, and du Bois analyses included 2108, 123, and 131 patients, respectively. Mean age was approximately 74 years, with >80% male (table 1). Mean ppFVC was 81%, 86%, and 85% across the 3 cohorts. The longitudinal GAP model demonstrated the strongest overall performance, with excellent calibration (ICI 0.009 at 1 year and 0.002 at 2 years) and discrimination (AUC 0.84 at 1 year and 0.87 at 2 years). The baseline GAP model also performed well, with good calibration (ICI 0.024 at 1 year and 0.066 at 2 years) and discrimination (AUC 0.73 at both 1 and 2 years). The du Bois model demonstrated good discrimination (AUC 0.79) at 1 year, with calibration indicating modest overestimation (ICI 0.048). Sensitivity analyses demonstrated consistent findings.

Conclusions This study is the first real-world validation of the IPF mortality prediction models. All 3 models demonstrated moderate-to-strong calibration and discrimination using real-world data. The findings support their reliability beyond trial settings and their utility for prognostic assessment in routine practice, helping clinicians identify patients at higher risk and inform risk stratification, monitoring, and care planning.

S65 SOCIAL, GEOGRAPHIC AND ENVIRONMENTAL DETERMINANTS OF MORTALITY IN PULMONARY FIBROSIS: A PROSPECTIVE PROFILE COHORT ANALYSIS

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10.1136/thorax-2026-BTSabstracts.71

Background Social and environmental factors influence outcomes in pulmonary fibrosis (PF), but their interplay is poorly characterised. We investigated social deprivation, access to specialist care $PM_{2.5}$ exposure and their interactions with PF mortality.

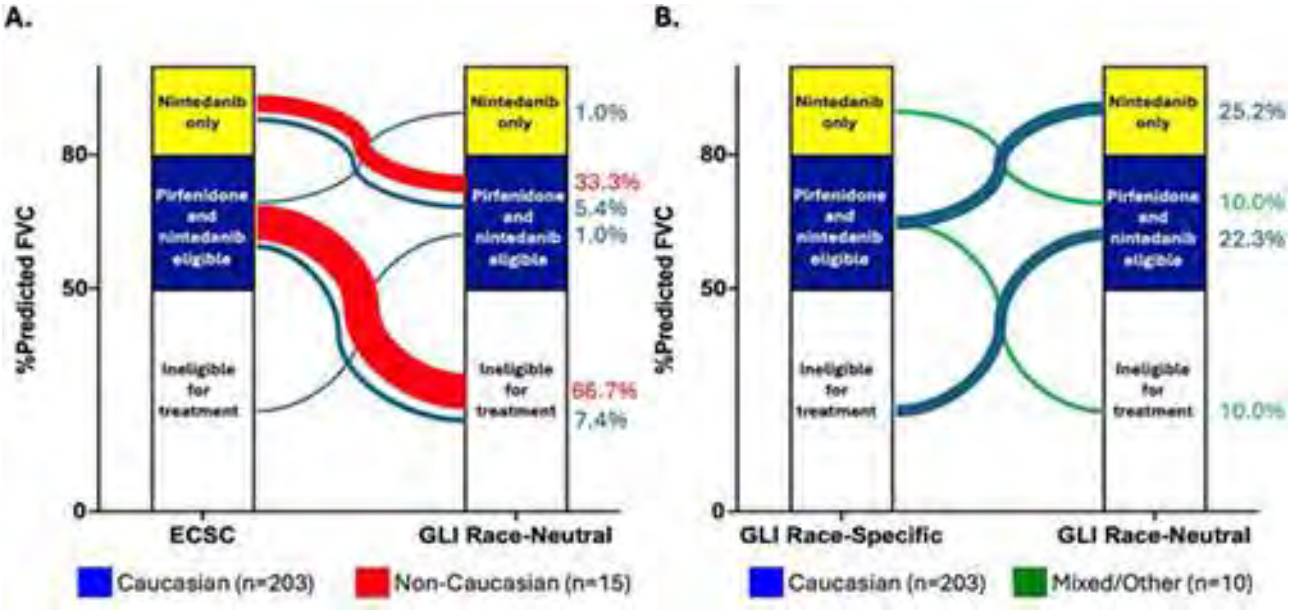
Methods PROFILE is a UK multicentre prospective cohort of incident PF. Deprivation was assessed using Index of Multiple Deprivation quintiles (Q1 most deprived) and distance to the nearest expert ILD centre was calculated using the Haversine formula. $PM_{2.5}$ was estimated from satellite-derived hybrid models with 1 km^2 resolution and modelled as a time-varying five-year sliding mean in Cox proportional hazards models.
Results In 574 participants, greater deprivation was associated with worse survival; Q3–Q5 had lower mortality than Q1(HRs 0.68–0.80, p<0.05). Each 10 km increase in distance from an ILD centre was associated with worse survival (HR 1.06, 95%CI 1.01–1.10, p=0.008); patients beyond 50km had 28% greater risk (HR 1.28, 95%CI 1.01–1.63, p=0.045). Patients with higher $PM_{2.5}$ exposures trended towards worse survival, but significance was not achieved (HR 1.57 per 5 $\mu g/m^3$, 95%CI 0.97–2.53, p=0.065). The $PM_{2.5}$–mortality association was strongest in the most deprived patients and attenuated after Q3 (Q1–Q2: HR 3.30, 95%CI 1.45–7.48, p=0.004; interaction p=0.001) and was amplified beyond 50 km from specialist care (interaction p=0.054).
Conclusions Deprivation and distance from specialist ILD care were independently associated with mortality in PF. $PM_{2.5}$ influences on survival were concentrated in the most deprived, highlighting compounding environmental and social vulnerability and the need for targeted air quality and equitable care policies.

S66 THE IMPACT OF RACE AND GENDER ON DIFFERENT SPIROMETRIC REFERENCE EQUATIONS USED TO ASSESS %-PREDICTED FVC FOR ANTIFIBROTIC ELIGIBILITY IN IDIOPATHIC PULMONARY FIBROSIS

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10.1136/thorax-2026-BTSabstracts.72

Background Eligibility for antifibrotic treatment for IPF, as set out in NICE-UK guidelines, is contingent on meeting treatment thresholds based on %-predicted forced vital capacity (%pred-FVC) most commonly derived using the European Community of Steel and Coal (1993), the Global Lung Initiative Race-Specific (2012) or increasingly, the GLI Race-Neutral (2022) references. Comparison of these equations in a UK IPF cohort stratified by ethnicity and sex has not been undertaken. This study tested the hypothesis that equation-specific %predFVC differences underpin the 'misclassification' that might disproportionately affect non-Caucasian and female patients.
Methods 716 patients were retrospectively identified from an IPF antifibrotic treatment database (2011–2021) at a tertiary IPF referral centre. The closest %predFVC prior to antifibrotic treatment was generated for all three equations. Continuous



Abstract S66 Figure 1 Directional visualisation of reclassification between %predicted FVC treatment thresholds amongst borderline patients when switching reference equations to GLI Race-Neutral. Panel A presents reclassification from ECSC to GLI Race-Neutral and Panel B from GLI Race-Specific to GLI Race-Neutral. Flow lines represent the proportion of borderline patients within each ethnic subgroup moving between treatment eligibility zones, with line thickness proportional to the annotated percentage. Abbreviations: FVC, forced vital capacity; ECSC, European Community of Steel and Coal; GLI, Global Lung Function Initiative

data were compared using the Friedman test (with post-hoc Dunn's or Mann-Whitney U tests) and categorical data using Fisher's exact test ($p<0.05$). Bland-Altman plots were used to detect systematic bias between equations stratified by ethnicity and sex.

Results Amongst White Caucasian (WC) subjects, GLI-Race Neutral (GLI-RN) produced results that were 3-percentage points higher than GLI-Race Specific (GLI-RS) ($p<0.0001$) but were not significantly different to ECSC. In contrast, GLI-RN-predicted FVC results were 7 and 8% lower than GLI-RS ($p=0.0033$) and ECSC ($p=0.0016$) respectively in Black subjects. GLI-RN results in Mixed/Other ethnicity subjects were similarly lower compared to GLI-RS and ECSC (1% and 8% respectively, $p<0.0001$). The median degree of bias in %predFVC from ECSC to GLI-RN ranged from +0.33 (WC males), −8.26 (WC females), −6.45 (non-Caucasian males) to −16.14 (non-Caucasian females). Gender differences in %predFVC rederivation using GLI-RN from ECSC were -8.6% in Caucasians to -9.7% in non-Caucasians ($p<0.0001$). Notably, switching from ECSC to GLI-RN reclassified 100% 'FVC-borderline' (i.e. within 5% of antifibrotic treatment threshold) patients of non-Caucasian ethnicity versus only 1-in-6 Caucasian patients ($p<0.0001$).

Conclusion Adoption of the GLI-RN spirometric reference can impact IPF antifibrotic eligibility due to differential race and gender-related weighting. Patients with ECSC-derived %predFVC near the lower antifibrotic eligibility threshold may be particularly disadvantaged. Effort is required to ensure that reference equations are accurate and representative in the quest for equitable patient care.

Paediatric respiratory medicine: vaccines, asthma and innovation

S67 BLOWING AWAY INEQUALITY: 12-MONTH OUTCOMES FROM A CHILDREN AND YOUNG PEOPLE (CYP) ASTHMA DIAGNOSTIC HUB

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10.1136/thorax-2026-BTSabstracts.73

Introduction The 2024 BTS/NICE/SIGN guidelines recommend objective testing to diagnose asthma in children and young people (CYP) aged over 5 years. NHS Community Diagnostic Centres (CDCs) can deliver such testing in community, consistent with NHS 10 Year Plan. We report 12-month outcomes of CYP referred from primary care to an inner-city CDC for asthma diagnosis, seen within two weeks.

Methods Service evaluation of prospectively collected data on CYP aged 5–16 years referred to a one stop diagnostic clinic (March 2025–April 2026), seen by a paediatric respiratory physiologist and an asthma nurse. The BTS/NICE/SIGN algorithm was followed: fractional exhaled nitric oxide (FeNO) and spirometry were performed in symptomatic children, with a trial of treatment where tests were normal or unobtainable. Demographics, medications, previous attacks, Asthma Control Test (ACT) scores and caregiver feedback were recorded. Descriptive statistics and parametric tests were used for analysis.

Results Two hundred and nineteen CYP were seen within 2 weeks of referral. Median age was 9 years (range 5–16); 58% (126/219) were male, 92% (201/219) from ethnic groups and 95% (208/219) lived in most deprived deciles. ACT was <20 in 65% (138/213). Acceptable quality FeNO was obtained in 65% (144/219), spirometry in 62% (136/219), at least one test in 93%(203/219). FeNO> 35 ppb was in 44% (63/144); spirometry showed airflow obstruction in 11% (15/136). Neither differed by symptom control. 62% (136/219) were prescribed inhaled corticosteroids (ICS) at referral, of whom only 31% (34/90) were adherent; non-adherent children 69% (56/90) had higher FeNO (median 29 vs 10 ppb; $p=0.017$). 81% (177/219) were diagnosed by objective measures or treatment trial, 9% (20/219) had suspected and 9% (20/219) excluded asthma, and only 1 child required secondary care referral. 99% (217/219) of families rated the service excellent.

Conclusions A paediatric nurse and physiologist delivered one stop CDC asthma diagnosis to underserved inner city children, confirming asthma in 81% of cases with a single secondary care referral. FeNO positivity reached 44%, exceeding the 30% reported by Hakizimana et al. (2024). Treatment trials remained essential. Co delivering diagnosis with essential asthma care could narrow inequity and improve outcomes.

S68 LEFT BREATHLESS: GAPS IN BASIC ASTHMA CARE FOR CHILDREN AND YOUNG PEOPLE IN ENGLAND — A RETROSPECTIVE COHORT STUDY

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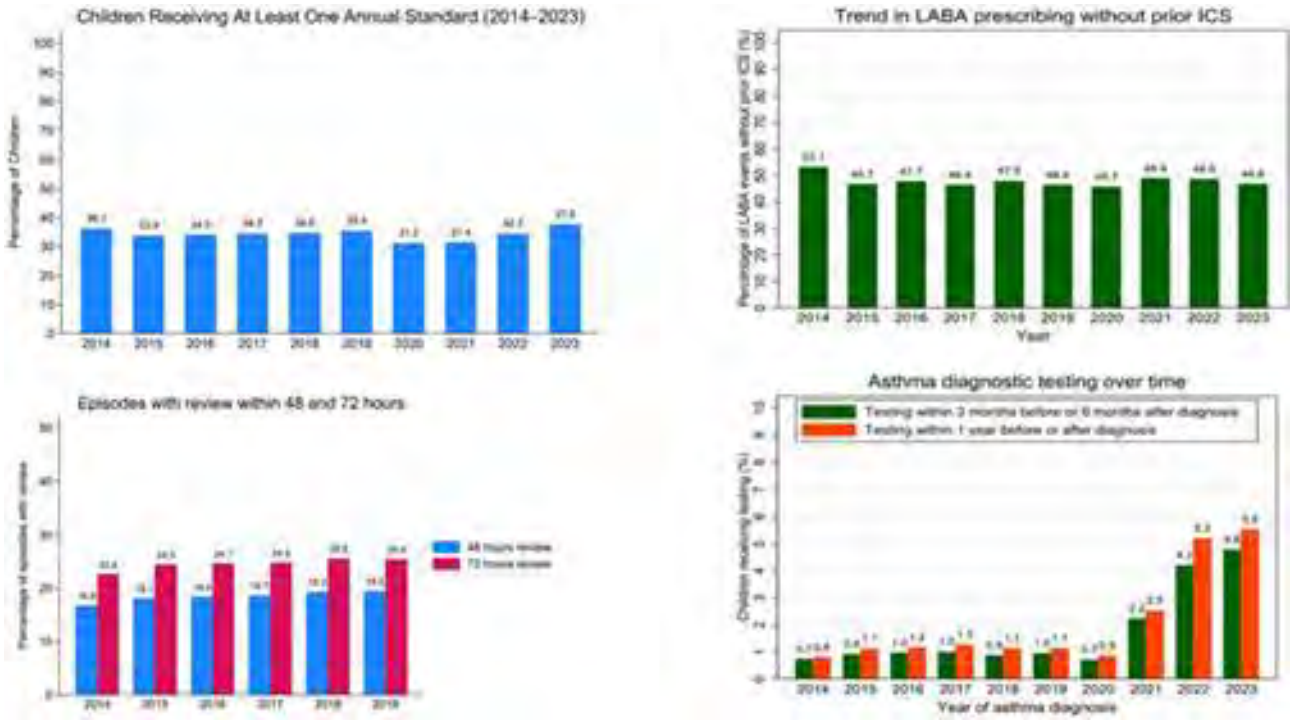
10.1136/thorax-2026-BTSabstracts.74

Background The UK National Review of Asthma Deaths Report in 2024 issued multiple recommendations for asthma standards of care. These informed national guidelines and the quality and outcomes framework used to renumerate UK general practices.

Aim To benchmark asthma care delivered to children and young people (CYP) in England between 2014-2023 against national recommendations.

Methods Retrospective cohort study using data from the Clinical Practice Research Datalink, linked with Hospital Episodes Statistics, Office of National Statistics, and the Index of Multiple Deprivation. We included CYP with asthma, aged 5-21 registered with a GP between 2014-2023. We calculated the percentage of CYP who received the nationally recommended standards of care: basic asthma care (annual review, personalised action plan (PAAP), inhaler technique check, smoking/smoking exposure status, asthma control check), evidence of objective testing 3 months before or 6 months after diagnosis, prescription of inhaled corticosteroid (ICS) if long-acting beta2-agonist (LABA) prescribed, evidence of a review within 2 working days after acute healthcare presentation.

Results There were 601,542 CYP with asthma between 2014 and 2023, 56% male, 65% of white ethnic background, and



Abstract S68 Figure 1 Trends in the delivery of asthma care standards over the years in children and young people

24% from the most deprived quintile. On average per year, 34% of CYP received at least one of the basic asthma care standards with a stable trend over time. In 2023, 23% of CYP had an asthma review, 24% received PAAP, 14% had inhaler technique check, 26% had asthma control check, and 35% had their smoking status recorded.

199,824 CYP had an asthma diagnosis code; only 6,017 (3%) had evidence of diagnostic testing within the recommended timeframe.

54,539 LABA inhaler were prescribed, of which only 48% were issued with an ICS within the preceding 60 days.

Between 2014-2019 (last linkage date) there were 222,076 acute asthma A&E /hospital attendances; only 41,217 (19%) were reviewed within 48 hours of discharge.

Conclusions Despite recommendations, there was no observable improvement in the delivery of basic elements of asthma care to CYP, and only a small increase in diagnostic testing. There was no clear disparity across demographic groups. Further research exploring the barriers to delivery of standards of care is recommended.

S69 CLESROVIMAB SAFETY AND RSV-ASSOCIATED DISEASE INCIDENCE RATES AMONG INFANTS AT INCREASED RISK FOR SEVERE RSV DISEASE BY SUBGROUP: RESULTS FROM THE PHASE 3 SMART TRIAL

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Background SMART (NCT04938830) was a phase 3 trial of clesrovimab, a long-acting RSV monoclonal antibody, in infants at increased risk of severe RSV disease due to prematurity, chronic lung disease (CLD) of prematurity, or hemodynamically significant congenital heart disease (CHD). We evaluated safety and RSV-associated disease incidence rates across infant subgroups in season 1 of SMART.

Methods Infants were randomized 1:1 to receive a single 105mg dose of clesrovimab or monthly palivizumab. Safety was evaluated as proportions of participants experiencing solicited adverse events (AEs) days 1-5 after any dose, non-serious AEs and adverse events of special interest (AESI) days 1-42 post-dose 1, and serious AEs (SAEs) throughout season 1. Incidence rates of RSV-associated medically attended LRI (MALRI) requiring >1 indicator of LRI or severity and of RSV-associated hospitalization were assessed through day 150. Safety and RSV-associated disease incidence rates were assessed by predefined subgroups: participant condition, age, and weight at randomization.

Results In total, 997 participants were randomized and received clesrovimab (n=498) or palivizumab (n=499); 28.1% CLD, 10.6% CHD, 7.9% non-CLD/CHD <29 weeks gestational age (GA), and 53.4% non-CLD/CHD >29-35 weeks GA. The proportions of participants experiencing AEs were generally comparable between treatment groups and across subgroups. The incidence of RSV-associated disease was generally comparable between treatment groups within each subgroup; the highest rates were observed among infants with CHD (table 1).

Conclusions Clesrovimab was well tolerated in infants at increased risk for severe RSV disease across subgroups based on participant condition, age, or weight. The safety profile across subgroups was consistent with the overall population. Differ-

Abstract S69 Table 1 RSV-associated disease incidence by predefined infant subgroup through Day 150

	Clesrovimab (N=498)				Palivizumab (N=499)			
Predefined subgroup	**n**	**No. of cases**	**Total follow-up time in months**	**Incidence rate, % (95% CI)**	**n**	**No. of cases**	**Total follow-up time in months**	**Incidence rate, % (95% CI)**
RSV-Associated Outpatient and Inpatient MALRI								
Participant Condition								
CLD	137	4	663.3	3.0% (0.8, 7.7)	138	5	661.2	3.8% (1.2, 8.8)
CHD	52	4	231.7	8.6% (2.4, 22.1)	51	4	239.2	8.4% (2.3, 21.4)
Non-CLD/CHD <29 weeks GA	41	0	197.6	0% (0.0, 0.9)	38	2	184.5	5.4% (0.7, 19.6)
Non-CLD/CHD ≥29 weeks GA	265	7	1282.3	2.7% (1.1, 5.6)	258	5	1251.3	2.0% (0.6, 4.7)
Age at randomization								
<6 months	452	13	2182.2	3.0% (1.6, 5.1)	416	11	2017.5	2.7% (1.4, 4.9)
≥6 months	43	2	192.8	5.2% (0.6, 18.7)	69	5	318.8	7.8% (2.5, 18.3)
Weight at randomization								
<5 kg	391	10	1893.2	2.6% (1.3, 4.9)	390	11	1890.4	2.9% (1.5, 5.2)
≥5 kg	104	5	481.8	5.2% (1.7, 12.1)	95	5	445.9	5.6% (1.8, 13.1)
RSV-Associated Hospitalization								
Participant Condition								
CLD	137	2	667.9	1.5% (0.2, 5.4)	138	3	669.2	2.2% (0.5, 6.6)
CHD	52	2	236.8	4.2% (0.5, 15.3)	51	3	241.9	6.2% (1.3, 18.1)
Non-CLD/CHD <29 weeks GA	41	0	197.6	0% (0.0, 9.3)	38	1	188.1	2.7% (0.1, 14.8)
Non-CLD/CHD ≥29 weeks GA	265	1	1296.2	0.4% (0.0, 2.1)	258	1	1265.4	0.4% (0.0, 2.2)
Age at randomization								
<6 months	452	4	2202.7	0.9% (0.2, 2.3)	416	6	2032.4	1.5% (0.5, 3.2)
≥6 months	43	1	195.8	2.6% (0.1, 14.2)	69	2	332.1	3.0% (0.4, 10.9)
Weight at randomization								
<5 kg	391	3	1908.6	0.8% (0.2, 2.3)	390	7	1901.8	1.8% (0.7, 3.8)
≥5 kg	104	2	489.9	2.0% (0.2, 7.4)	95	1	462.8	1.1% (0.0, 6.0)

CHD=hemodynamically significant congenital heart disease; CLD=chronic lung disease of prematurity; GA=gestational age; MALRI=Medically attended lower respiratory infection; RSV=Respiratory syncytial virus.

ences in RSV-associated disease incidence rates across participant conditions support efforts to strengthen RSV prevention for all infants in their first year of life.

REFERENCE

1. SMART trial registration: NCT04938830.

S70 A SECONDARY ANALYSIS OF A PHASE 3, RANDOMISED, DOUBLE-BLIND STUDY OF V116, A PNEUMOCOCCAL CONJUGATE VACCINE, IN CHILDREN AND ADOLESCENTS WITH INCREASED RISK OF PNEUMOCOCCAL DISEASE (STRIDE-013)

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Background V116 is a pneumococcal conjugate vaccine designed to broaden protection against invasive pneumococcal disease (IPD) and pneumonia in adults. Children and adolescents aged 2 to <18 years with chronic medical conditions are also at increased risk of IPD and could potentially benefit from the broader disease coverage provided by V116. These data from the V116-013 study evaluated immunogenicity of V116 by subgroup in this at-risk population.
Methods The study enrolled 882 participants aged 2 to <18 years who were randomised 3:2 to receive a single dose of V116 or 23-valent pneumococcal polysaccharide vaccine (PPSV23), respectively. Immunogenicity was assessed 30 days post-vaccination by measuring serotype-specific opsonophagocytic activity (OPA) geometric mean titers (GMTs). OPA GMTs were evaluated within the following subgroups: age (2 to <6, 6 to <12, or 12 to <18), chronic medical condition (diabetes mellitus or chronic heart, kidney, liver, or lung disease), prior pneumococcal vaccine (PCV7, PCV10, PCV13), and prior pneumococcal vaccination regimen (2+1 or 3+1). Safety data has been previously published.
Results V116 induced responses to all 21 vaccine serotypes as assessed by OPA GMTs at 30 days post-vaccination. V116 was comparable to PPSV23 for the 12 common serotypes, and higher for the 9 unique serotypes. These findings were consistent across subgroups.
Conclusions V116 is immunogenic in children and adolescents aged 2 to <18 years with chronic medical conditions that increase risk of IPD, irrespective of age, prior pneumococcal vaccination regimen, or at-risk condition. V116 could address an unmet need in prevention of IPD in this population.

S71 A DOUBLE-BLIND, MULTI-SITE, RANDOMIZED STUDY EVALUATING THE EFFICACY AND SAFETY OF PCV15 IN THE PREVENTION OF ACUTE OTITIS MEDIA (AOM) IN INFANTS AND TODDLERS (PNEU-ERA)

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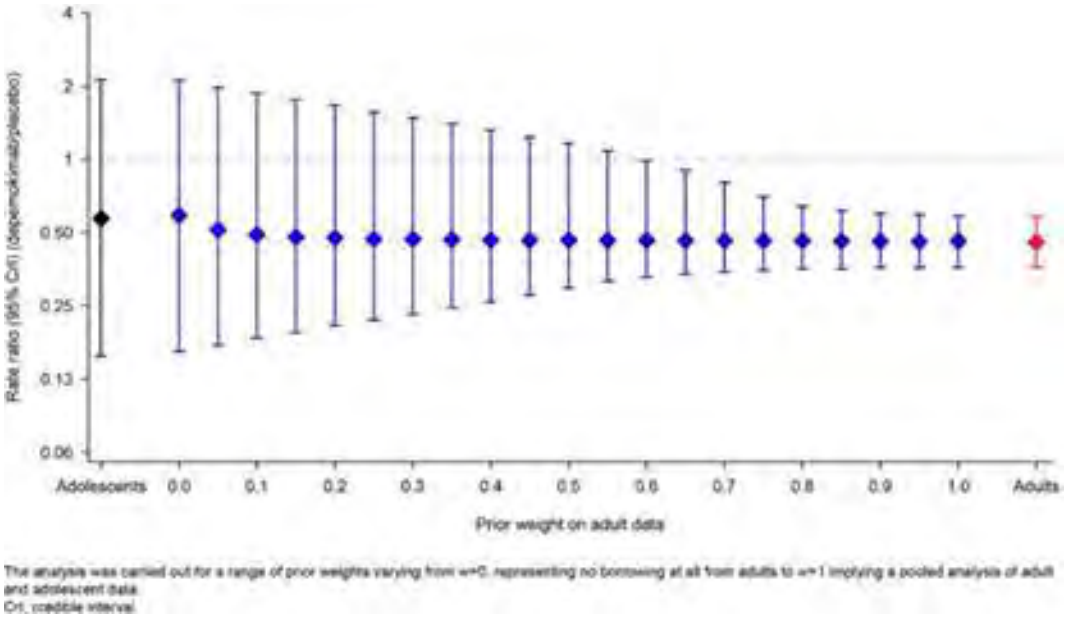
Background Streptococcus pneumoniae remains a leading and often more severe cause of acute otitis media (AOM) in early childhood. Lower-valency pneumococcal conjugate vaccines (PCVs) have demonstrated efficacy and effectiveness against AOM, but evidence for newer, higher-valency PCVs is limited. This study evaluated the efficacy of the 15-valent PCV (PCV15, VAXNEUVANCE(TM)) for preventing AOM when administered routinely to healthy infants and toddlers.
Methods Healthy infants (n=7,119) approximately 2 months of age were randomized 1:1 to receive PCV15 (3+1 schedule) or no PCV15. Recommended childhood vaccines were administered concomitantly with both groups receiving the same number of injections (single and multivalent vaccines) per visit to preserve blinding. Primary endpoints were 1) efficacy of PCV15 in preventing the first episode of vaccine-type pneumococcal AOM (VT-AOM) and 2) safety, assessed by the proportion of participants with serious adverse events (SAEs). An AOM episode was defined as pneumococcal AOM detected by PCR, diagnosed >=14 days after the third study vaccination (approximately 6 months of age).
Results PCV15 reduced the incidence of the first episode of VT-AOM versus control, with vaccine efficacy of 43.71%. Accounting for subsequent episodes of AOM, PCV15 reduced VT-AOM incidence with vaccine efficacy of 45.12%. Efficacy of PCV15 against AOM from all pneumococcal serotypes (vaccine and non-vaccine serotypes) was 34.90%. The proportions of participants with SAEs were comparable between groups.
Conclusions PCV15 is efficacious in preventing AOM in infants and toddlers. Results align with precedent from lower-valency PCVs and support prevention of mucosal pneumococcal disease by PCV15.

S72 EFFICACY AND SAFETY OF TWICE-YEARLY DEPEMOKIMAB IN ADOLESCENT PATIENTS WITH ASTHMA: SUBGROUP ANALYSIS OF THE SWIFT-1/-2 STUDIES

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Rationale Depemokimab is the first ultra-long-acting biologic with enhanced interleukin-5 binding affinity, high potency, and an extended half-life, enabling twice-yearly dosing in patients with asthma. In the Phase III SWIFT-1/-2 studies, depemokimab significantly reduced exacerbations by 54% with sustained suppression of inflammation assessed by blood eosinophil count (BEC) in patients with type 2 asthma. However, the efficacy and safety of depemokimab in adolescents with asthma have not yet been specifically characterised.
Methods Patients with type 2 asthma characterised by BEC and ≥2 exacerbations (past year) were randomised 2:1 to receive depemokimab 100 mg or placebo subcutaneously every 26 weeks, plus standard care. Here, we report prespecified analysis of the primary endpoint, annualised rate of clinically significant exacerbations (CSEs) over 52 weeks and safety data among adolescent patients (aged 12–17 years) in pooled SWIFT-1/-2 pop-



Abstract S72 Figure 1 The Bayesian dynamic borrowing approach identified a minimum prior weight of 0.6 leading to the equivalent of a statistically significant p-value

ulations. A prespecified Bayesian dynamic borrowing approach supported extrapolation of the primary endpoint from adults to adolescents.

Results The pooled SWIFT-1/-2 population comprised 30 adolescents (15 treated with depemokimab)and 732 adults. Annualised rates of CSEs were consistent across adolescents and adults, with 43% and 54% rate reductions (RR [95% CI]: 0.57 [0.15,2.13] and 0.46 [0.36,0.58]), respectively, for depemokimab versus placebo. The Bayesian dynamic borrowing approach indicatedthe minimum historical data borrworing required for statistically significant p-value was 0.6 and was consistent across borrowing scenarios (figure 1). A similar proportion of participants with any adverse event (AE) was observed in the adolescent versus adult treatment groups (73% vs 72%). Among adolescent participants, 2 AEs were reported as treatment-related and 1 serious AE (abdominal pain, not considered treatment-related by the investigator). No deaths or AEs leading to study withdrawal were reported among adolescents.

Conclusions Twice-yearly depemokimab reduced exacerbations and was well tolerated in adolescents in the SWIFT-1/-2 trials, though patient numbers were low. Given prior clinical knowledge of similarities between adult and adolescent patients with asthma, borrowing 60% of adult data is assumed to be reasonable to support extrapolating the impact of depemokimab on CSEs from adults to adolescents. Findings from this analysis support a favorable benefit–risk profile of depemokimab for adolescents.

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Sleepy futures: from hospital to home

S73 INFORMATION FLOW FROM OXYGEN SATURATION TO HEART RATE PREDICTS CARDIOVASCULAR MORTALITY AND MORBIDITY IN OBSTRUCTIVE SLEEP APNOEA

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Introduction Area-based hypoxic burden metrics are associated with increased cardiovascular burden in patients with obstructive sleep apnoea. Limitations include lack of standardisation, linear analysis methodology and the need for multi-channel sleep studies. Non-linear analysis methods could provide a simple standardised measure of cardiovascular burden in this population. Therefore, this study sought to calculate the Transfer Entropy of oxygen saturation (SpO2) and heart rate (HR) and assess its association with cardiovascular disease related mortality and morbidity in patients with obstructive sleep apnoea.

Methods The study used data from the previously well described Sleep Heart Health Study (1) and included participants with a diagnosis of sleep apnoea (AHI ≥ 5events/hr). Two main outcome measures were assessed: cardiovascular mortality and time-to-first MACE defined as composite outcome of myocardial infarction, congestive heart failure, stroke or cardiovascular death. SpO2 and HR signals were extracted and analysed using linear and non-linear analysis methodology. Cox proportional hazard ratios were used to evaluate associations between these measures, cardiovascular death / MACE.

Abstract S73 Table 1 Unadjusted and adjusted hazard ratios of CVD mortality

	Unadjusted hazard ratio (95% CI)	P value	Adjusted hazard ratio* (95% CI)	P value
Traditional metrics				
Apnoea-hypopnoea index (events/hr)	1.00 (1.00 – 1.01)	0.27	1.00 (0.99 – 1.00)	0.57
Total number of oxygen saturation with desaturations >= 3%	1.00 (1.00 – 1.00)	0.28	1.00 (1.00 – 1.00)	0.44
Total number of oxygen saturation with desaturations >= 4%	1.00 (1.00 – 1.00)	0.47	1.00 (1.00 – 1.00)	0.30
Mean SpO2 (%)	0.92 (0.87 – 0.98)	**0.006**	0.96 (0.90 – 1.02)	0.21
Time spent with saturations below 90% (%)	1.02 (1.01 – 1.02)	**<0.001**	1.01 (1.00 – 1.02)	**0.028**
Network Physiology mapping metrics †				
SEHR	1.03 (0.95 – 1.11)	0.48	1.17 (0.60 – 2.30)	0.65
SESats	1.00 (0.86 – 1.15)	0.96	0.94 (0.21 – 4.30)	0.94
TE SpO2→HR*	0.70 (0.63 – 0.77)	**<0.001**	0.85 (0.77 – 0.94	**0.002**
TE HR→SpO2*	1.10 (0.95 – 1.28)	0.22	0.99 (0.85 – 1.14)	0.88

†To improve the interpretability of the effect estimate, TE SpO2→HR and TE HR →SpO2 were rescaled so that hazard ratios were expressed per 0.1-unit increase.
*adjusted for age, gender, BMI, race, smoking history, hypertension, diabetes, myocardial infarction, stroke, heart failure.

Results A total of 3310 participants were analysed (mean(SD) age 64(11) years), 252 died from cardiovascular disease and 578 had MACE. In multivariable Cox proportional hazard models, each 0.1-unit increase in TESpO2toHR was associated with a 15% lower risk of cardiovascular death (aHR 0.85, 95%CI 0.77–0.94;p=0.002). This is shown in table 1. The time-dependent ROC curve for the Cox model in predicting CVD mortality at 5 years had an AUC of 0.88 (95%CI 0.84 - 0.91). Furthermore, participants with a TESpO2toHR below the cohort median had a higher probability of cardiovascular death (log-rank p = 0.0027). TESpO2toHR was also associated with a lower hazard of first MACE at baseline (aHR 0.44, 95%CI 0.24–0.80;p = 0.008), with the effect attenuating over time.

Conclusion TESpO2toHR is independently associated after adjustment with cardiovascular mortality and morbidity and can be calculated from a single-channel oximeter, is standardised and not sensitive to event annotations making it easy to apply in clinical practice. Reduced values illustrate reduced information exchange between SpO2 and HR, meaning reduced system engagement and increased overall cardiovascular risk. Further studies are necessary to validate this association prospectively.

S74 TRANSFORMING SLEEP DIAGNOSTIC PATHWAYS: IMPLEMENTING A CLINICAL DECISION SUPPORT SYSTEM (CDSS) ACROSS A DISTRICT GENERAL HOSPITAL AND COMMUNITY DIAGNOSTIC CENTRE (CDC) SETTING

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Introduction and Objectives The Optimal Sleep Pathway (2025) provides a national framework for delivering scalable, efficient sleep services within NHS trusts, responding to rising demand, and the need for timely assessment. It emphasises adoption of digital diagnostics, expanded roles for allied health professionals, and delivery of care closer to home.

In 2023, sleep studies at our DGH were exclusively consultantreported. Patients meeting criteria set by an external device provider were referred for CPAP following a brief physiologist consultation outlining study results and treatment rationale, while others were discharged or reviewed in facetoface consultant clinics. Escalating referrals created unsustainable service pressure, contributing to delays and potential patient safety risks. Following an NHSE Physiological Science review, a service transformation programme was implemented in January 2025 through our local CDC, incorporating a clinical decision support system, multichannel diagnostics, sleeprelated blood profiling, and enhanced physiologistled care.

Methods In January 2025, a hybrid model was adopted, maintaining the traditional consultantled pathway alongside a digitally enabled CDSS pathway. Within the CDSS model, patients underwent virtual assessment and multichannel sleep studies, with care coordinated through a digital platform. Patients meeting CPAP thresholds were referred directly for treatment, while others were triaged to virtual consultant review (VR). Outcomes were compared between pathways and against 2023 baseline data, focusing on pathway efficiency, clinical outcomes, and service utilisation.

Results Comparison of 2023 and 2025 consultantled pathways demonstrated stable clinical outcomes, with time from study result to clinic review improving from 62 to 34.6 days, reflecting increased physiologist involvement. However, the CDSS pathway delivered greater efficiencies: 49% of patients referred directly for CPAP vs. 0% in earlier models; consultant case review reduced, 100% to 51%; discharge rates following VR increased 23% to 64%; and onward clinic requirements fell, 72% to 35%. Overall clinic conversion to CPAP was lower (42% vs 74%), indicating improved pretest stratification and earlier, more targeted referral of complex cases.

Abstract S74 Table 1 Service outcomes compared between traditional and digitally enhanced models

	Consultant led Sleep Service 2023	Consultant Led Sleep Service 2025	CDSS – Digital enhanced system 2025
No. of Total Sleep Studies Performed	1037	715	698
Study result to clinic appointment	62 days	34.6 days	20.4 days
% Straight to CPAP	0%	0%	49% (341)
% consultant virtual r/v	100%	100%	51% (357)
% CPAP from virtual r/v	0.4% (4)	1% (7)	4.8% (17)
% discharged from virtual r/v	22% (226)	24.8% (176/708)	64.7% (231/357)
% Physiology/consultant clinic	71.5% (739) (All Consultant*)	72.2% (511)	35.2% (126) *(Only 4% specified Cons review)*
% Clinic conversion to CPAP	74% (544/739)	74% (378/511)	42% (53/126)
% Clinic to discharge/PIFU** (**2025)	25.7% (190/739)	18% (92)	57.9% (73)

Conclusion Implementation of a CDSS-enabled service improved pathway efficiency, reduced time to diagnosis, and optimised workforce utilisation through expanded physiologis-tled care. Enhanced pretest probability enabled consultants to focus on higher-risk patients.

S75 MULTI-NIGHT HOME SLEEP APNEA TESTING IMPROVES DIAGNOSTIC ACCURACY COMPARED WITH SINGLE-NIGHT ASSESSMENT

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Introduction and Objectives Obstructive sleep apnea (OSA) is conventionally classified from a single-night recording, yet apnea-hypopnea index (AHI) varies substantially night-to-night. Single-night home sleep apnea testing (HSAT) produces meaningful misclassification, but characterizing the underlying phenotypic structure remains limited at scale. We aimed to quantify AHI variability, single-night misclassification, incremental detection yield with additional nights, and OSA burden as a novel phenotypic dimension.

Methods Retrospective analysis of 57,149 patients referred for diagnostic sleep evaluation in the US, completing 3–7 analyzable consecutive nights (total sleep time ≥2h, within a 10-day window; 8,399 excluded) of photoplethysmography (PPG)-based HSAT. Cumulative AHI (total respiratory events across all nights divided by aggregate total sleep time) served as the reference. Night-to-night variability was quantified by the root



Abstract S75 Figure 1

mean square of successive differences (RMSSD). OSA burden was defined as the proportion of recorded nights exceeding AHI thresholds of 5, 15, and 30 events/hr.

Results Mean age 49.5 ± 14.9 years, 60% male, BMI 32.0 ± 7.1 kg/m^2 (10.7% normal, 26.0% overweight, 45.3% obese), total recording time 7.20 ± 1.48 hrs, total sleep time 5.96 ± 1.28 hrs, and cumulative AHI 14.8 ± 15.9 events/hr. Most patients contributed 3 nights (80.7%) and the remainder had 4–7 nights. RMSSD increased with disease severity (median 1.4–8.5 events/hr) but did not vary across number of recorded nights. One in five patients were reclassified by cumulative AHI relative to Night 1, and one in four had at least one night exceeding their Night 1 classification. Single-night testing missed 11.4% of detectable moderate-to-severe disease, and three nights reduced this to 0.8%. OSA burden was sharply bimodal at every threshold, with 10–25% of patients in an intermittent zone concentrated in the mild and moderate strata, defining a distinct phenotypic group.

Conclusions Multi-night PPG-based HSAT reveals an intermittent OSA phenotype invisible to single-night testing, concentrated where clinical decisions are most consequential. Three nights reduce missed moderate-to-severe disease 14-fold. These findings support a shift from single-night diagnosis to multi-night phenotyping as the foundation for OSA care. Additional validation in NHS diagnostic pathways is warranted to evaluate the generalizability to a UK-based population

S76 DO SCORING METHODS MATTER? THE EFFECT OF WATCHPAT AUTOSCORING ON TREATMENT DECISION-MAKING

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Introduction Obstructive sleep apnoea (OSA) is on the rise and sleep services are struggling to test patients within 6 weeks, with national data suggesting 30% are waiting longer than this. Home-testing devices have been proposed as an alternative to polygraphy and oximetry for OSA screening to increase capacity and support the reduction in waiting times[1,2]. Historically, sleep study results have been manually checked by healthcare scientists, but most devices now offer autoscoring and using this will help to reduce results to test (RTT) times. We have assessed one such device, the WatchPAT 300, to determine if there are different outcomes for patients between autoscore and manual checking.

Methods Consecutive patients referred for OSA screening who returned a WatchPAT device with at least 4 hours of data between December 2025 until May 2026 were included. Studies were autoscored via the zzzPAT platform and data for apnoea-hypopnea index (AHI) using 3% desaturation, time under 90% oxygen saturation (T90), supine AHI and Epworth Sleepiness Scale (ESS) score were collected. Data was compared using autoscore versus manual checking (by a Healthcare Scientist with > 10 years experience) and any changes in severity (categorised as mild = AHI of 5-14.9, moderate=15-29.9 and severe= ≥30) or treatment eligibility.

Results 70 patients had both autoanalysis and manual checking data available. 46% had an ESS >10. 11/70 (16%) had a T90 ≥10%. Median AHI by autoscoring was 14.25 (IQR 29.03) and by manual checking was 14.60 (IQR 28.93). Table 1 shows the agreement. Only 3% differed in severity for AHI between the 2 methods (no OSA to mild OSA and vice versa), but neither changing treatment eligibility. For Supine AHI, 7.5% scored differently, with 3 increasing in severity when manually scored (no OSA to mild, moderate OSA to severe and mild OSA to moderate), the latter being eligible for treatment, and 2 patients decreasing severity category (mild to no OSA and moderate to mild OSA) with no treatment eligibility change.

Conclusion Autoscoring of WatchPAT 300 devices shows excellent agreement with manual checking, freeing up staff time and potentially reducing RTT times.

S77 CARDIOVASCULAR OUTCOMES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE, OBSTRUCTIVE SLEEP APNOEA AND THE OVERLAP SYNDROME

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Introduction Chronic obstructive pulmonary disease (COPD) and obstructive sleep apnoea (OSA) are both independently associated with an increased risk of cardiovascular disease. It is not clear whether this risk is further increased in COPD-OSA overlap. We aimed to determine whether COPD-OSA overlap has a higher cardiovascular burden compared to COPD or OSA alone.

Methods This was a matched cohort study using electronic health records from the Clinical Practice Research Datalink Aurum database (England). Exposures of interest were a diagnosis of COPD, OSA or both based on validated code-lists. Our primary outcome of interest was cardiovascular mortality defined using ICD-10 codes. Our secondary outcomes of interest included: all-cause mortality, incident acute coronary syndrome (ACS), incident stroke and incident heart failure. Multivariable Cox proportional hazard models were used to estimate hazard ratios.

Abstract S76 Table 1 OSA agreement between severity categories for both auto and manual scoring

	Both Mild	Both Moderate	Both Severe	No OSA	Scored differently	Cohen's Kappa
AHI	30%	17.1%	31.4%	18.6%	2.9%	0.961
Supine AHI	22.4%	19.4%	35.8%	14.9%	7.5%	0.898

Abstract S77 Table 1 Hazard ratios for Cardiovascular mortality, all-cause mortality, incident acute coronary syndrome, heart failure and stroke

Group	Reference	Unadjusted Hazard ratio (95% CI)	P value	Adjusted Hazard ratio* (95% CI)	P value
Cardiovascular Mortality*					
COPD-OSA	COPD	0.97 (0.90 – 1.04)	0.43	0.99 (0.92 – 1.06)	0.70
COPD-OSA	OSA	3.07 (2.82 – 3.35)	<0.001	1.72 (1.58 – 1.88)	**<0.001**
OSA	COPD	0.32 (0.30 – 0.33)	<0.001	0.57 (0.54 – 0.60)	**<0.001**
All-cause Mortality*					
COPD-OSA	COPD	0.83 (0.80 – 0.85)	<0.001	1.03 (1.00 – 1.07)	0.06
COPD-OSA	OSA	3.55 (3.41 – 3.69)	<0.001	2.29 (2.20 – 2.38)	**<0.001**
OSA	COPD	0.23 (0.23 – 0.24)	<0.001	0.45 (0.44 – 0.46)	**<0.001**
Acute Coronary Syndrome**					
COPD-OSA	COPD	1.55 (1.41 – 1.70)	**<0.001**	1.27 (1.16 – 1.40)	**<0.001**
COPD-OSA	OSA	2.57 (2.31 – 2.86)	**<0.001**	1.58 (1.42 – 1.76)	**<0.001**
OSA	COPD	0.60 (0.57 – 0.64)	**<0.001**	0.81 (0.76 – 0.86)	**<0.001**
Heart Failure***					
COPD-OSA	COPD	1.80 (1.70 – 1.90)	**<0.001**	1.69 (1.60 – 1.79)	**<0.001**
COPD-OSA	OSA	3.47 (3.26 – 3.71)	**<0.001**	2.01 (1.89 – 2.15)	**<0.001**
OSA	COPD	0.52 (0.50 – 0.54)	**<0.001**	0.84 (0.81 – 0.88)	**<0.001**
Stroke****					
COPD-OSA	COPD	1.00 (0.92 – 1.10)	0.93	1.05 (0.96 – 1.15)	0.27
COPD-OSA	OSA	1.83 (1.66 – 2.02)	**<0.001**	1.17 (1.07 – 1.30)	**0.001**
OSA	COPD	0.55 (0.52 – 0.57)	**<0.001**	0.89 (0.85 – 0.94)	**<0.001**

*adjusted for age, gender, smoking history, heart failure, myocardial infarction, unstable angina, stroke, diabetes, hypertension, obesity, hypercholesterolaemia
**adjusted for age, gender, smoking history, heart failure, stroke, diabetes, hypertension, obesity, hypercholesterolaemia
***adjusted for age, gender, smoking history, unstable angina, stroke, diabetes, hypertension, obesity, hypercholesterolaemia (myocardial infarction was omitted from the model due to collinearity.
****adjusted for age, gender, smoking history, heart failure, myocardial infarction, unstable angina, stroke, diabetes, hypertension, obesity, hypercholesterolaemia

Results The initial study cohort comprised of 365,667 people with COPD (mean (SD)) age 68 (11) years; 59,526 with OSA age 57 (11) years and 11,040 with COPD-OSA age 65(10) years. Cardiovascular mortality was significantly higher in patients with COPD-OSA overlap compared to OSA alone (adjusted HR 1.72 (1.58 – 1.88), p<0.001) but not compared to COPD alone. People with COPD-OSA overlap had a significantly increased all-cause mortality compared to OSA alone (aHRE 2.29 (2.20-2.38), p<0.001) but not compared to COPD alone. People with COPD-OSA overlap had 27% and 58% increased risk of developing ACS compared to COPD and OSA alone respectively, and 69% and 101% increased risk of developing heart failure compared to COPD and OSA respectively. These results are illustrated in table 1.

Conclusions We have demonstrated that people with COPD-OSA overlap have a significantly higher cardiovascular and all-cause mortality compared to OSA alone. This increased mortality was not seen when comapred to people with COPD alone. We have also demonstrated that people with COPD-OSA overlap have a significantly higher risk of developing ACS and heart failure compared to people with COPD or OSA alone suggesting an increased cardiovascular burden in the overlap population. Earlier and targeted treatment with cardiovascular optimisation can potentially reduce downstream sequelae in people with COPD-OSA overlap.

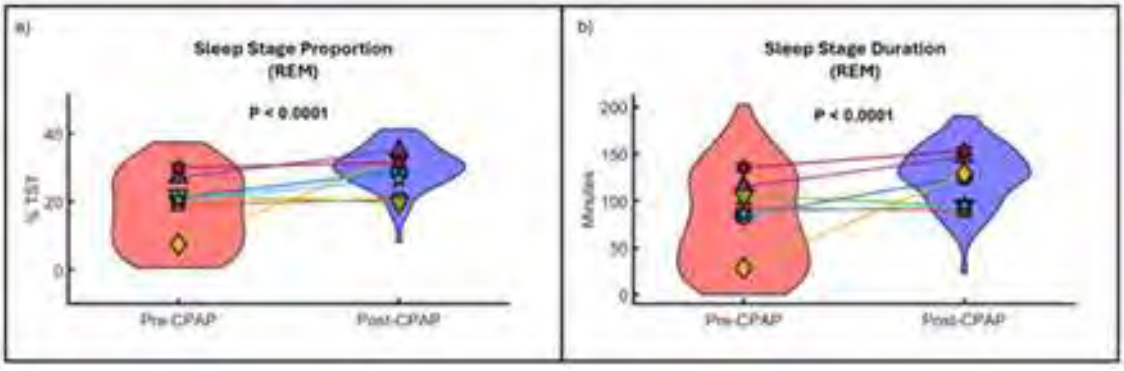
S78 CPAP IS ASSOCIATED WITH IMPROVEMENTS IN REAL-WORLD DRIVING PERFORMANCE AND SLEEP QUALITY IN OBSTRUCTIVE SLEEP APNOEA

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10.1136/thorax-2026-BTSabstracts.84

Background Patients with untreated Obstructive Sleep Apnoea (OSA) experience disrupted sleep and impaired driving performance, both of which may improve following continuous positive airway pressure (CPAP) initiation. However, standard assessment methods have limitations.[1] Sleep quality is typically assessed over a single-night, often in an unfamiliar clinical environment, while driving risk assessment remains reliant on subjective reporting and driving simulators have limited ecological validity.[2] Here, we assessed whether passive smartphone telematics could detect changes in real-world driving performance and, in a subset of participants, whether portable EEG could detect sleep quality changes Pre/Post CPAP initiation. Together, these technologies may provide objective markers of impairment.

Methods Patients undergoing OSA assessment at Royal Papworth Hospital between August 2023 and August 2024 were



Abstract S78 Figure 1 Violin plots displaying improvement in (a) REM sleep proportion, and (b) REM sleep duration, for all recordings and individual participants across treatment conditions. Pre-CPAP (red) and Post-CPAP (blue). Coloured shapes / lines reflect the mean values of individual participants

invited to participate in a study of driving and sleep quality. Participants downloaded the Insights smartphone application (Sentiance), which passively recorded braking, acceleration and turning metrics during driving. A subset additionally used a portable EEG headband (Dreem 2/3, Beacon Biosignals) to assess nightly sleep quality Pre/Post CPAP initiation. Data were analysed using linear mixed-effects models.

Results 48 participants (mean age 54 years; 37% female; mean ODI 25.4; mean ESS 13.6) completed 7,134 journeys (>1,800 hours). Post-CPAP driving was associated with significant changes in 6/9 driving metrics: decreases in brake duration (P<0.0001), acceleration duration (P<0.0001), significant acceleration frequency (P<0.05), turn magnitude (P<0.0001), turn duration (P<0.0001), and significant turn frequency (P<0.0001). Seven participants (mean age 63 years; 29% female; mean ODI 35.7; mean ESS 15.6) completed 144 nightly EEG sleep assessments. Post-CPAP recordings demonstrated increased REM duration (P<0.0001), and reductions in micro-arousals (P<0.0001) and sleep stage shifts (P<0.0001).

Discussion These findings demonstrate the feasibility of passive smartphone telematics for longitudinal assessment of driving impairment in OSA. The portable EEG findings suggest that improvements in sleep quality may represent a plausible mechanism underlying the observed behavioural changes. Together, these approaches may support ecologically valid monitoring of treatment response and recovery following CPAP initiation, and may contribute to approaches for assessing driving-related impairment and recovery within sleep medicine.

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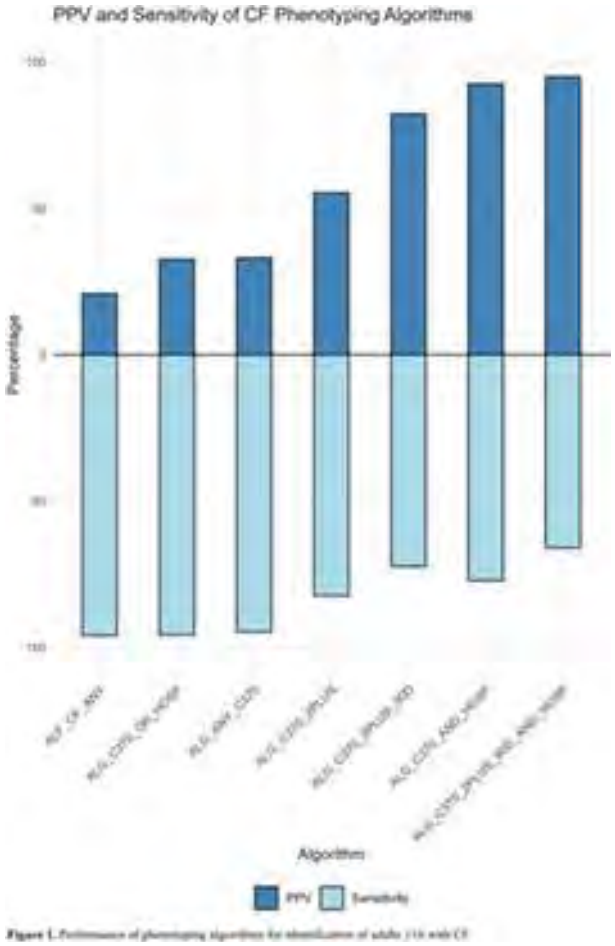
Advances in cystic fibrosis

S79 **DEVELOPMENT AND VALIDATION OF PHENOTYPING ALGORITHMS FOR ACCURATE IDENTIFICATION OF ADULTS WITH CYSTIC FIBROSIS IN ELECTRONIC HEALTH RECORDS**

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10.1136/thorax-2026-BTSabstracts.85

Background As survival improves in people with cystic fibrosis (PwCF), concerns are emerging about diseases of aging. Cystic fibrosis (CF) registries are well validated epidemiological tools but have limited data on emerging morbidities. As most CF epidemiological research has been conducted in CF registries to date, there are limited validated algorithms for CF case ascertainment, which hampers ability to understand epidemiology of age-associated conditions in CF.



Abstract S79 Figure 1 Performance of phenotyping algorithms for identification of adults ≥16 with CF

Objectives To develop and validate an algorithm for identification of adults with CF using linked electronic health records
Methods We harnessed the unique linkage between SAIL Databank and the UK Cystic Fibrosis Registry (CFR) to link the CFR with the Welsh Longitudinal General Practice (WLGP) dataset and hospital admissions data. Adult PwCF with a CFR annual review were included. Seven algorithms were developed using combinations of CF-specific Read codes, temporal separation between codes, and CF-related hospital admissions. Registry-confirmed cases of CF were used as the reference standard. Algorithm performance was evaluated using sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).
Results The SAIL cohort included 508 registry-confirmed PwCF and over 3.9 million adults without CF. Algorithm performance varied considerably depending on the balance between sensitivity and precision (see figure 1). The least restrictive algorithm (any CF code) achieved the highest sensitivity (95.9%) but a PPV of 20.8%. The most restrictive algorithm with the highest specificity required ≥2 C370 CF codes separated by ≥90 days and a CF-related hospital admission (ICD-10 code) and achieved the highest PPV (94.9%) with 65.9% sensitivity. An algorithm including ≥ CF read code in the C370 family and hospital admission performed best with high PPV (92.5%) and well-preserved sensitivity (77.1%).
Conclusions We utilised linked CFR and primary care datasets to define the performance of various diagnostic phenotyping algorithms. Algorithms incorporating repeated CF coding and hospital evidence greatly reduce the number of false positives identified, while still identifying a high proportion of PwCF. For the first time, these findings provide a validated framework for future epidemiological studies investigating long-term health outcomes in PwCF in datasets not linked to CF registries.

S80 SUBJECTIVE EVALUATION OF SPUTUM CHARACTERISTICS SHOWS POOR INTEROBSERVER RELIABILITY

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10.1136/thorax-2026-BTSabstracts.86

Background Within Cystic Fibrosis (CF) care, sputum appearance is routinely described in patient notes, with changes linked to clinical deterioration. Aliberti et al[1] demonstrated, using the 4-point Murray Scale, that sputum colour in non-CF Bronchiectasis is strongly predictive of future exacerbations, highlighting the need for more standardised descriptions of sputum colour. As part of a program to address this, we examined the range and consistency of current terms used to describe sputum samples.
Methods We surveyed clinical staff working in two large UK adult and paediatric CF centres. The first section of the digitally-administered survey required respondents to describe appearance of five sequential sputum photographs using a maximum of three words with no prompting. To assess consistency, the first and fifth photographs were the same. The second section used the photographs from the Murray Sputum Colour Chart and required a maximum of two words to describe the samples. Finally, new sputum sample photographs were presented alongside directed single-option descriptors from the Murray scale. As before, repeated photographs were included.
Results Responses were collected from 45 staff: 20 Doctors, 18 Physiotherapists, 6 Nurses and 1 other. 44% of staff had over 10 years' experience in their current role.

In the first section, a total of 40 different sputum appearance descriptors were used. Descriptors used for the identical photographs 1 and 5 were different in 14 (31.1%) cases.

In section 2, 31 different terms were used to describe the photographs from the Murray Chart. In section 3, respondents agreed with the mode in 67-96% cases. Even with the scale, different responses were given for the same photograph in 13.3% of cases.
Conclusion A wide range of terms are used to describe sputum appearance and were not consistently applied, even by the same respondents describing identical samples. Use of a simple ordinal scale improved agreement, although significant inconsistencies remained. For sputum colour to be a robust measure of clinical change, more reliable and repeatable assessment methods are required.

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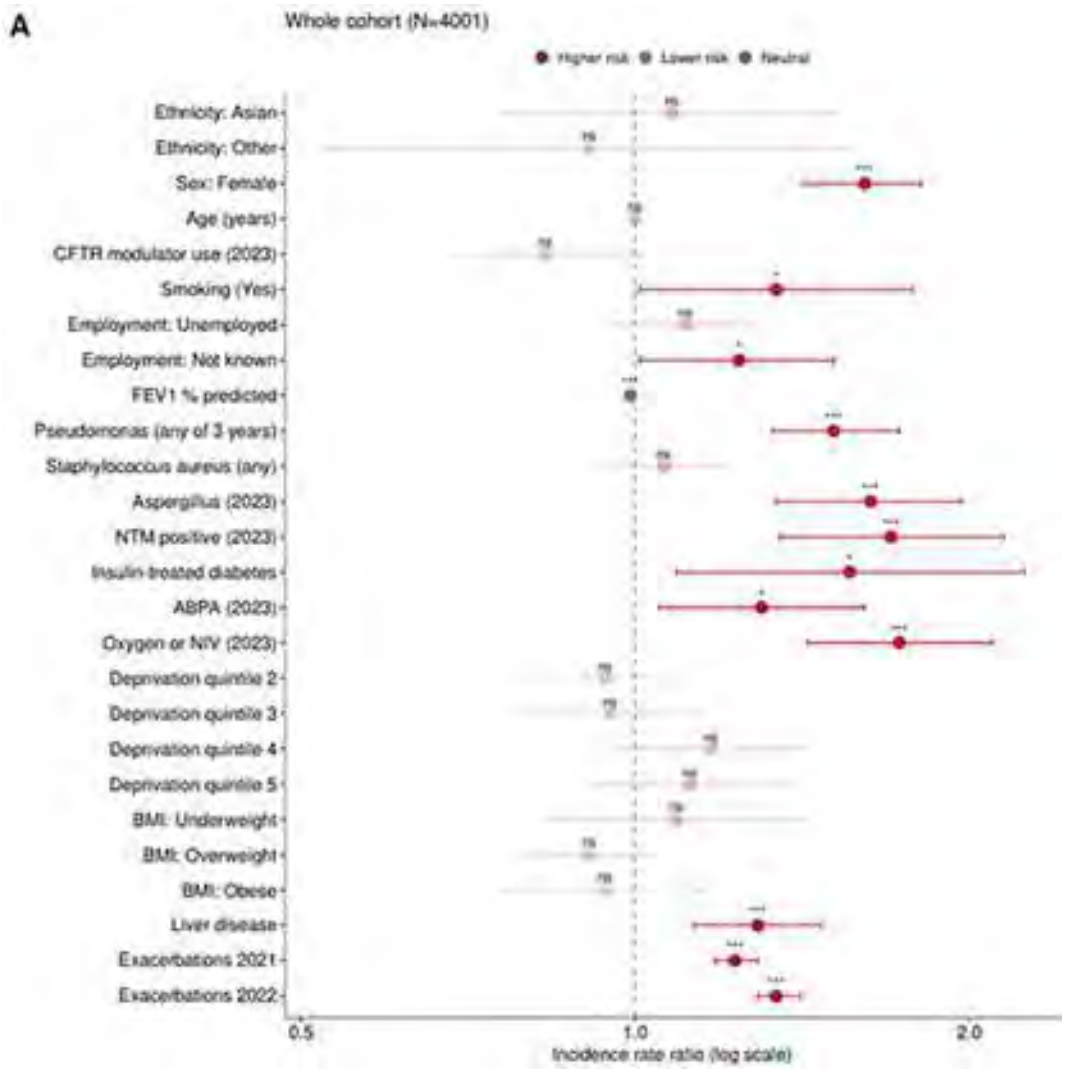
S81 FACTORS ASSOCIATED WITH RESPIRATORY EXACERBATIONS IN ADULTS WITH CYSTIC FIBROSIS IN THE POST-MODULATOR ERA – A UK REGISTRY DATA STUDY

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10.1136/thorax-2026-BTSabstracts.87

Introduction and Objectives The introduction of modulator therapies has greatly improved the respiratory health of many of those with cystic fibrosis (CF). However, respiratory exacerbations still occur in around a quarter of adults with CF and remain an important driver of morbidity and mortality. A better understanding of the factors associated with respiratory exacerbations remains essential.

Using data obtained from The UK Cystic Fibrosis Registry (a database of patient information that holds data from over 99% of people with CF in the UK), we examined factors associated with respiratory exacerbations.
Methods Cross-sectional data were obtained from the UK CF registry for all adults (those under the care of an adult centre and aged 19 or over in 2021) from the 2021 to 2023 data period. We used negative binomial regression to investigate covariates associated with annual exacerbation count in the 2023 data year in three nested cohorts: (1) the whole population; (2) those treated with CF modulators and (3) those treated with CF modulators who were pseudomonas positive. Exacerbations were defined as a course of IV antibiotics, received either at



Abstract S81 Figure 1 Incidence rate ratios (IRR) for exacerbations, with 95% confidence intervals from negative binomial regression from whole population (N=4,001)

home or in hospital, that were marked as given for a pulmonary exacerbation. Covariates investigated included demographic, socioeconomic and lifestyle factors, comorbidities and infection, lung function, nutrition, treatment and support, and exacerbation history.

Results Data were obtained for 4001 adults with CF. Of these, 3573 were CFTR modulator users (89.3%) and 1947 (48.6%) were CFTR modulator users who were chronically infected with Pseudomonas aeruginosa. Figure 1 shows the incidence rate ratios for exacerbations associated with each covariate for the whole population cohort (N=4001).

Higher FEV1% predicted was the only factor associated with reduced exacerbations across all cohorts. Prior exacerbation history was the strongest and most consistent risk factor across all cohorts. Other risk factors included female sex, smoking, oxygen or NIV use, comorbidities including insulin-treated diabetes, liver disease and infection. CFTR modulator use was non-significant after adjusting for FEV_1% and *Pseudomonas* status.

Conclusions These data provide insight into the factors associated with exacerbations in adults with CF in the post-modulator era, and have potential to guide targeted clinical management.

S82 ASSOCIATIONS BETWEEN PERCEPTION OF EXACERBATIONS, VIRAL INFECTION AND SYMPTOM BURDEN IN CYSTIC FIBROSIS

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10.1136/thorax-2026-BTSabstracts.88

Background Pulmonary exacerbations (PEx) are a major cause of morbidity in cystic fibrosis (CF). Respiratory viral infections (VRIs) are possible triggers of PEx but are also frequently detected in clinically stable individuals. This study explores associations between respiratory viral detection, symptoms, and perceived exacerbations in pwCF.

Methods Clinically stable adults with CF were recruited to the CF-Tracker study, an ongoing longitudinal observation-

al study investigating PEx. Participants completed fortnightly home monitoring over 6-months, including spirometry, saliva sampling, respiratory viral PCR swabs, and the Cystic Fibrosis Respiratory Symptom Diary (CFRSD-CRISS). Additional questionnaires assessed illness perception and lifestyle exposures. Participants also completed additional sampling during periods of worsening symptoms.

Results 544 viral swabs were collected between May 2025-April 2026. 38 (7.1%) were positive for viral infection. Rhinovirus was the most frequently detected virus (58%), followed by respiratory syncytial virus (RSV, 15%) and SARS-CoV2 (13%). 68% of positive samples were obtained during routine scheduled sampling (n=26), though this only constituted 5% of all routine samples. By contrast, 23% (n=12) of symptom-triggered samples were PCR-positive. RSV was found in 7.7% of all symptomatic episodes.

Full questionnaire data were available for 31 virus-positive episodes. Tiredness, increased sputum production, and cough were the most commonly reported symptoms. Participants with PCR-confirmed viral infections had significantly higher CFRSD-CRISS scores than the overall cohort (36.96 vs 31.32; p=0.041), indicating greater symptom burden.

On 154 occasions patients reported feeling worse than usual, with 44 of these episodes being identified by respondents as PEx. Seven (16%) participants with a perceived PEx had PCR-confirmed viral infection. Of the 22 participants who identified a cause for their exacerbation, 18 (82%) attributed symptoms to a viral infection, correct in 7 (32%) cases. Attendance at public events (47%) and contact with individuals with respiratory symptoms (26%) were the most frequently reported recent exposures.

Conclusion PCR-confirmed VRIs were associated with increased symptom burden but were also commonly identified during routine sampling. Self-reported illness and presumed viral infection correlated poorly with virological confirmation, highlighting the limitations of symptom-based identification of VRIs in adults with CF. Ongoing follow-up will further define the role of VRIs in pulmonary exacerbations.

S83 LUNG FUNCTION RECOVERY FOLLOWING INPATIENT TREATMENT OF PULMONARY EXACERBATIONS IN ADULTS WITH PRIMARY CILIARY DYSKINESIA

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10.1136/thorax-2026-BTSabstracts.89

Background The impact of pulmonary exacerbations (PEx) and their treatment on forced expiratory volume in 1 second (FEV1) in adults with Primary Ciliary Dyskinesia (PCD) is not well described.

Objectives To assess changes in FEV1 from pre-exacerbation baseline at PEx onset and recovery following inpatient treatment.

Methods Retrospective study of adults with PCD admitted to an inpatient cystic fibrosis ward with PEx for intravenous (IV) antibiotics and physiotherapy between April 2022-April 2026. PEx was defined as ≥3 of: increased cough, change in sputum, increased breathlessness, physician decision to treat with IV antibiotics due to pulmonary symptoms, lethargy, new/increased haemoptysis or fever. Main outcomes were changes in FEV1 and the proportion of patients achieving ≥90% of baseline FEV1 at end of admission.

Results 46 admissions for PEx in 28 patients occurred in the period analysed. 23 admissions (in 19 patients) had baseline, admission and discharge FEV1 data available. Of these, 10 (44%) were in females; mean (range) for: age at admission was 33.5±12 years (19-61); admission length 11±2 days (8-16), baseline FEV1 2.06±0.69L (0.63-3.7L) and FEV1 %predicted 59.5±18.1% (range 17-86%). Sputum cultures were obtained during all admissions and *Pseudomonas aeruginosa* was isolated in 14 (61%). A respiratory virus was identified in 4 (18%) of 22 admissions where PCR testing was conducted. Mean relative decline in FEV1 at admission compared with baseline was 13.2%±12.9. Relative drop in admission FEV1 versus baseline was ≥5% in 15 (65%) admissions and ≥10% in 12 (52%) admissions. Mean relative increase in FEV1 from admission to discharge was 10.4%±12.4 (range -8.2-50.5%). Mean discharge FEV1 was 95.4%±13.6 of baseline (range 71.4-124.3%). Discharge FEV1 was ≥90% of baseline in 14 (61%) admissions and ≥85% of preadmission baseline in 18 (78%).

Conclusions PEx in adults with PCD are associated with acute decline in FEV1 that improves following inpatient treatment with IV antibiotics and physiotherapy. However, despite inpatient treatment, a significant number of cases do not recover to ≥90% of baseline FEV1. Larger studies are required to assess the potential impact of PEx on longer-term lung function decline in adults with PCD and predictors of lung function recovery.

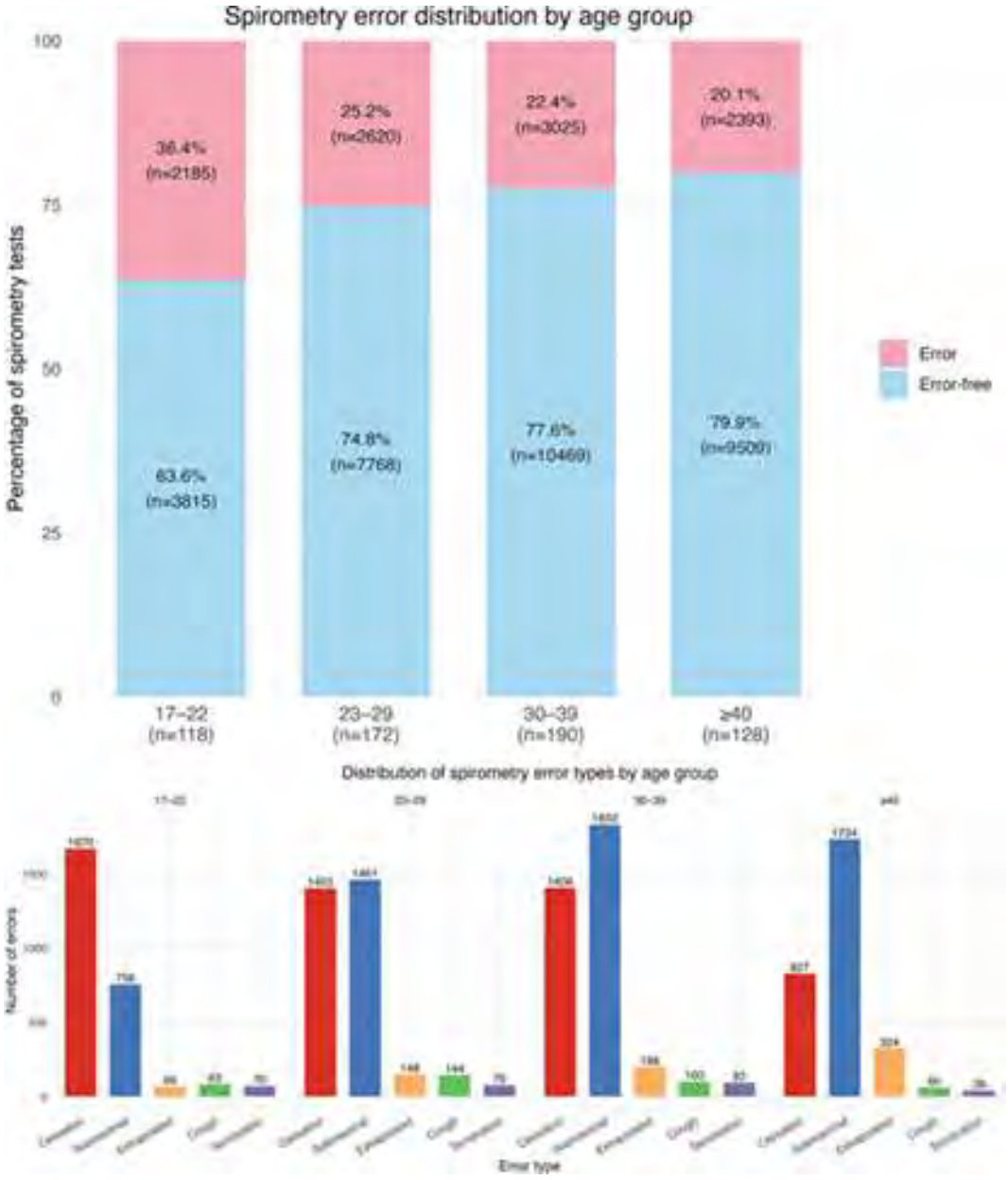
S84 INCREASING AGE IS ASSOCIATED WITH IMPROVED HOME SPIROMETRY QUALITY AND DISTINCT ERROR PHENOTYPES IN PEOPLE WITH CYSTIC FIBROSIS

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10.1136/thorax-2026-BTSabstracts.90

Background Remote monitoring has become increasingly integrated into cystic fibrosis (CF) care, with home spirometry now widely used by most people with CF (pwCF). To ensure accuracy of assessment and reduce effort for users, it is important to achieve an error-free manoeuvre where possible. We investigated the association between age and home spirometry error patterns in pwCF attending our adult CF centre.

Methods Home spirometry data collected using the NuvoAir (Sweden) platform between 2020 and 2024 were analysed. Error types included submaximal effort (or Time-to-Peak), cessation, cough, early termination and back-extrapolated volume (BEV), as per the ATS/ERS 2019 technical statement. Age was categorised a priori: 17-22, 23-29, 30-39, and ≥40 years, and treated as a time-varying covariate, allowing individuals to contribute tests to multiple age groups over time. Mixed-effects logistic regression was used to account for repeated measures and data clustering within pwCF. A spirometry test could contain multiple errors. Therefore, percentages were not mutually exclusive (denominator used was the number of erroneous tests).



Abstract S84 Figure 1

Results 41,784 spirometry tests were performed by 469 pwCF (median age 33 years [IQR 26–41]). The proportion of error-free tests increased progressively across age groups (p<0.001) (figure 1).

Compared with pwCF aged 17-22 years, the odds of achieving an error-free test increased in those aged:

23-29 years (OR 1.72, 95% CI 1.48-1.99),

30-39 years (OR 2.13, 95% CI 1.77-2.56),

and ≥40 years (OR 3.00, 95% CI 2.42-3.71) (all p<0.001).

As a sensitivity analysis, age was analysed as a continuous variable using mixed-effects regression adjusted for multiple covariates, showing increasing OR of 1.03 per additional year of age (95% CI 1.01-1.04, p <0.001).

Cessation errors were most common in pwCF aged 17-22 years: 1,670 (76.4%) vs 756 (34.6%) submaximal errors. With increasing age, the error phenotype shifted towards submaximal errors. In pwCF aged ≥40 years, submaximal errors exceeded cessation: 1,734 (72.5%) vs 827 (34.6%). The frequencies of other errors were similar across age groups.

Conclusion Increasing age was associated with higher odds of achieving error-free home spirometry. Error patterns also differed by age. These findings may inform age-specific training strategies to improve home spirometry quality.

Randomised Controlled Trial Session

S85 CORMORANT: PROSPECTIVE DIAGNOSTIC ACCURACY STUDY OF HAND-HELD CAPNOGRAPHY USING N-TIDAL DIAGNOSE COMPARED TO SPIROMETRY FOR THE DIAGNOSIS OF COPD IN UK PRIMARY AND COMMUNITY CARE SETTINGS

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10.1136/thorax-2026-BTSabstracts.91

Introduction COPD remains substantially underdiagnosed in the UK, with many patients diagnosed at first exacerbation or after irreversible lung damage has occurred. Although spirometry is the current diagnostic standard, access to high-quality testing and interpretation has become increasingly challenging in primary care. Large gaps or delays in community spirometry provision leave many patients without a diagnosis or appropriate treatment. The NHS 10-year plan seeks to shift care from hospital to community, accelerate adoption of digital technologies, and prioritise earlier diagnosis and prevention, and there is an urgent need for alternative diagnostic approaches that can be implemented in routine practice.

Methods N-Tidal Diagnose 1 (NTD1) is a novel, Class IIa EU MDR certified medical device used in combination with a hand-held capnometer (the N-Tidal Handset) as a rapid point-of-care diagnostic test for COPD. NTD1 processes a breath record, produced with 75 seconds of effort-independent tidal breathing through the handset, and applies machine learning-derived models to provide a diagnostic output. We present the findings of the CORMORANT (COPD tRansforMation of diagnOstic pathways in pRimary cAre using N-Tidal) study (ISRCTN 18133836), a prospective diagnostic accuracy study evaluating NTD1 for COPD diagnosis in adults undergoing spirometry for suspected COPD in primary or community care.

Results Over 500 participants have been recruited across 27 GP surgeries, Community Diagnostic Centres and diagnostic hubs. The study compares the diagnostic performance of NTD1 against a consensus reference standard based on independent, blinded interpretation of spirometry and clinical history. Alongside diagnostic accuracy, the study is evaluating the quality of routine primary care spirometry, patient and clinician acceptability, implementation feasibility and is collecting data to inform future health economic evaluation.

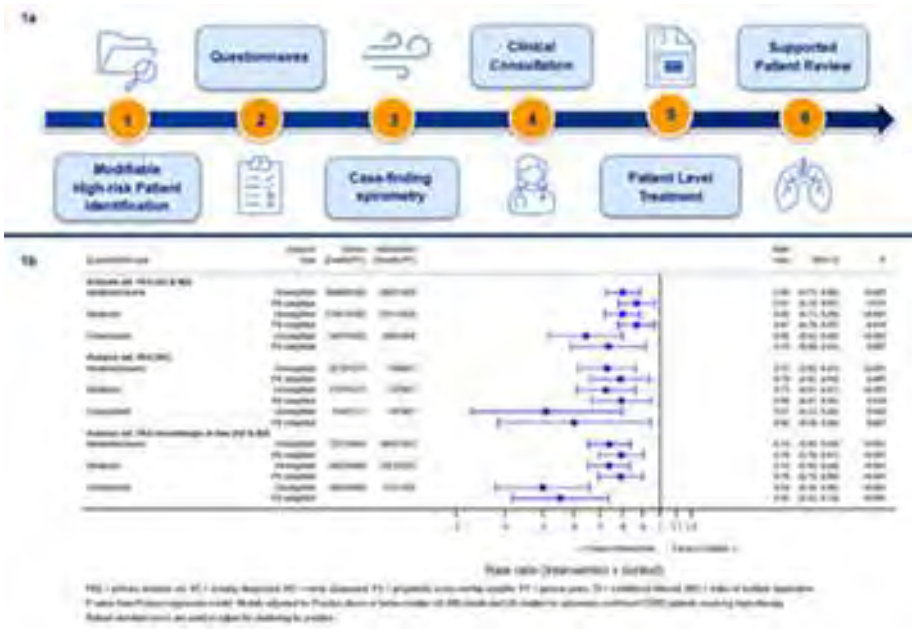
Conclusion CORMORANT will provide the first large-scale independent real-world evidence evaluating NTD1 in its intended setting. This study addresses a major evidence gap limiting adoption of AI-enabled COPD diagnostics within the NHS. The results will be directly relevant to respiratory clinicians, physiologists, commissioners and policymakers responsible for future COPD diagnostic pathways, with translation into practice already being discussed with national stakeholders awaiting CORMORANT findings.

S86 IMPROVING OUTCOMES FOR PRIMARY CARE PATIENTS WITH UNDIAGNOSED OR DIAGNOSED COPD ON MONOTHERAPY OR LESS THROUGH TARGETED CASE-FINDING AND PROACTIVE OPTIMISATION: RESULTS FROM THE PREVAIL CLUSTER-RANDOMISED CLINICAL TRIAL

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10.1136/thorax-2026-BTSabstracts.92

Background COPD patients with recent exacerbations and suboptimal maintenance treatment, e.g. monotherapy (deemed modifiable high-risk [MHR]), are at increased risk of exacerbations compared to non-MHR patients. The CONQUEST quality improvement programme (QIP) provided case-finding spirometry, clinical review for assessment and treatment optimisation, and follow-up for MHR patients with COPD; supporting primary care practices to deliver guideline-based care. The PREVAIL cluster randomised trial investigated the impact of the CONQUEST QIP on patients with newly diagnosed (ND) and already diagnosed (AD) MHR COPD.



Abstract S86 Figure 1 Results panel. 1a: Exacerbation rates in MHR patients with COPD; 1b: Rates of steroid courses related to exacerbations in MHR patients with COPD

Aim To compare the annual rate of moderate/severe COPD exacerbations and steroid courses between MHR patients prescribed monotherapy or less at baseline in CONQUEST practices versus usual care.

Methods General practices in England were randomised 1:1 to CONQUEST (intervention) or usual care (control). Eligible patients were aged 40-80 years, with recorded COPD diagnoses or positive smoking histories, >=2 moderate or >=1 severe exacerbation in the prior 24 months (>=1 in last 12 months) and prescribed monotherapy or less at baseline. ND patients were identifid via CONQUEST case-finding spirometry or usual care. Exacerbations with subsequent hospitalisation/oral steroids/antibiotics 8-28 days after treated exacerbation were defined as complicated. Electronic health records were analysed using propensity score weighted models.

Results 85 intervention and 90 control practices contributed 5,631 patients (mean follow-up: 24 months). Exacerbations reduced by 21% (p<0.001) in AD and ND patients, with a similar effect observed in both cohorts (AD: 22%, p=0.001; ND: 23%, p=0.006) (figure 1a). Complicated exacerbations reduced by 45% (p<0.001) in AD and ND patients, and by 47% (p<0.001) and 44% (p=0.023) in AD and ND cohorts respectively. Exacerbation-related steroid courses reduced by 34% (p=0.009) in AD patients, with no effect observed in the ND (rate ratio 1.04, p=0.817) or overall (rate ratio 0.80, p=0.073) cohorts (figure 1b).

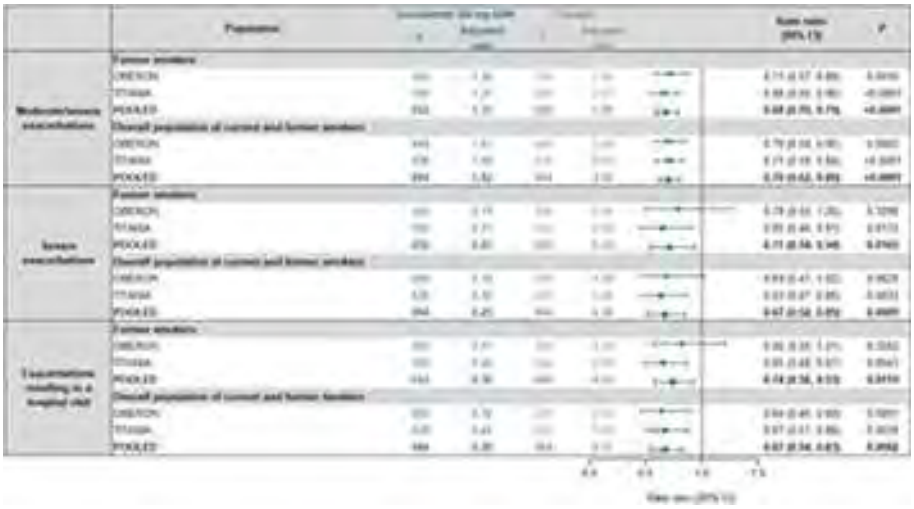
Conclusions The CONQUEST QIP reduced exacerbations by a fifth, and complicated exacerbations by almost half in MHR patients with already and newly diagnosed COPD on monotherapy or less at baseline. The number of exacerbation-related steroid courses reduced by a third in already diagnosed patients in the same population. MHR patients on monotherapy or less represent a key population for proactive treatment optimisation.

S87 TOZORAKIMAB FOR PREVENTION OF EXACERBATIONS IN COPD: POOLED ANALYSIS OF THE OBERON AND TITANIA TRIALS

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10.1136/thorax-2026-BTSabstracts.93

Introduction Tozorakimab, an anti-IL-33 monoclonal antibody, inhibits both oxidized and reduced IL-33. In the replicate phase 3 OBERON (NCT05166889) and TITANIA (NCT05158387) trials, tozorakimab significantly reduced the annualized rate of moderate/severe exacerbations versus placebo in former smokers and in the overall populations of current and former smok-



Abstract S87 Figure 1 Annualized rate of exacerbation-related endpoints in former smokers and the overall population of current and former smokers (pooled analysis from OBERON and TITANIA)
P values are multiplicity-controlled and inferential for individual studies, and exploratory and not multiplicity-controlled for the pooled analysis. Moderate exacerbations were worsening COPD symptoms requiring systemic glucocorticoids for >=3 days, antibiotics or emergency department visits for <24 hours with intensive treatment; severe exacerbations were those leading to hospitalization for >=24 hours or death. Exacerbations resulting in a hospital visit were worsening COPD symptoms resulting in hospitalization and/or an emergency department visit of any duration, or death.

ers with COPD and a history of exacerbations. This prespecified analysis examined pooled efficacy and safety data from OBERON and TITANIA.

Methods In OBERON and TITANIA, eligible patients were current or former smokers with COPD and a history of exacerbations in the past year while on stable standard-of-care inhaled maintenance therapy. There were no selection criteria related to blood eosinophil count. This primary pooled analysis evaluated add-on subcutaneous tozorakimab 300 mg versus placebo every 4 weeks (Q4W) for 52 weeks. The annualized rate of moderate/severe exacerbations was assessed in former smokers (primary endpoint) and in the overall population of current and former smokers (first key secondary endpoint). The annualized rates of severe exacerbations (hospitalization >24h or death) and exacerbations resulting in a hospital visit (hospitalization and/or an emergency department visit of any duration, or death) were secondary endpoints.

Results The pooled dataset included 1750 patients (tozorakimab Q4W: 884, placebo: 866). Baseline characteristics were balanced between treatment groups. Tozorakimab Q4W reduced the annualized rate of moderate/severe exacerbations in former smokers and in the overall population of current and former smokers (figure 1). The annualized rates of severe exacerbations and exacerbations resulting in a hospital visit were also reduced versus placebo in former smokers and in the overall population of current and former smokers (figure 1). In subgroup analyses, a clinically meaningful benefit was observed independent of screening blood eosinophil count thresholds, baseline severity of airflow obstruction and smoking status. Adverse events were generally balanced across treatment groups.

Conclusion In this prespecified pooled analysis, add-on treatment with tozorakimab led to a 30% reduction in the rate of moderate/severe exacerbations in a broad population of current or former smokers with COPD and a history of exacerbations. Similar reductions were observed in the rate of severe exacerbations and exacerbations resulting in a hospital visit. Tozorakimab was well tolerated.

S88 IMPALA-2: A RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED CLINICAL TRIAL OF ONCE-DAILY INHALED MOLGRAMOSTIM IN PATIENTS WITH AUTOIMMUNE PULMONARY ALVEOLAR PROTEINOSIS (APAP)

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Background aPAP is a rare chronic lung disease caused by autoantibodies blocking granulocyte-macrophage colony-stimulating factor (GM-CSF) signalling, resulting in alveolar macrophage dysfunction and surfactant accumulation. Molgramostim inhalation solution (MOL) is a recombinant human GM-CSF in development as a pharmacotherapy for aPAP. IMPALA-2 is a randomised, double-blind, placebo-controlled phase 3 trial comprising a 48-week double-blind (DB) treatment period (completed) followed by a 96-week open-label (OL) treatment period (ongoing) to assess the long-term efficacy and safety of MOL. Here we report results from the first 48 weeks of the 96-week OL period.

Methods aPAP patients were randomised to receive inhaled MOL (300 micrograms) or placebo (PBO) once daily during the DB period; all patients received daily MOL during the OL period. Efficacy was assessed by change from baseline in percent predicted diffusing capacity of the lungs for carbon monoxide (DLco%) and by St. George's Respiratory Questionnaire Total

(SGRQ-T) and Activity (SGRQ-A) Scores assessing respiratory health-related quality of life (HRQoL).

Results Of 164 patients receiving MOL (n=81) or PBO (n=83) during the DB period, 160 (98%) completed the DB period and continued into the OL period. At Week 48, the mean (standard error) changes from baseline in DLco% were 11.6 (1.4) for the MOL group and 3.7 (1.5) for the PBO group; the difference in least-squares mean (LSM) changes between the MOL and PBO groups was 6.9 (P=0.0008). During the OL period, patients receiving MOL during both DB and OL periods (MOL-MOL) continued to show improvement in DLco% through Week 96; the mean increase during Weeks 48-96 was 2.8 (1.2) with an overall mean increase from baseline (Weeks 0-96) of 14.7 (1.6). In PBO patients crossing over to MOL in the OL period (PBO-MOL), DLco% increased during the OL treatment period through Week 96; the mean increase in DLco% during Weeks 48-96 was 8.8 (1.6). Continued improvements in SGRQ were also observed in both treatment groups. Safety and tolerability were consistent with the DB period.

Conclusions Long-term treatment with MOL continuously improved pulmonary gas transfer and respiratory HRQoL in aPAP patients. In addition, PBO cross-over patients demonstrated improved pulmonary gas transfer and HRQoL.

S89 REINTRODUCTION OF ANTI-TUBERCULOSIS THERAPY FOLLOWING DRUG-INDUCED LIVER INJURY: STUDY PROTOCOL FOR A RANDOMISED CONTROLLED TRIAL (TB-DILI)

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Funder NIHR HTA.

Background Drug-induced liver injury (DILI) is the commonest serious adverse effect of standard anti-tuberculosis therapy (ATT) and frequently necessitates interruption of treatment. Following recovery from ATT-related DILI, treatment must be reintroduced. Current practice is informed by two guideline-recommended strategies: sequential full-dose reintroduction of a pyrazinamide (Z)-containing regimen as recommended by NICE, and sequential full-dose reintroduction of a non-pyrazinamide-containing regimen as recommended by the American Thoracic Society. Both strategies are widely used in routine practice, yet recommendations are based on weak evidence, with substantial variation in clinical practice. Pyrazinamide-containing regimens may be associated with a higher risk of recurrent DILI but allow shorter overall treatment duration, whereas non-Z-containing regimens may reduce hepatotoxicity risk at the expense of longer treatment.

Methods TB-DILI is a pragmatic, multicentre, individually randomised, open-label, parallel-group superiority trial with concealed allocation (1:1). Adults who experienced DILI while receiving standard four-drug ATT for active tuberculosis and who were medically suitable for treatment reintroduction were randomised to sequential full-dose reintroduction of either a Z-containing regimen (ethambutol, isoniazid, rifampicin, pyrazinamide) or a non-Z-containing regimen (ethambutol, isoniazid, rifampicin). Participants were followed for 12 months following randomisation. The primary outcome is recurrence of DILI. Secondary outcomes include severity of recurrent DILI, clinical cure, treatment duration, ATT conformance, adverse events, mortality, quality of life (QoL) measured using EQ-5D-5L, healthcare resource use, and cost-effectiveness.

Results 49 sites across the UK participated in TB-DILI plus one site in India. Between Nov 2022 and May 2025, 79 participants were enrolled (mean age 42.4 years; 44% male; 14% White, 34% Indian, 15% Black African, 11% Pakistani, 11% Other Asian). DILI-severity at randomisation was graded as mild, moderate and severe in 68%, 32% and 0%, respectively; 81% reported DILI-related symptoms, nausea most commonly (67%). 91% were treated per protocol. Completion of follow-up is expected by end June 2026 with preliminary results anticipated Nov 2026.

Discussion Trial results will provide data on DILI recurrence rates and QoL for the two main reintroduction options in current practice in the NHS. These data will enable patients and their clinicians to make informed choices regarding the most appropriate strategy to adopt.

Trial registration Registered with the International Standard Randomised Controlled Trial Number (ISRCTN), registry ID: ISRCTN92067975, 25 August 2022.

S90 RAPTOR-F: A MULTICENTRE RANDOMISED FEASIBILITY TRIAL OF RAPID-GUIDED MANAGEMENT IN PLEURAL INFECTION

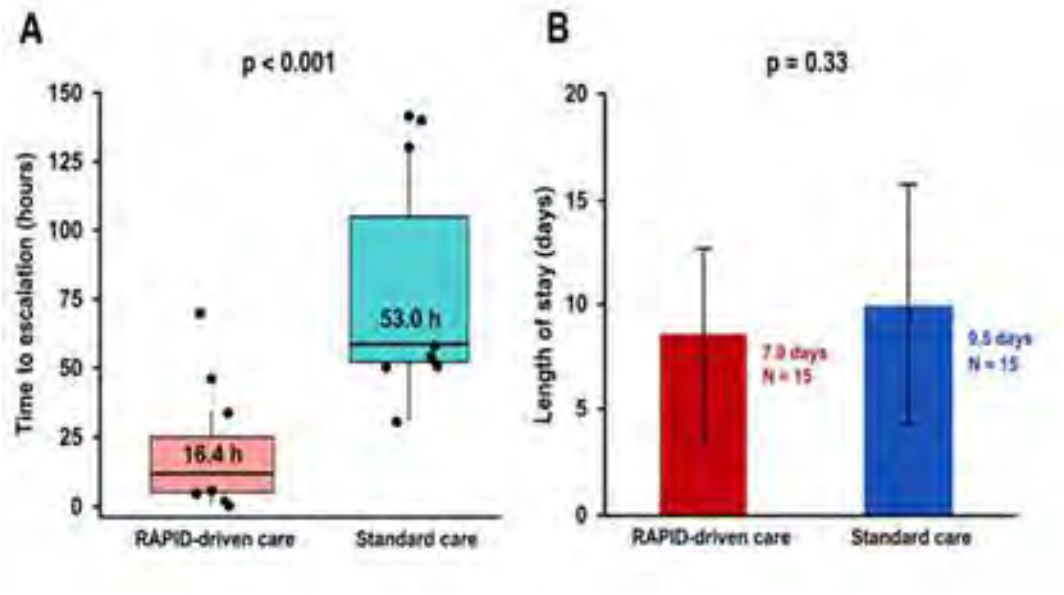
[1,2,3,4]A Elsheikh, [1,2]M Bhatnagar, [1]B Iqbal, [2]E Hedley, [1]D Addala, [1,2]O Walsh, [1]D Abdul-Rahman, [1]F Al-arrayed, [2]J Sadler-Ladell, [2]T Panduro, [2]M Dobson, [1]I Mechie, [5]C Pearce, [6]A Budds, [6]P Ridge, [6]M Mwadeyi, [7]C Hardy, [2]W Talbot, [7]T Sarkar, [5]Z Sein, [6]V Lennox, [8]Y Feng, [8]J Chu, [8]N Kanellakis, [6]C Daneshvar, [5]J Corcoran, [9]R Miller, [10]G Collins, [1,2]R Hallifax, [4,7]E Mishra, [1,2,3,8]NM Rahman, [1,2]EO Bedawi. *[1]Oxford Pleural Unit, Oxford University Hospitals, Oxford, UK; [2]Oxford Respiratory Trials Unit, Nuffield Department of Medicine, University of Oxford, Oxford, UK; [3]Oxford National Institute for Health and Care Research Biomedical, Research Centre, Oxford, UK; [4]University of East Anglia, Norwich Medical School, Norwich, UK; [5]Royal Devon University Healthcare NHS Foundation Trust, Devon, UK; [6]University Hospitals Plymouth NHS Trust, Plymouth, UK; [7]Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, UK; [8]Chinese Academy of Medical Science Oxford Institute, Nuffield Department of Medicine, Medical Sciences Division, University of Oxford, Oxford, UK; [9]University College London, Institute for Global Health, London, UK; [10]Department of Applied Health Sciences, College of Medicine and Health, University of Birmingham, UK*

10.1136/thorax-2026-BTSabstracts.96

Background The RAPID score is the only externally validated prognostic tool in pleural infection. However, its use to guide treatment has not been evaluated.

Objective To assess the feasibility and acceptability of a multicentre randomised trial comparing RAPID-guided management with standard care (SC) in pleural infection, and to explore potential effects on treatment pathways and patient outcomes.

Methods RAPTOR-f was a prospective multicentre open-label randomised feasibility trial conducted across four UK hospitals (ISRCTN71615131). Adults with pleural infection were randomised 1:1 to SC or RAPID-guided management. In the



Abstract S90 Figure 1 (A) Time to treatment escalation (hours), presented as median (IQR), according to treatment allocation.; (B) Length of hospital stay (days), presented as mean +/- SD, according to treatment allocation

intervention arm, low-risk patients received conservative management with consideration of early discharge, while intermediate/high-risk patients underwent early escalation to intrapleural enzyme therapy (IET) and parallel surgical referral. Primary outcomes were recruitment feasibility and acceptability, assessed against prespecified progression criteria including recruitment, protocol adherence and qualitative interview completion. Secondary outcomes included time to treatment escalation, length of stay, safety and patient-reported outcomes. Analyses were on an intention-to-treat basis.

Results Of 33 eligible patients, 30 (91%) were randomised, 29 (97%) completed 2 weeks follow-up and 28 (93%) adhered to their allocated pathway. Prespecified progression criteria for recruitment, data completeness and interview completion were met. Median time to treatment escalation was shorter with RAPID-guided management than SC (16.4 vs 53.0 hours; $p<0.001$) (figure 1A). Mean length of stay was shorter (7.9 versus 9.5 days) (figure 1B). Serious adverse events occurred in 2/15 (13.3%) RAPID-guided and 1/15 (6.7%) SC participants. Pain, breathlessness and EQ-VAS improved over 90 days in both groups without significant between-group differences. EQ5D-VAS improved over time ($p<0.001$), with no treatment effect by group ($p=0.258$) or treatment-by-time interaction ($p=0.071$).

Conclusion RAPTOR-f is the first randomised trial to evaluate a risk-stratified management strategy in pleural infection. The study demonstrated that RAPID-guided management is feasible across multiple centres and influenced clinical decision making and treatment escalation pathways, potentially accelerating treatment escalation and reducing hospital stay without worsening patient reported outcomes. The results support progression to a definitive multicentre trial.

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From the old to the new: innovative physiological assessments

S91 INVESTIGATION OF A NON-INVASIVE, NON-VOLITIONAL INDEX OF DIAPHRAGM STRENGTH USING TWITCH SURFACE MECHANOMYOGRAPHY

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10.1136/thorax-2026-BTSabstracts.97

Introduction Accurately quantifying respiratory muscle strength is clinically important but remains challenging in practice. In amyotrophic lateral sclerosis, the strongest prognostic biomarker is twitch transdiaphragmatic pressure (TwPdi); however, this measurement is invasive, requires specialist expertise, and is not widely available. A reliable non-invasive alternative would therefore be of considerable clinical value.

Aims and Objectives To determine whether surface mechanomyogram (sMMG) responses to anterolateral magnetic phrenic nerve stimulation (TwsMMG) provide a non-volitional, non-invasive index of diaphragm strength.

We hypothesised that TwsMMG and TwPdi amplitudes would be positively correlated during ramped stimulation between 30 and 100% of maximum stimulator output (MSO).

Methods Thirteen healthy adults (BMI 20.0 ± 1.7 kg/m^2, age 24 ± 3 years; 9 female) were studied. sMMG was recorded using triaxial accelerometers placed bilaterally at the 7th/8th intercostal space and over the upper abdomen. Pdi was measured using a dual pressure catheter. TwPdi and TwsMMG were

Abstract S91 Table 1 Correlations between normalised TwPdi and TwsMMG peak-to-peak and RMS amplitudes during incremental stimulation (30%-100% MSO)

Signal	P2P(ρ)	RMS (ρ)
LeftStim_TwsMMG$_L$	0.679*	0.711*
LeftStim_TwsMMG$_R$	0.555*	0.550*
LeftStim _TwsMMG$_{abd}$	0.744*	0.741*
RightStim_TwsMMG$_L$	0.684*	0.700*
RightStim _TwsMMG$_R$	0.733*	0.742*
RightStim _TwsMMG$_{abd}$	0.724*	0.718*
BilateralStim_TwsMMG$_L$	0.879*	0.845*
BilateralStim_TwsMMG$_R$	0.875*	0.881*
BilateralStim_TwsMMG$_{abd}$	0.904*	0.904*

recorded concurrently during ramped bilateral and unilateral phrenic nerve stimulation from 30 to 100% MSO. For each participant, TwPdi and TwsMMG amplitudes were normalised to mean values at 100% MSO, and associations were assessed using Spearman correlation coefficients.

Results Strong positive correlations were observed between TwPdi and TwsMMG amplitudes across the stimulation range (table 1). These associations were consistent across all three sMMG placements and for both peak-to-peak and RMS amplitude measures.

N=13;*P<0.001;TwsMMG$_L$ = Twitch surface mechanomyogram on left, TwsMMG$_R$ = Twitch surface mechanomyogram on right, TwsMMG$_{abd}$ = Twitch surface mechanomyogram recorded on upper abdomen, P2P = peak-to-peak amplitude, RMS = root mean square amplitude.

Conclusion TwsMMG signals are promising non-invasive, non-volitional indices of diaphragm strength. The strong correlation with TwPdi suggests that surface mechanomyography may offer a practical surrogate for invasive pressure measurement. Validation in larger cohorts and in patient populations is required before clinical application.

S92 BEYOND FEV1 FOR THE EARLY DETECTION OF COPD: UNLOCKING THE SILENT ZONE THROUGH NOVEL MEASUREMENT OF SMALL AIRWAYS FUNCTION

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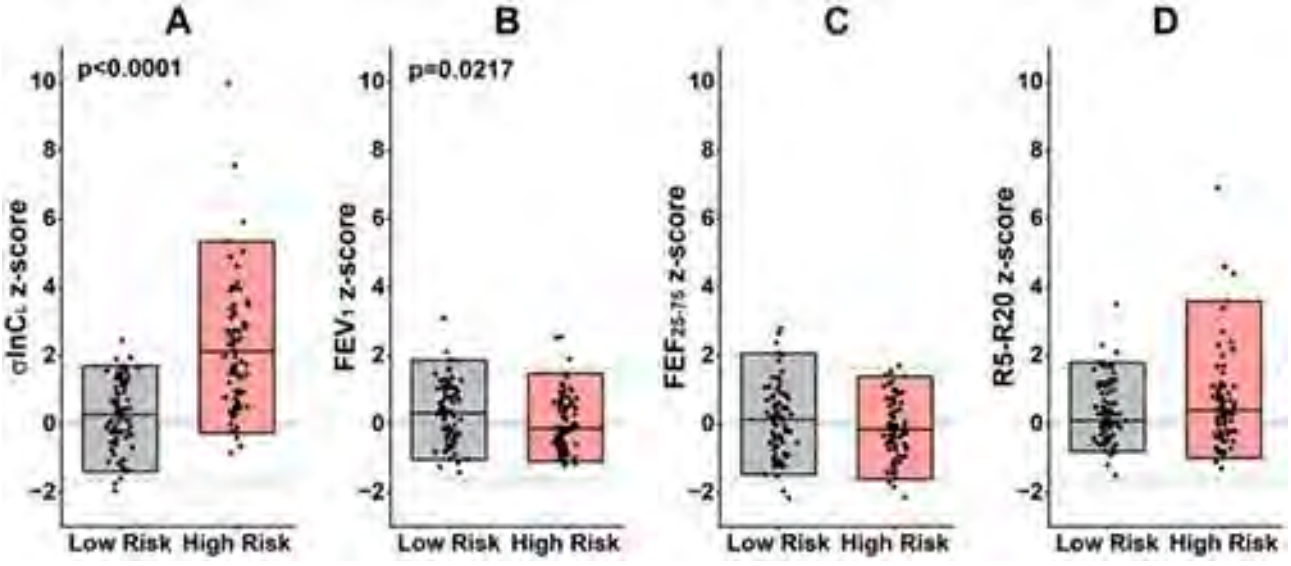
Introduction COPD diagnosis relies on spirometry, which is insensitive to early changes in the small airways, where disease begins and progresses. Irreversible pathology therefore typically occurs before diagnosis.

Computed cardiopulmonography (CCP) non-invasively quantifies ventilation inhomogeneity using highly-accurate respired gas measurements and a mechanistic respiratory-physiology model (Mountain *et al.*, Journal of Applied Physiology, 124:3, 2018). It provides a novel index of small-airway dysfunction, σlnCL, which is abnormal in mild COPD and increases with disease severity (Smith *et al.*, Frontiers in Physiology, 13, 2022).

We investigated whether CCP can detect airway abnormalities in individuals at high risk for COPD earlier than conventional lung function tests.

Methods Current tobacco smokers, aged 25-60 years, with a smoking history of ≥10 pack-years, but without prior respiratory diagnosis or treatment, were recruited as a high-risk population. Participants underwent CCP and other conventional assessments of small-airway function (spirometry and oscillometry) and symptom questionnaires. Results were compared with those from low-risk never-smoking controls.

Results Among 97 smokers, 18 (19%) had abnormal spirometry, either obstructive (FEV$_1$/FVC<0.7) or preserved ratio



Abstract S92 Figure 1 Lung function measures for never-smokers (Low Risk), and smokers (High Risk), with normal spirometry. (A) olnCL z-score, (B) FEV1 z-score, forced expiratory volume in 1 second (C) FEF25-75 Z-score, forced expiratory flow at 25-75% of forced vital capacity, (D) R5-R20 z-score, difference between resistance at 5 and 20Hz. Boxes illustrate median and 5th to 95th percentile. Dashed lines show upper and lower limits of normal. Significant differences between the groups were identified for olnCL and FEV1 z-scores (t-test)

impaired spirometry (FEV_1<80% pred). In these participants, σlnCL was abnormal in 94%, where established small-airway indices FEF_{25-75} (39%), and oscillometry measures R5–R20 (47%) and AX (53%) identified fewer abnormalities.

79 smokers (81%), mean age 47±9 years, had normal spirometry (FEV_1>80% pred and FEV_1/FVC >0.70). These participants were compared with 79 age-matched never-smokers. σlnCL was abnormal (z-score>1.65) in 59% of smokers with normal spirometry, exceeding the sensitivity of other small-airway tests (figure 1). The high-risk group reported more symptoms (p<0.0001) and had lower FEV_1 z-scores (p=0.0217). σlnCL correlated significantly with conventional indices of small-airway dysfunction, including FEF_{25-75} (r=-0.36,p<0.0001) and R5–R20 (r=0.30,p<0.001). However, in those with normal spirometry, σlnCL was the only small-airway index that differed significantly between smokers and never-smokers.

Conclusion The CCP-derived index of inhomogeneity σlnCL can detect lung function abnormalities in people at risk of developing COPD when spirometry and other conventional tests of small-airways function are normal. CCP presents an opportunity to move COPD disease detection - and ultimately therapy - upstream of irreversible airway modelling, and should be further explored in longitudinal cohort studies.

S93 LONG TERM PATTERNS OF AEROBIC CAPACITY CHANGE IN ACHD PATIENTS

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Introduction Adults with congenital heart disease (ACHD) demonstrate a reduced aerobic capacity (AC) compared to healthy populations, with peak $\dot{V}O_2$ shown previously to decline over time. This reduction is usually due to the interactions between their complex cardiac physiology, prior surgery, and peripheral limitations. While differences in exercise capacity between ACHD subtypes are recognised, the long-term rate of decline in AC across groups remains unclear.

Aims To evaluate the longitudinal changes in peak $\dot{V}O_2$ in ACHD patients and determine whether the rate of decline differs between predominant ACHD subtypes.

Methods Retrospective analysis of cardiopulmonary exercise testing (CPET) data from ACHD patients was performed. Patients were grouped according to their predominant congenital heart disease subtype (CI: conduit issues, VI: valve issues, CD: cardiac dysfunction). Peak $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1} \cdot year^{-1}$) values from all patients who completed at least more than one CPET were collected. Comparisons between groups were performed using an F-test and pairwise comparison using Bonferroni adjustment. Differences in mean annual change in peak $\dot{V}O_2$ between ACHD subtypes was assessed using linear regression. Statistical significance was defined as p<0.05.

Results Patient demographics are shown in table 1. There is no difference in peak $\dot{V}O_2$ decline between predominant ACHD type. Pairwise comparisons demonstrated no statistically significant differences between CI, VI and CD types (p>0.05). A non-significant trend towards greater decline in VI was observed (difference -0.92 $ml \cdot kg^{-1} \cdot min^{-1} \cdot year^{-1}$, p=0.083) (table 2; figure 1).

Abstract S93 Table 1 Data are presented as median (interquartile range, IQR) for continuous variables and number (percentage, %) for categorical variables. IQR represents the 25th–75th percentile. CPET = cardiopulmonary exercise testing; ACHD = adult congenital heart disease. RER = Respiratory Exchange Ratio. METS = Metabolic Equivalent of Task. BMI = Body Mass Index. Sys = Systolic Blood Pressure. Dia = Diastolic Blood Pressure. Percentages are calculated based on available data

	n=31
Male sex, (%)	36
Age, years	27 (22-33)
Body Mass Index, kg/m^2	26 (23-30)
Peak VO_2, $mL.kg^{-1}.min^{-1}$	22.4 (17.1-26)
Peak VO_2, % predicted	72 (63-90)
Maximum heart rate, % predicted	85 (80-92)
Peak metabolic equivalents (METs)	6.4 (4.9-7.4)
VO_2/HR, $mL.beat^{-1}$	10 (8-13)
Systolic blood pressure, mmHg	148 (135-175)
Diastolic blood pressure, mmHg	80 (66-86)
VE/VCO_2 slope, units	33 (30-37)
Respiratory exchange ratio (RER), units	1.12 (1.09-1.16)
ACHD subtype, %	
Cardiac Dysfunction (CD)	10
Conduit (CI)	39
Valvular (VI)	51
Beta blocker medication, %	16

Discussion Our single centre, small cohort study, observes broadly similar patterns of $\dot{V}O_2$ change across ACHD subtypes, suggesting that ACHD classification may have limited value in detecting deterioration in AC. This has potential implications for risk stratification and monitoring, although larger studies are needed to confirm these findings and determine whether important subgroup differences may be present.

S94 MEASURING RESPIRATORY VOLUMES WITH A 3D DEPTH CAMERA

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10.1136/thorax-2026-BTSabstracts.100

Background Accurate measurement of airflow and lung volumes during pulmonary function testing is central to respiratory diagnostics. Spirometry is the current gold standard for these measurements; however, it has several limitations, including infection risk, poor tolerability in certain patient groups, and need for specialist operator training. In addition, spirometry provides limited insight into underlying respiratory mechanics. Recent advances in 3D imaging technology suggest that respiratory flow and volume may be estimated from thoracoabdominal shape changes during breathing. If sufficiently accurate, such camera-based systems could offer a non-contact alternative to conventional spirometry

Aim The aim of this research was to evaluate the accuracy of a 3D camera system for the measurement of respiratory volumes during tidal breathing.

Methods HRA provided ethical approval (21/EM/0221), and all participants provided informed consent. Twenty-one participants were recruited to the study (height: 174.5±10.3 cm, weight: 74.1±15.7 kg). Participants were instructed to breathe normally in relaxed standing while unclothed from the waist up (male) or wearing a sports bra (female). A set of 30 green markers were placed on each participant's torso to identify anatomical landmarks. Two Intel L515 depth-cameras captured colour and depth data synchronously with spirometer data. Trials were repeated until a minimum of 10 breaths were recorded.

Custom software was then used to estimate respiratory volumes from 3D coordinate data for each participant over the duration of the trial. This estimate of breathing volume was then divided into breaths to calculate tidal volumes. These tidal volumes were compared with spirometry with a breath-by-breath Bland-Altman analysis and percentage discrepancies.

Results and Discussion 490 breaths were analysed. We found that the camera system underestimated volume by 11 mL with 95% limits of agreement of -188 – 165 mL. This bias translates to a percentage discrepancy of -1.3%. Individual patient discrepancies range from -13.6% to 21.8% (table 1). These results show that the cameras can accurately predict tidal volumes. This is the first step in a camera-based spirometry system. As the accuracy of depth camera technology continue to improve, it is likely that differences between spirometry and 3D measurements volume will reduce.

Abstract S94 Table 1 Participant mean tidal volumes measured by the cameras and the spirometer, difference between them and the percentage discrepancy. All (pooled) is the bias when each individual breath is analysed. The mean of means is the means of all the participants' means

Participant ID	Camera TV (L)	Spirometer TV (L)	Camera – Spirometer (L)	Discrepancy (%)
Spiro2	1.434	1.469	-0.035	-2.4
Spiro3	1.411	1.214	0.197	16.2
Spiro4	1.504	1.598	-0.094	-5.9
Spiro5	0.910	0.923	-0.013	-1.5
Spiro6	1.287	1.144	0.143	12.4
Spiro7	0.637	0.648	-0.011	-1.8
Spiro8	0.732	0.717	0.015	2.1
Spiro9	1.037	1.001	0.036	3.6
Spiro10	0.392	0.440	-0.048	-11.0
Spiro11	0.555	0.643	-0.088	-13.6
Spiro12	1.463	1.489	-0.026	-1.7
Spiro13	0.600	0.617	-0.017	-2.7
Spiro14	1.084	1.141	-0.057	-5.0
Spiro15	0.736	0.699	0.037	5.3
Spiro16	0.366	0.397	-0.031	-7.7
Spiro17	1.236	1.341	-0.105	-7.8
Spiro19	0.825	0.781	0.044	5.6
Spiro20	1.244	1.278	-0.034	-2.7
Spiro21	0.742	0.728	0.014	1.9
Spiro22	0.862	0.707	0.155	21.8
Spiro24	0.574	0.620	-0.046	-7.4
All (pooled)	**0.864**	**0.875**	**-0.011**	**-1.3**
Mean of means	**0.935**	**0.933**	**0.002**	**-0.1**

S95 CARDIOPULMONARY EXERCISE TESTING INSIGHTS INTO PATIENTS REFERRED WITH UNEXPLAINED DYSPNOEA

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Introduction Unexplained dyspnoea remains a common clinical presentation with a broad differential diagnosis. Patients often undergo multiple investigations before a cause is identified. Cardiopulmonary exercise testing (CPET) provides a non-invasive assessment of integrated cardiac, pulmonary and peripheral muscle responses to exercise and may help identify the physiological basis of dyspnoea. Although CPET is established in the assessment of unexplained dyspnoea, there is limited data describing non-invasive CPET referral outcomes and service utilisation. This retrospective cohort study aimed to describe the prevalence of different CPET outcomes in patients with unexplained dyspnoea.

Methods As part of a service evaluation, CPET data was retrospectively extracted from all referrals between March 2021 to 2025 at a Birmingham hospital. Information was accessed via a local CPET database. From the 1160 referrals extracted, 220 (19%) were for investigation of unexplained dyspnoea. CPETs were performed and reported by respiratory physiologists. Data collected included referral indication, referring specialty, demographics and primary CPET outcome. Patients were assigned diagnostic categories based on CPET reports.

Results Of the 220 dyspnoea referrals, 103 (46.8%) were female and 117 (53.2%) were male. Median age was 53 years (21-87) and median BMI was 29.4kg/m2 (17.0-53.8). Smoking frequency was reported as 40 %. Most referrals for unexplained dyspnoea were made from respiratory medicine (87.3%), followed by cardiology (11.4%) and thoracic surgery (1.3%). Primary CPET outcomes are shown in table1. Only 39 patients (17.7%) demonstrated ventilatory limitation and 13 (5.9%) had cardiac limitation or abnormality. In contrast, 46 patients

Abstract S95 Table 1 Primary CPET outcomes from dyspnoea referrals

Category	Frequency (n)	Percentage (%)
Normal	32	14.5%
BPD-related	29	13.2%
Deconditioning	46	20.9%
Obesity-related functional limitation	34	15.5%
Ventilatory limitation	39	17.7%
Hyperventilation	14	6.4%
Cardiac limitation / abnormality	13	5.9%
Upper airway disorders (ILO, EDAC, TBM)	8	3.6%
Pulmonary vascular disease	3	1.4%
Poor test	2	0.9%

BPD=Breathing Pattern Disorder; ILO=Intermittent Laryngeal Obstruction; EDAC= Excessive Dynamic Airway Collapse; TBM= Tracheobronchomalacia

(20.9%) had deconditioning, 34 (15.5%) had obesity-related limitation, and 29 (13.2%) had breathing pattern disorder-related limitation.

Conclusion This study demonstrated that fewer than 1 in 5 patients referred for CPET to investigate dyspnoea demonstrated mechanical ventilatory limitation. Non-ventilatory causes, particularly de-conditioning, obesity related functional limitation and breathing-pattern disorder were common. These findings suggest that CPET can redirect management towards targeted interventions such as pulmonary rehabilitation and weight management and may help reduce unnecessary investigations. Despite this, dyspnoea referrals accounted for only 19% of total CPET activity, with most tests performed for surgical work-up, suggesting that CPET may be underutilised in the diagnosis of unexplained dyspnoea.

S96 ROLE OF EXERCISE IN THE FUNCTIONAL CAPACITY AND CLINICAL OUTCOMES OF SMOKERS

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Background Preserved Ratio Impaired Spirometry (PRISm) is defined as the ratio of forced expiratory volume in 1 second (FEV1) to forced vital capacity (FVC) (FEV1/FVC) ≥ [endif]--> 70%, but FEV1 <80%. It is more common in smokers with increased risk of developing chronic obstructive pulmonary disease (COPD), adverse cardiovascular outcomes, and mortality. There is limited data on the role of exercise in functional capacity and clinical outcomes of smokers with PRISm.

Methods We performed a randomised controlled trial on smokers or ex-smokers with PRISm (ClinicalTrials.gov ID NCT07387432). We randomised them 1:1:1 into exercise group, exercise with telehealth group and no exercise group, with a 26-week intervention period and a 52-week follow-up. In exercise group, walking exercise with step goal was advised. In exercise with telehealth group, we conducted a pilot program in Hong Kong for exercise classes by physiotherapists via telehealth. In no exercise group, no exercise advice was given. All groups were given smoking cessation advice. The change in 6-minute walk test (6MWT) distance and the modified Medical Research Council dyspnoea scale (mMRC) were assessed. Clinical outcomes with hospitalisation were documented. Chi-square test or one-way ANOVA were used to estimate any statistically significant difference between groups.

Results 90 participants were recruited, with 75 males and 15 females, 14 current smokers and 76 ex-smokers. The median age was 66.5+/-12.7 years (table 1).

In the interim analysis at week 12, the mean change in 6MWT distance was 0.7+/-2.1m in exercise group, 3.4+/-29.5m in exercise with telehealth group, and -8.6+/-15.2m in control group (P= 0.618). The mean mMRC in exercise group was 1.3+/-0.9 at baseline and 1.1+/-1.0 at 12 weeks, exercise with telehealth group was 1.2+/-0.9 at baseline and 1.2+/-0.9 at 12 weeks, and control group was 1.4+/-0.9 at baseline and 1.2+/-0.8 at 12 weeks (P=0.895). 5 participants (5.6%) were hospitalised for cardiovascular respiratory events (4 in exercise group,1 in exercise with telehealth group).

Conclusion Exercise with telehealth was feasible in Hong Kong and demonstrated improvement in functional capacity of smokers with PRISm in 12 weeks. Further follow-up will be done.

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More than a barrier: the airway epithelium

S97 LIMITED IL-5 RECEPTOR EXPRESSION IS ASSOCIATED WITH ABSENCE OF DIRECT ANTIVIRAL EFFECTS OF IL-5 IN ASTHMA

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Background Type 2 cytokines are known to impair antiviral immunity in asthma, but the direct role of interleukin-5 (IL-5) in this process remains unclear. IL-5 is the key regulator of eosinophil differentiation and activation, and its receptor (IL-5R) has been reported on various airway and immune cells; however, its functional relevance outside eosinophils has not been established.

Objective To determine whether IL-5 directly modulates antiviral interferon (IFN) responses in airway epithelial cells,

Abstract S96 Table 1 Baseline characteristics of study individuals

	Exercise group (n=30)	Exercise with telehealth group (n=30)	No exercise group (n=30)	*P* values
Gender (Females)	5	4	6	0.787
Age (median ± SD)	65.5 ± 13.3	67.0 ± 13.0	66.0 ± 12.1	0.885
Current smoker	4	3	7	0.333
Ex-smoker	26	27	23	
FEV1 (% pred)	73.5 ± 9.6	74.9 ± 8.0	73.5 ± 9.6	0.552
FEV1/FVC (%)	76 ± 6.6	74 ± 5.3	77 ± 8.2	0.780
6-minute walk test distance (m)	369.2 ± 73.8	378.4 ± 87.3	367 ± 30.4	0.760
mMRC dyspnoea scale	1.3 ± 0.9	1.2 ± 0.9	1.4 ± 0.9	0.662

Abbreviation:FEV1 = Forced expiratory volume in 1 second, FVC = forced vital capacity, mMRC = Modified Medical Research Council

peripheral blood monocytes (PBMCs), plasmacytoid dendritic cells (pDCs) and bronchial alveolar lavage (BAL) cells from people with asthma during rhinovirus (RV-A16) infection and to assess the expression and functionality of IL-5 receptors in these cell types.

Methods Primary bronchial epithelial cells (BECs) differentiated at air–liquid interface (ALI, N=5-6), submerged BECs (N=4-5), PBMCs (N=6), pDCs (N=6) and BAL cells (N=5-7) isolated from people with mild asthma were pretreated with IL-5 (10–100 ng/mL) and infected with RV-A16. Expression of type I and III interferons (IFN-α, IFN-β, IFN-λ), interferon-stimulated gene viperin, and viral RNA load were measured by qPCR and ELISA/MSD. IL-5Rα (IL5RA) and β-chain (CSF2RB) expression were analyzed by qPCR and flow cytometry. The biological activity of IL-5 was validated by flow cytometric assessment of CD69 expression in eosinophils (N=4-5).

Results IL-5 pretreatment did not significantly affect RV-A16–induced IFN-α, IFN-β, or IFN-λ responses, viral replication or IL-5 receptor subunits expression in any cell type tested. qPCR analysis demonstrated low expression of IL5RA and CSF2RB across PBMCs, pDCs, BAL cells, and submerged BECs. Differentiated ALI cultures exhibited the highest baseline IL5RA expression, which was significantly downregulated following RV-A16 infection ($P<0.05$, 0.01), while CSF2RB expression remained highest in PBMCs and BAL cells and low or undetectable in epithelial populations. Flow cytometry did not detect surface IL-5Rα or β-chain on epithelial cells, although intracellular staining indicated cytoplasmic IL-5Rα localization in ALI ($P<0.05$, 0.01). Experiments further demonstrated enhanced CD69 expression on IL-5–treated eosinophils, supporting the biological activity of the cytokine ($P<0.001$).

Conclusions Collectively, these findings indicate that IL-5 lacks direct immunosuppressive effects on epithelial and non-epithelial antiviral responses, likely due to incomplete receptor assembly or signaling capacity.

S98 JAK INHIBITION ATTENUATES RHINOVIRUS-DRIVEN INTERFERON RESPONSES IN COPD AIRWAY EPITHELIUM WITHOUT ENHANCING VIRAL REPLICATION: A SINGLE-CELL TRANSCRIPTOMIC STUDY

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10.1136/thorax-2026-BTSabstracts.104

Background COPD exacerbations are a major cause of morbidity and commonly occur in the setting of respiratory viral infection. The mechanisms driving virus-induced airway inflammation and the therapeutic potential of targeting interferon-(IFN) signalling remain incompletely understood.

Aim To characterise airway epithelial cell responses to human rhinovirus (HRV) using single-cell transcriptomic profiling and determine whether post-infection treatment with baricitinib attenuates inflammatory signalling.

Methods Primary bronchial epithelial cells from patients with moderate-to-severe COPD were differentiated at air–liquid interface for ≥28 days. Cultures were mock-infected or infected apically with HRV16 (MOI 0.01) for 6 hours. Quantification of HRV viral titres by qPCR confirmed successful infection. At 48 h post-infection (hpi), cultures received apical baricitinib (1 μM) daily for 72 h. At 120 hpi, cells were fixed using Evercode Cell Fixation v3 (Parse Biosciences) and processed for single-cell RNA-sequencing.

Results scRNA-seq identified distinct epithelial cell populations, including basal, club, goblet, ciliated cells and ionocytes. HRV infection induced marked upregulation of IFN-stimulated genes, including *OAS1–3* and *IFIT1–3*, with greater transcriptional dysregulation observed at 48 hpi than at 120 hpi, both in the magnitude of differential expression and the number of altered genes. While IFN-related responses were shared across epithelial cell types, HRV induced distinct cell type-specific transcriptional signatures. Ciliated and goblet cells exhibited the greatest degree of dysregulation, whereas club and basal cells demonstrated comparatively fewer transcriptional changes. Post-treatment with baricitinib attenuated expression of mucus-associated genes (*MUC5AC*, *MUC5B*) and suppressed a broad IFN-driven inflammatory programme, including reduced expression of *CXCL11, CXCL10, IFIT1–3, OAS1–3, ISG15, MX1/2, RSAD2 and STAT1/2*. Additional downregulation of innate antiviral sensing and immune response genes, including *IFIH1, EPSTI1* and *CMPK2*, was observed, consistent with inhibition of JAK–STAT signalling. Despite attenuation of IFN-signalling, baricitinib treatment was not associated with increased viral load.

Conclusions HRV infection drives robust, cell type-specific inflammatory and IFN-mediated responses in COPD airway epithelium, with ciliated and goblet cells showing the greatest transcriptional perturbation. Post-infection treatment with baricitinib attenuated inflammatory and interferon-driven pathways, together with mucus-associated gene expression, without evidence of enhanced viral replication. These findings support further investigation of JAK inhibition as a therapeutic strategy for virus-induced COPD exacerbations.

S99 ORMDL3 REGULATES NON-TYPEABLE HAEMOPHILUS INFLUENZAE INFECTION IN AIRWAY EPITHELIAL CELLS

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10.1136/thorax-2026-BTSabstracts.105

Background Polymorphisms of *ORMDL3* on human chromosome 17q21 confer the major genetic susceptibility to asthma. Recent research found ORMDL3 plays important roles in haemoglobin metabolism, ferroptosis and necroptosis. In this research, we infected *ORMDL3* knockout and *ORMDL3* overexpression human epithelial cells with *Haemophilus influenzae* (NTHi) to investigate ORMDL3's roles in NTHi diseases.

Methods *ORMDL3* knockouts in human airway epithelial HBEC3-KT cells were created by CRISPR/Cas9 techniques. *ORMDL3* overexpression cells were established in HBEC3-KT cells. Wild-type cells, knockout cells, control cells, and overexpression cells were infected with NTHi (from ATCC) for 6 and 16 hours. The copy numbers of NTHi in epithelial cells were measured by quantifying the core gene *hpd3* with qPCR. Colony-forming units (CFU) of NTHi in cells were also counted. The transcript levels of human ferroptosis and necroptosis markers *ICAM-1, HMOX1, GSDMB, MLKL and RIP3* in cells were quantified by qPCR. The cytokine levels of IL-6 and IL-8 from supernatants were examined with ELISA.

Results The bacterial copy levels of *hpd3* in cells were lower at 6 hours and 16 hours in *ORMDL3* knockout cells ($P=0.08$, $P=0.01$, respectively); the copy levels were higher at 6 hours in *ORMDL3* overexpression cells ($P=0.01$). The results were confirmed by CFU counting. In wild-type cells, the transcript levels of *ICAM-1*, *GSDMB*, *HMOX1, MLKL and RIP3* were all increased after NTHi infections at 6 hours (fold change 24.4, 2.4, 3.1,2.3 and 9.9, respectively) and 16 hours (fold change 34.2, 9.8, 9, 3.3 and 55.7, respectively). *ICAM-1, HMOX1 and MLKL* levels decreased in knockout cells and increased in overexpression cells at 6 hours, but all decreased at 16 hours except for *MLKL*. *GSDMB* levels increased in knockout cells and overexpression cells at 6 hours but decreased at 16 hours. The IL-6 and IL-8 levels decreased in supernatants of knockout cells at 6 hours after NTHi infection.

Conclusions ORMDL3 influences NTHi infections in airway epithelial cells. ORMDL3 regulates the expression levels of ICAM-1, GSDMB, HMOX1, MLKL, and RIP3, which play roles in ferroptosis and necroptosis during NTHi infection. Understanding ORMDL3's role in NTHi infection will yield new insights into the mechanisms underlying asthma exacerbations.

S100 ORMDL3 REGULATES HMOX1 EXPRESSION AND LIPID PEROXIDATION FOR FERROPTOSIS IN HUMAN AIRWAY EPITHELIAL CELLS

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10.1136/thorax-2026-BTSabstracts.106

Background Single-nucleotide polymorphisms of *ORMDL3* on human chromosome 17q21 show a strong association with childhood asthma. It is a transmembrane protein localised to the endoplasmic reticulum, where it regulates sphingolipid synthesis, endoplasmic reticulum stress, and the unfolded protein response. In a previous study, we found that ORMDL3 modulates ferroptosis markers HMOX1 and SLC7A11. Since HMOX1 plays an important role in iron metabolism and lipid peroxidation. In this research, we further investigate the mechanism of ORMDL3 in ferroptosis, including the effect on lipid peroxidation and iron accumulation.

Methods *ORMDL3* knockout cells were created with CRISPR/Cas9 in the bronchial epithelium cell line (HEBC3-KT) from previous work. *ORMDL3* overexpression cells were established with expression plasmids. Cells were stimulated with 10 μm Erastin for 48 hours to induce ferroptosis. Liproxstatin-1 was used as an inhibitor to protect cells from ferroptosis. The ferroptosis markers GPX4, SLC7A11 and HMOX1 were analysed by immunofluorescence and western blot. Lipid peroxidation and iron assays were detected by flow cytometry.

Results In *ORMDL3* knockout cells, the expression levels of ferroptosis markers (*SLC7A11* and *GPX4*) were significantly increased, whereas there were no differences in expression levels in *ORMDL3* overexpression cells. Erastin induction induced the expression of *SLC7A11*, *GPX4*, and *HMOX1 in all experimental cell groups*. However, HMOX1 expression levels were lower in *ORMDL3* knockout cells *and higher in ORMDL3* overexpression cells after Erastin stimulation. *ORMDL3* knockout reduced lipid peroxidation in *ORMDL3* knockout cells after Erastin stimulation. The ongoing work included examining lipid peroxidation in ORMDL3-overexpressing cells and conducting iron assays across cell types.

Conclusion Our findings suggest that ORMDL3 contributes to ferroptosis-related responses in human airway epithelial cells by regulating HMOX1 expression and lipid peroxidation. These findings provide new insights into the molecular mechanisms underlying airway epithelial injury and asthma pathogenesis.

S101 EPITHELIAL-FIBROBLAST CROSSTALK INDUCES TRANSCRIPTIONAL DIFFERENCES IN SMOOTHENED-HEDGEHOG SIGNALLING IN COPD-DERIVED AIRWAY EPITHELIAL CELLS

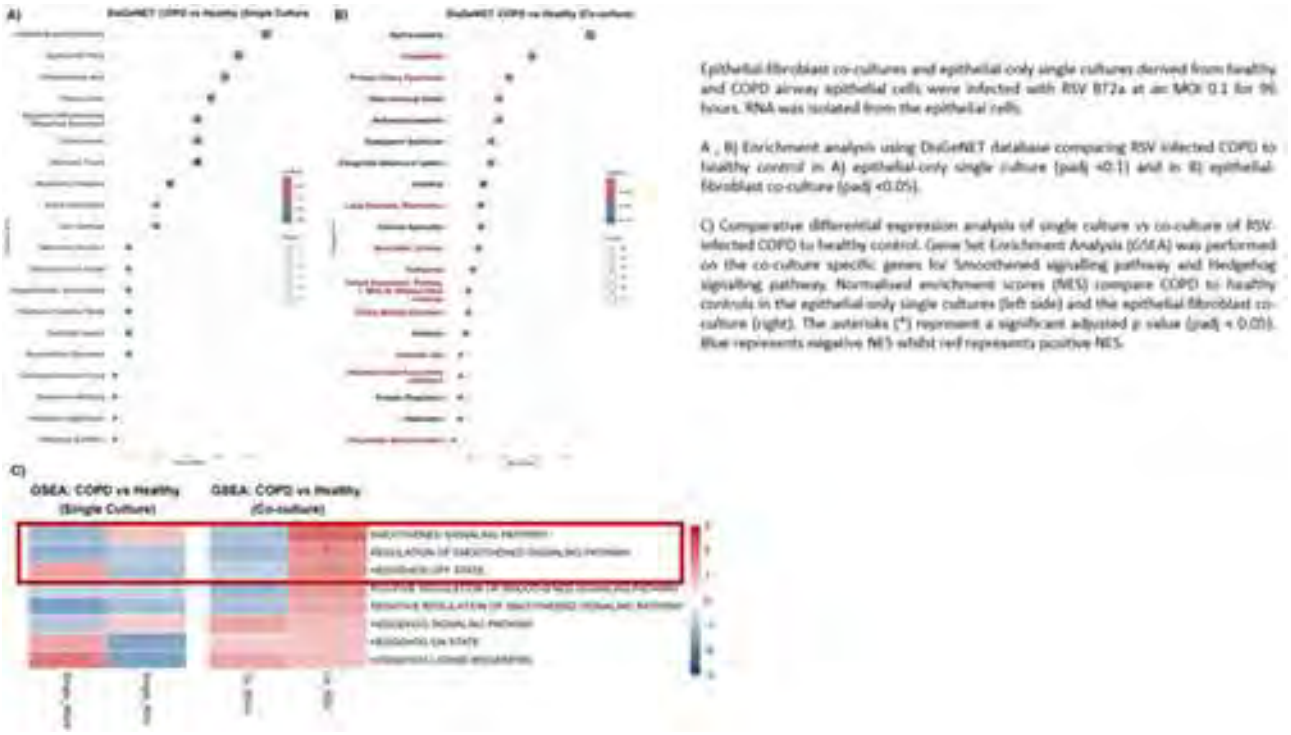
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Respiratory Syncytial Virus (RSV) is the leading cause of lower respiratory tract infections in children under the age of 5, and an important pathogen in the exacerbation of chronic lung diseases (CLDs) in adults. Chronic obstructive pulmonary disorder (COPD) is the third leading cause of death worldwide, and RSV plays a crucial role in exacerbation cases, contributing to the high morbidity and mortality. The burden of RSV in adults is similar to seasonal influenza, further highlighting the importance of this respiratory virus in the adult population. However, despite this, the mechanisms behind the progression of CLDs and respiratory virus pathogenesis as well as virus-induced exacerbations of lung disease are poorly understood.

Cellular crosstalk is known to be an important factor in lung homeostasis and tissue repair, and dysregulation has been reported in CLDs. In this study, we aimed to determine the role of epithelial-fibroblast crosstalk in RSV-induced lung pathogenesis comparing COPD and healthy derived airway epithelial cells. Disease enrichment analysis using the DisGeNET database revealed an 'exacerbation' phenotype only seen in the co-culture model, defined by enriched terms for respiratory diseases and ciliopathies (figure 1A & B).

Comparative differential gene expression analysis established the culture effect between single culture vs co-culture comparing RSV-infected COPD to healthy. This revealed a marked increase in the number of co-culture specific genes in comparison to the single culture and the conserved genes in COPD-derived cells, demonstrating a transcriptional signature driven by fibroblasts. Gene Ontology (GO) analysis also revealed that the co-culture specific genes were positively enriched for terms related to ciliary function and Smoothened signalling, with several overlapping genes. Smoothened is the signalling transduction protein for the Hedgehog signalling pathway. Gene Set Enrichment Analysis (GSEA) revealed that despite significant positive enrichment for Smoothened signalling in the co-culture model (NES=1.99; padj <0.05), there was also significant positive enrichment for Hedgehog OFF state (NES=1.54; padj <0.05) (figure 1C). This suggests a potential partial or incom-



Epithelial-fibroblast co-cultures and epithelial-only single cultures derived from healthy and COPD airway epithelial cells were infected with RSV B72a at an MOI 0.1 for 96 hours. RNA was isolated from the epithelial cells.

A , B) Enrichment analysis using DisGeNET database comparing RSV infected COPD to healthy control in A) epithelial-only single culture (padj <0.1) and in B) epithelial-fibroblast co-culture (padj <0.05).

C) Comparative differential expression analysis of single culture vs co-culture of RSV-infected COPD to healthy control. Gene Set Enrichment Analysis (GSEA) was performed on the co-culture specific genes for Smoothened signalling pathway and Hedgehog signalling pathway. Normalised enrichment scores (NES) compare COPD to healthy controls in the epithelial-only single cultures (left side) and the epithelial-fibroblast co-culture (right). The asterisks (*) represent a significant adjusted p value (padj < 0.05). Blue represents negative NES whilst red represents positive NES.

Abstract S101 Figure 1 Epithelial-fibroblast co-cultures alter the transcriptional regulation in COPD- derived airway epithelial cells

plete activation of Smoothened signalling or alternative noncanonical Hedgehog signalling.

Overall, the significant overlap between the ciliary terms and Smoothened signalling pathway suggests a potential ciliogenesis dysfunction through Smoothened-Hedgehog transduction mediated by epithelial-fibroblast crosstalk.

S102 A MULTICELLULAR HUMAN COPD BRONCHIAL ORGANOID MODEL RECAPITULATES EPITHELIAL DYSFUNCTION, ENDOTHELIAL DISORGANISATION AND CELLULAR SENESCENCE

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10.1136/thorax-2026-BTSabstracts.108

Introduction Chronic obstructive pulmonary disease (COPD) is characterised by dysregulated inflammation, accelerated lung ageing and cellular senescence. Existing *in vitro* and animal models do not adequately recapitulate the complex multicellular microenvironment that drives disease pathogenesis. We developed a multicellular human bronchial organoid model to investigate structural and functional features of COPD-associated senescence.

Methods Bronchial organoids were generated by self-aggregation of primary bronchial epithelial cells obtained from healthy donors and patients with COPD together with human pulmonary microvascular endothelial cells and human bronchial fibroblasts in ultra-low attachment culture plates. Organoids were differentiated for 21 days and characterised using immunofluorescence microscopy, senescence-associated β-galactosidase (SA-β-gal) staining and analysis of senescence-associated secretory phenotype (SASP) mediators.

Results Healthy and COPD-derived organoids consistently formed reproducible three-dimensional bronchial-like structures and underwent epithelial differentiation, demonstrating expression of airway lineage markers including β-tubulin-positive ciliated cells, MUC5AC-positive goblet cells and CC10-positive club cells. Compared with healthy organoids, COPD cultures exhibited marked structural abnormalities characterised by disrupted ciliary architecture and shorter cilia. In addition, CD31 staining demonstrated altered endothelial organisation within COPD organoids, whereas healthy cultures displayed a more organised endothelial network. COPD organoids were smaller in size and exhibited significantly increased cellular senescence, demonstrated by enhanced SA-β-gal activity. Analysis of secreted inflammatory mediators revealed elevated levels of key SASP cytokines, including IL-6 and IL-8, compared with healthy controls. Collectively, these findings demonstrate that COPD organoids reproduce multiple hallmarks of COPD pathobiology, including epithelial dysfunction, endothelial disorganisation, cellular senescence and chronic inflammatory signalling.

Conclusions We have established a multicellular human COPD organoid model that recapitulates key structural and functional features of COPD. This platform provides a physiologically relevant system for investigating mechanisms of cellular senescence and identifying novel therapeutic strategies targeting age-related pathways in COPD.

Lung infection: that's just (a)typical

S103 RADIOLOGICAL FINDINGS, INTER-OBSERVER AGREEMENT AND IMAGING BURDEN IN PATIENTS WITH POSSIBLE NON-VENTILATOR ASSOCIATED HOSPITAL ACQUIRED PNEUMONIA RANDOMISED TO CXR OR CT

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10.1136/thorax-2026-BTSabstracts.109

Introduction Chest x-rays(CXR) are central to diagnosing non-ventilator hospital-acquired pneumonia(nv-HAP), but may lead to over-diagnosis of pneumonia compared with CT. HAP-FAST was a pilot SMART trial evaluating diagnostic pathways for suspected nv-HAP.[1] This analysis estimated interobserver agreement for radiological pneumonia, non-pneumonia findings on initial imaging, and subsequent imaging burden over 90 days - to inform the implementation of a definitive trial.

Methods Patients with suspected nv-HAP were recruited at four UK hospitals and randomised to initial CXR or low-dose CT within routine care, with 90-day follow-up. Initial images were reported by local radiologists; findings were entered into a trial case report form(CRF) by site investigators. After recruitment, images were anonymised, transferred to a single site and independently reviewed by general radiologists blinded to the original report. Reviewers were informed that nv-HAP was clinically suspected, and their reports were used to complete a second CRF. Radiology findings and subsequent imaging were summarised descriptively and agreement on pneumonia between clinical and independent reports was estimated.

Results Of 220 randomised participants, 188 had initial radiology reports available; median age was 77.5 years (IQR 71–84) in the CT arm and 76.5 years (IQR 67–84) in the CXR arm. During 90-day follow-up, 27/188 (14.4%) underwent additional imaging: 19/112 (17.0%) after initial CXR and 8/76 (10.5%) after initial CT. CT reports recorded more additional findings, most commonly emphysema(20%), pulmonary nodules(20%), bronchiectasis(17%) and atelectasis(14%). Additional findings on CXR were less frequent: emphysema(9%), pleural effusion(6%), atelectasis(5%) and heart failure(4%). Paired clinical and independent reports were available for 159 participants; interobserver agreement for pneumonia is shown in the table 1.

Conclusions In suspected nv-HAP, CT generated more non-pneumonia information than CXR and was associated with fewer subsequent imaging tests over 90 days. Interobserver agreement for pneumonia was higher for CT than CXR. These data suggest a definitive trial could effectively compare CXR to CT but extending follow-up beyond 90 days will be important to capture the full impact of CT on subsequent imaging.

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S104 EXPLORING COSTS ASSOCIATED WITH SUSPECTED NON-VENTILATOR-ASSOCIATED HOSPITAL-ACQUIRED PNEUMONIA (NV-HAP) WITHIN THE HAP-FAST TRIAL

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10.1136/thorax-2026-BTSabstracts.110

Background Diagnosis of nv-HAP is unreliable with no randomised trials of diagnostic pathways. HAP-FAST was a pilot sequential multiple-assignment randomised trial comparing chest x-ray (CXR) with low-dose CT and empirical antibiotics

Abstract S103 Table 1 Inter-observer agreement on pneumonia reporting between CXR and low-dose CT

Index test	Paired reports	Reader 1 pneumonia positive	Reader 2 pneumonia positive	Agreement counts*	Observed agreement	Cohen's κ†
	n	n (%)	n (%)	YY / YN / NY / NN	% (95% CIs)	
Overall	159	78 (49·1)	83 (52·2)	70 / 8 / 13 / 68	86·8 (81·1 - 91·6)	0·74 (0·62 - 0·84)
CXR	97	41 (42·3)	44 (45·4)	34 / 7 / 10 / 46	82·5 (75·3 - 89·7)	0·64 (0·49 - 0·79)
CT	62	37 (59·7)	39 (62·9)	36 / 1 / 3 / 22	93·5 (87·1 - 98·4)	0·86 (0·72 - 0·97)

* Agreement counts where Y = pneumonia recorded as present and N = pneumonia recorded as absent by the first and second verification readers respectively. †Cohen's Kappa is a statistical measure of inter-observer agreement between two raters. 1.0 implies perfect agreement, 0 implies no better than chance and values <0 are worse than chance. >0.60 is the generally accepted threshold for a diagnostic test. >0.81 is regarded as a reference standard test value.

Abstract S104 Table 1 Inpatient and post-discharge costs to 90 days

A. Index admission inpatient costs

Comparison	Group	n	Age, years mean (SD)	Female n/N (%)	Total inpatient costs £: Mean (SD); Median (IQR)	Base inpatient costs £: Mean (SD); Median (IQR)	Inpatient excess bed-day cost £ Mean (SD); Median (IQR)
Stage 1 CT vs CXR	CT	44	74.0 (12.8)	25/44 (56.8)	9187 (10334) 6,196 (4248–9886)	6832 (3632) 6196 (4248–8251)	2355 (8,764) 0 (0–0)
	CXR	57	75.3 (14.0)	29/57 (50.9)	9806 (11357) 6196 (3419–10119)	6960 (5816) 6040 (3406–7817)	2845 (9405) 0 (0–0)
Stage 2 FAPP vs standard	FAPP	21	69.6 (14.8)	15/21 (71.4)	7261 (4696) 5268 (4248–7592)	6879 (4467) 5268 (4248–7592)	382 (1205) 0 (0–0)
	Standard	18	73.8 (10.2)	7/18 (38.9)	9127 (10979) 6196 (3804–7471)	5688 (2502) 6173 (3586–6307)	3439 (9561) 0 (0–0)

B. Post-discharge NHS/PSS cost drivers among questionnaire responders

Cost component	Item completion, n/55 (%)	Any use, n/55 (%)	Cost, £ Mean (SD); Median (IQR)	Share of aggregate total, %
Readmission	43/55 (78.2)	17/55 (30.9)	2342 (4718) 0 (0–2781)	80.0
Community services	55/55 (100.0)	13/55 (23.6)	260 (596) 0 (0–0)	11.4
Outpatient clinics	53/55 (96.4)	22/55 (40.0)	91 (120) 0 (0–192)	3.8
Investigations	53/55 (96.4)	19/55 (34.5)	70 (170) 0 (0–28)	3.0
GP appointments	52/55 (94.5)	29/55 (52.7)	38 (49) 37 (0–46)	1.6
New medications	49/55 (89.1)	14/55 (25.5)	8 (21) 0 (0–3)	0.3

with BIOFIRE® FILMARRAY® pneumonia panel (FAPP)-guided antibiotics in adults with suspected nv-HAP. We embedded a costing sub-study to identify cost drivers to inform the health economic methods for a definitive trial.

Methods HAP-FAST was funded by the National Institute for Health and Care Research (300669) and approved by NHS Research Ethics (22/WA/0315). Costs were assessed from an NHS and personal social services perspective over the index admission, then from discharge to day 90. In-patient costs from two of four sites (101/220 recruits) were derived from HRG-based spell (admission) costing and separated into base costs (the standard spell tariff) and excess bed-day costs (additional per-day charges beyond the expected length of stay for the HRG). Post-discharge resource use among questionnaire responders was micro-costed from a bespoke day-90 survey. Share of aggregate post-discharge cost was calculated as the sum of each component divided by the sum of available-case post-discharge totals.

Results Panel A shows inpatient costs were similar between participants randomised to CXR and CT. Excess bed-day costs were skewed: most participants had no excess bed-day cost, while a minority with prolonged stays generated high costs, giving median values of £0 despite non-zero means. In the subgroup eligible for stage-2 randomisation, costs were lower in the FAPP arm, because excess bed-day costs were lower. Panel B shows post-discharge costs in 55 questionnaire responders across both study arms. Readmissions were the dominant post-discharge cost driver, with community service use the next largest contributor.

Conclusions In suspected nv-HAP, NHS and personal social services costs were driven by inpatient and readmission bed-days and were sensitive to variation in these components, with community service use also contributing to post-discharge costs. A definitive trial should prioritise robust ascertainment of bed-days and pneumonia associated HRG modification through routine data linkage and improve measurement of community service use; other post-discharge items could be simplified.

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S105 SAFETY OF V116, A 21-VALENT ADULT-SPECIFIC PNEUMOCOCCAL CONJUGATE VACCINE, IN ADULTS ≥18 YEARS: A POOLED SECONDARY ANALYSIS FROM THE PHASE 3 PROGRAM

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Background V116 is an adult-specific, 21-valent pneumococcal conjugate vaccine (PCV) for prevention of invasive pneumococcal disease (IPD) and pneumonia. It includes serotypes responsible for the majority of adult IPD in countries with established pediatric vaccination programs. Safety of V116 in the phase 3 program involving healthy adults and adults with medical conditions that increase risk of pneumococcal disease was evaluated in the phase 3 program.

Methods We evaluated the proportions of participants with solicited injection-site and systemic adverse events (AEs) through day 5 post-vaccination, and vaccine-related serious AEs through month 6. Data for this secondary analysis included healthy adults (Group 1, n=3,220) and adults with one or more risk-conferring medical conditions (Group 2, n=2,080).
Results Overall, 2,044 (63.5%) participants in Group 1 and 1,182 (56.8%) in Group 2 reported at least one solicited AE. The proportions of participants with solicited AEs were comparable between groups, with the exception of injection-site pain, which was numerically higher in participants from Group 1 (1,812; 56.3%) than in Group 2 (978; 47.0%). Most solicited AEs were mild to moderate in intensity and resolved within three days. Grade 4 events were rare in both groups. One participant in each group experienced a serious vaccine-related AE (injection-site cellulitis in Group 1; bronchospasm in Group 2). There were 7 total deaths, and none were considered vaccine related.
Conclusions Across a large phase 3 program, V116 was well tolerated in adults with and without conditions that increase pneumococcal disease risk. Results support the use of V116 in adults.

S106 THE BRITISH THORACIC SOCIETY MULTIDRUG RESISTANT-TUBERCULOSIS CLINICAL ADVICE SERVICE: ACTIVITY SUMMARY 2018-2025

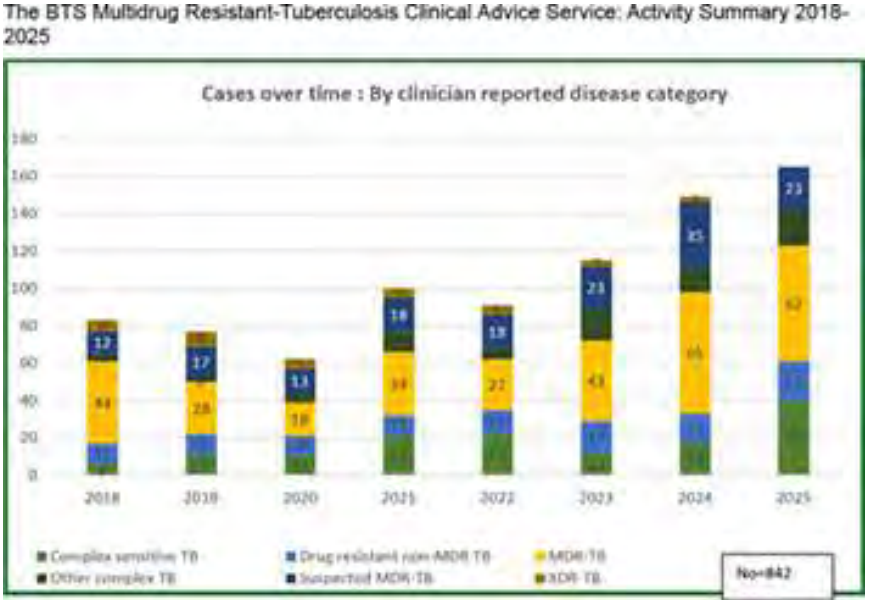
[1]P Haldar, [2]P Papineni, [3]S Howard, [4]M Dedicoat, [5]OM Kon, [6]G Bothamley, [7]T Capstick, [8]S Coles, [9]S Hindle, [10]M Lipman, [8]A Mackin, [11]F Perrin, [12]E Robinson, [13]M Shadab. *[1]Glenfield Hospital, Leicester, UK; [2]Ealing Hospital, London, UK; [3]British Thoracic Society, London, UK; [4]Birmingham Heartlands Hospital, Birmingham, UK; [5]St Mary's Hospital, London, UK; [6]Homerton University Hospital, London, UK; [7]St James' Hospital, Leeds, UK; [8]UKHSA, London, UK; [9]NHSE, London, UK; [10]Royal Free Hospital, London, UK; [11]Kings College Hospital, London, UK; [12]NMRL, London, UK; [13]N/A Patient Representative, London, UK*

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Introduction The BTS Multidrug Resistant-Tuberculosis Clinical Advice Service (CAS) provides an on-line forum and monthly virtual multi-disciplinary meeting of experienced TB and public health physicians, paediatricians, pharmacists, nurses and representatives from the English and Scottish Mycobacterial Reference Laboratories. The team provides consensus advice for drug-resistant or complex cases of TB and ratifies use of novel therapies. Here we report the patients reviewed between January 2018-December 2025.
Methods Data were extracted from a template capturing anonymised information on patients and disease type; and categorised as: **A**) Extensively Drug Resistant-TB (XDR-TB), Multidrug Resistant-TB (MDR-TB), Suspected MDR-TB; **B**) Drug resistant non-MDR-TB, Complex sensitive TB, Other complex TB.
Results 778 patients (465 category A, 313 category B) from 112 UK institutions were discussed.

Category A comprised 33 (7%) XDR-TB; 280 (60.2%) MDR-TB and 152 (32.7%) Suspected MDR-TB. **Category B** comprised 32.9% Drug resistant non-MDR TB, 42.8% Complex sensitive TB and 24.2% Other complex TB. Over time, the number in each Category increased (figure 1).

Over two-thirds of both groups had pulmonary TB (72% Category A and 68% Category B). Patients in Category B were older (mean age 42 years vs 35 years) with a higher UK-born proportion (34% vs 13%). While origin from Bangladesh, India and Pakistan, and Africa predominated across both categories, a higher proportion in Category A were from Eastern Europe (12.7% vs 7.6%). Clinical risk factors associated with TB were recorded in 54% of Category A and 72% of Category B patients. Immunosuppression was a notably more prevalent risk factor in Category B (37% vs 15.5%), and attributable to biological therapies in 36%. Alcohol misuse was the highest sited social risk factor in both categories at 41.5% and 55.3% for A & B respectively followed by homelessness (30.2%, 34.2%). Supervised treatment (DOT or VOT) was implemented in a greater proportion of Category A patients (60% vs 32%).
Conclusions The BTS MDR-TB CAS has rapidly evolved to provide national expert advice for complex TB and a platform for education. It is an exemplar of digital health innovation to deliver equity of healthcare excellence that can be applied to other complex chronic diseases.



Abstract S106 Figure 1 Total of cases referred in categories A & B including those where no advice request submitted

S107 A RANDOMISED TRIAL EVALUATING ADHERENCE AND SAFETY OF RIFAPENTINE-BASED TB PREVENTIVE TREATMENT

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Background Short rifapentine-based tuberculosis preventive treatment (TPT) regimens may improve treatment completion compared with 3 months of daily isoniazid plus rifampicin (3HR), but could pose different adherence demands in routine care. Digital adherence technologies may help support TPT delivery.

Methods An open-label, multi-centre, four-arm RCT in England evaluating adherence and safety of TPT strategies combining regimen choice (short-course rifapentine-based or 3HR) with routine or additional adherence support. Adults 16-65 years eligible for TPT were randomised 2:1:2:2 to: 3HR with routine adherence support (RAS, Arm 1), 3HR with additional adherence support (AAS, Arm 2), weekly isoniazid plus rifapentine for 3 months (3HP) with AAS (Arm 3), or daily isoniazid plus rifapentine for 1 month (1HP) with RAS (Arm 4). AAS was pre-specified for 3HR and 3HP and included Wisepill electronic pill box reminders with supportive text messages. The primary outcome was adequate treatment, defined as taking ≥90% of allocated doses within the pre-specified time-frame, measured using Wisepill box-opening data. Risk ratios (RRs) were estimated using modified Poisson regression, with imputation for missing data.

Results Among 400 participants, 48% were female, 50% recent migrants from high TB settings, and median age 33 years (IQR 28-39). In co-primary analyses, 43.5% (95% confidence interval [CI], 34.2%-52.9%) of participants in Arm 1 achieved adequate treatment, compared with 66.7% (95%CI 57.9%-75.4%) in Arm 3 (risk ratio[RR] 1.55, 95%CI 1.20-1.99;P<0.001) and 56.8% (47.3%-66.4%) in Arm 4 (RR 1.31, 0.99-1.73;P=0.055). There was no evidence AAS improved adherence for 3HR (RR 1.17, 0.83-1.65). Treatment adherence based on pill counts was similar between arms. Drug-related discontinuations and grade ≥3 adverse events were low and similar across arms.

Conclusions Short rifapentine-based treatment strategies, especially 3HP with additional adherence support, achieved higher adequate treatment rates than standard 3HR with routine support. These findings support use of short-course rifapentine-based TPT in high-income settings.

S108 MICROBIOLOGIC OUTCOMES AND RECURRENCE IN PATIENTS WITH NEWLY DIAGNOSED MYCOBACTERIUM AVIUM COMPLEX LUNG DISEASE (MACLD) IN THE PHASE 3B ENCORE TRIAL

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Introduction and Objectives Over 40% of patients with MACLD who receive multidrug therapy (MDT) do not achieve

Abstract S107 Table 1 RID-TB treat – analyses of the primary outcome and key secondary outcomes

	Arm 1 3HR, routine adherence support	Arm 2 3HR, additional adherence support	Arm 3 3HP, additional adherence support	Arm 4 1HP, routine adherence support	Risk Ratio (95% CI) comparing Arm 3 vs Arm 1	Risk Ratio (95% CI) comparing Arm 4 vs Arm 1
Number of randomised participants	116	59	113	112		
Primary outcome						
Taking ≥90% of allocated doses based on Wisepill device Estimated proportion of participants (95% CI)*	43.5% (34.2 – 52.9%)	50.7% (37.4 – 64.0%)$	66.7% (57.9 – 75.4%)	56.8% (47.3 – 66.4%)	1.55 (1.20 – 1.99); P<0.001	1.31 (0.99 – 1.73); P=0.055
Key secondary outcomes						
Taking ≥90% of allocated doses based on pill counts Estimated proportion of participants (95% CI)*	81.8% (74.4 – 89.2%)	90.1% (82.0 – 98.3%)	82.2% (74.5 – 89.8%)	90.0% (83.4 – 96.5%)	1.00 (0.88 – 1.14); P=0.989	1.10 (0.98 – 1.22); P=0.102
Permanently stopping study treatment due to drug-related adverse event(s) Number (%) of participants+	4 (3.6%)	2 (3.6 %)	1 (0.9%)	4 (3.7%)	P= 0.370	P=1.000
Experiencing at least one adverse event(s) of grade 3 or above Number (%) of participants+	1 (1.9%)	0 (0%)	3 (5.6%)	3 (6.0%)	P=0.618	P=0.353

* Analyses based on modified intention-to-treat population, accounting for missing data. The co-primary analyses compared Arm 3 vs 1 and Arm 4 vs 1, respectively.
\+ Analyses based on participants who were prescribed study treatment (n=381).
$ The risk ratio comparing Arm 2 vs 1 was 1.17 (95% CI 0.83-1.65), P=0.362.

microbiologic success; recurrence is common. ENCORE (NCT04677569) was a phase 3b, randomized, placebo-controlled, multinational, active comparator, superiority trial, which evaluated the efficacy and safety of 12 months of amikacin liposome inhalation suspension (ALIS) with MDT in adults newly diagnosed with MACLD who have not initiated antibiotics; patients were then followed for an additional 3 months off treatment. In addition to clinical benefit and microbiological conversion, relapse and new infection rates were evaluated to further characterize treatment efficacy.

Methods Eligible patients were randomized 1:1 to receive once daily MDT (azithromycin 250 mg and ethambutol 15 mg/kg) with either ALIS 590 mg or comparator (empty liposome control) for 12 months. Recurrence analysis (relapse versus new infection) was conducted using whole genome sequencing and data were reported with descriptive statistics.

Results The ENCORE study met the primary endpoint of improvement in respiratory symptoms in the ALIS arm versus comparator arm at month 13 ($P=0.0299$). Significantly higher rates of culture conversion by months 6, 12, and 13, and durable culture conversion at month 15, were observed in the ALIS arm versus comparator arm ($P<0.0001$; Daley 2026). This analysis describes rates of relapse (same MAC species and genotype) or new MAC infection (different MAC species/genotype) among patients with culture conversion. Recurrence was more frequent in the comparator arm versus the ALIS arm, with overall fewer relapses than new infections. No new safety signals were observed.

Conclusions The ENCORE study demonstrated superior respiratory symptom improvement and microbiological efficacy, along with lower recurrence rates in the ALIS arm versus comparator arm in patients with newly diagnosed MACLD who have not initiated antibiotics.

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Decoding lung disease through 'omics

S109 SINGLE-CELL AND SPATIAL DISSECTION OF AIRWAY NICHES REVEAL DISTINCT IMMUNE AND EPITHELIAL DRIVERS OF ASTHMA PHENOTYPES

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Asthma is a chronic and heterogeneous inflammatory disease of the airways, yet its cellular determinants remain incompletely defined, limiting precision therapeutic strategies. To address this knowledge gap, we performed paired upper and lower airway sampling in 57 deeply phenotyped individuals with severe asthma and 21 healthy controls, generating single-cell transcriptomic profiles that resolved 59 distinct cell states across epithelial and immune compartments, encompassing 300,000 cells in total. We demonstrated with ML approaches that upper and lower airway are transcriptomically different, and that the lower airway, in particular the epithelium, plays a major role in discriminating between clinical asthma phenotypes. Integrating unbiased clinical metrics, including eosinophil counts, fractional exhaled nitric oxide (FeNO) and response to inhaled corticosteroids, with cell type prioritisation approaches, we identified clinical phenotype-specific driver cell populations. Overall, asthma tissue exhibited features of a hypoxic environment and a multicellular steroid-response signature. Across phenotypes, the lower airway showed epithelial-mesenchymal transition (EMT) features with ionocytes and basal cells being a common source of the signal. Neutrophil involvement was also pervasive but displayed functional variation by phenotype. Eosinophilic asthma was associated with increased pro-fibrotic gene expression and reduced involvement of the T-cell compartment. Conversely, FeNO non-suppressors showed enhanced endothelial activation coupled with adaptive immune cell recruitment. Allergic



Abstract S109 Figure 1

asthma was typified by type 2 inflammation, with CD4$^+$ Th2 cells and granulocytes showing a differential cytokine contribution, differential allergen binding by B-cell subsets and a shift towards long-lived plasma cells. Spatial transcriptomics across 23 individuals further identified a submucosal CD4$^+$ T-plasma cell niche enriched in allergic asthma compared to health. Together, we provide an atlas of the asthmatic airway, delineating distinct epithelial, stromal and immune mediators of disease heterogeneity with therapeutic implications.

S110 GERMLINE AND SOMATIC GENETIC SEQUENCING REVEAL CLONAL HAEMATOPOIESIS AND RARE, HIGH-IMPACT VARIANTS IN IL-17RA IN LATE-ONSET SEVERE EOSINOPHILIC ASTHMA

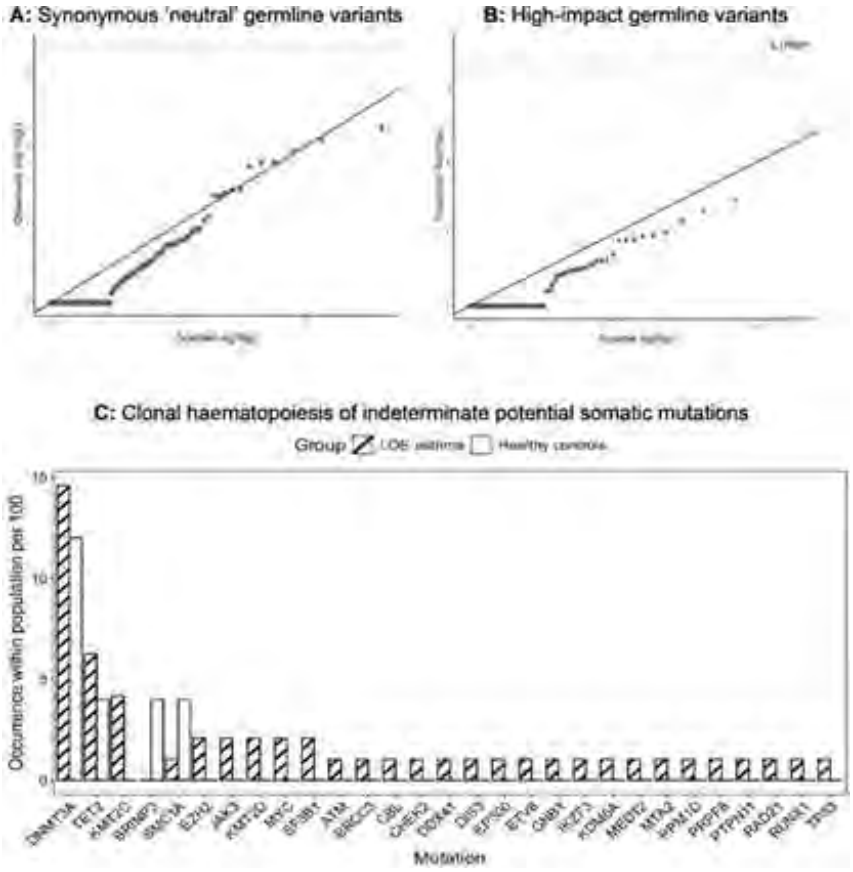
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Introduction Non-atopic, late-onset severe eosinophilic (LOE) asthma constitutes a significant proportion of severe eosinophilic asthma. Despite strong evidence suggesting a heritable component the genetic risk factors remain unknown. In clonal haematopoiesis of indeterminate potential (CHIP) a blood stem cell clone acquires a somatic mutation providing a growth advantage, causing that clone to expand, without meeting criteria for cancer. We hypothesised CHIP might promote systemic eosinophilia in LOE asthma.

Methods Germline: 96 closely phenotyped patients with non-atopic LOE asthma with minimal smoking and no history of malignancy were sequenced using a custom designed exon panel of 102 genes associated with T2 inflammation. Matched control data from gnomAD exomes 4.1 were used to assess the gene burden of rare, high-impact variants. Somatic: The LOE patients and 25 matched healthy controls were sequenced using a curated panel of 108 CHIP genes and variant calling performed using Mutect2 and Vardict (https://github/hinks-lab/).

Results Germline: Mean sequencing quality by depth was 14.2±8.5 for LOE asthma patients and 12.1±3.9 for gnomAD controls. There was a greater burden of rare, high, impact mutations in IL-17RA for LOE patients than in the gnomAD control population (OR = 7.2, 95%CI: 3.2–16.2; p<0.0003, figure 1B). Somatic: Mean coverage was 987± 416 reads/bp.



Abstract S110 Figure 1 A&B: QQ plots comparing observed and expected frequencies in gene burden of mutations in non-atopic late-onset eosinophilic (LOE) asthmatic patients [observed] and a large, ethnically matched control general population (gnomAD 4.1 exomes) [expected]. Each dot represents a gene. The horizontal dotted line marks the Bonferroni corrected a threshold. A: Comparison of the burden of synonymous ('neutral') variants shows well calibrated data and conservative testing parameters. B: A significantly greater burden of rare, high impact variants is found in IL-17RA for the LOE asthma group (red dot), OR=7.2, 95%CI: 3.2-16.2; p<0.0003. C: Occurrence of clonal haematopoiesis of indeterminate potential (CHIP) driver mutations with a variant allele frequency (vAF) of ≥1% in non- atopic late-onset eosinophilic (LOE) asthma patients and age and smoking matched healthy controls. The burden of CHIP mutations is higher in the LOE asthma group (incidence rate ratio 2.34, 95% CI: 1.05-5.45; exact Poisson rate test p = 0.039)

Highest frequency mutations were in the epigenetic regulators DNMT3A and TET2 (figure 1C). There were 54 CHIP clones at variant allele frequency ≥0.01 in the 96 LOE asthma patients and 6 in the 25 healthy controls (incidence rate ratio 2.34, 95% CI: 1.05-5.45; exact Poisson rate test p=0.039). There was a tendency towards multiple mutations in LOE patients (12/96, 12.5%) compared with healthy controls (0/25, 0%) which did not reach significance (p = 0.076).

Conclusion Non-atopic LOE asthma is associated with increased rates of high-impact mutations in IL-17RA. Dysregulation of the Th17 pathway in this patient group should be further investigated. There are higher rates of CHIP in LOE asthma patients than the general population, and relatively higher rates of multiple mutations than other disease populations, requiring confirmation by additional controls in our further ongoing work. Both IL-17RA polymorphisms and CHIP may contribute to the aetiopathogenesis of LOE asthma.

S111 SINGLE CELL PROFILING OF CORTICOSTEROID-RESPONSIVE AND NON-RESPONSIVE ENDOTYPES OF SEVERE ASTHMA

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Introduction Severe asthma is heterogeneous. The molecular mechanisms underlying corticosteroid-refractory severe asthma not driven by type-2 cytokines or associated with type-2 biomarkers (blood eosinophils, exhaled nitric oxide [FeNO]) remain poorly understood. Type-2 high severe asthma can also demonstrate relative resistance to inhaled corticosteroids, particularly in the 1/3 patients with persistently elevated FeNO despite electronically-monitored high-dose therapy: 'FeNO non-suppressors' with resistant type-2 inflammation.

Objective Characterise lymphoid populations in the lower airways, as compared to the upper airways, across immunologically distinct severe asthma phenotypes using scRNAseq approaches and provide novel insight into mechanisms driving corticosteroid resistant inflammation.

Methods This study enrolled 43 participants (healthy n=12; severe asthma n=31). Patients with severe asthma were stratified by blood eosinophil count and FeNO status into FeNO low (n=14), FeNO suppressors (n=8), and FeNO non-suppressors (n=9) following proven adherence to inhaled therapy and suppression testing. Bronchoscopy was performed on healthy non-atopic controls and patients with severe asthma. Endobronchial biopsies and nasal brushings underwent 10X genomics single cell immune profiling.

Results From paired endobronchial biopsies and nasal brushings approximately 85,000 high-quality lymphoid, 35,000 myeloid and 99,000 epithelial/stromal cells were profiled. Analysis of the lymphoid compartment identified 20 distinct cell states. Clear compartmental segregation by anatomical site was observed across health and severe asthma. There was greater diversity and abundance of tissue-resident memory T cells (ITGAE/CD69/ CXCR6+ CD8 TRM expressing GZMA, GZMB and GZMH), including cytokine secreting activated subsets (IFNhi/TNFhi and CCL4hi cells) in the lung compared to the upper airway, more notably in severe asthma compared to health. In addition, T/NK cell populations with high expression of GZMK and EGFR receptor ligand AREG (amphiregulin) were enriched in severe disease.

Conclusions These findings highlight compartment-specific lymphoid organisation and identify novel T cell subsets that may contribute to airways inflammation. In ongoing work transcriptomic data from myeloid and stromal populations are being analysed and integrated with single cell T cell repertoires, comparing airway compartments and delineating immunological mechanisms underlying distinct corticosteroid resistant asthma phenotypes.

S112 SINGLE-CELL TRANSCRIPTOMIC ANALYSIS REVEALS THE BRONCHOALVEOLAR LANDSCAPE IN INTERSTITIAL LUNG ABNORMALITIES

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10.1136/thorax-2026-BTSabstracts.118

Background Interstitial lung abnormalities (ILAs) are computed tomography (CT)-detected, non-dependent parenchymal abnormalities involving ≥5% of a lung zone. ILAs are increasingly recognised as an early, preclinical stage of interstitial lung disease (ILD), with approximately 10% of cases progressing annually to established fibrotic lung disease.[1] However, there are currently no validated biomarkers to identify individuals at highest risk of progression.[1] Understanding the biological mechanisms underlying ILA progression is critical for identifying at-risk individuals, discovering novel therapeutic targets, and informing strategies for early intervention. This pilot study aimed to characterise the cellular landscape of the distal airspace in individuals with ILA and to identify early immune mechanisms associated with disease progression using single-cell RNA sequencing (scRNA-seq) of bronchoalveolar lavage fluid (BALf).

Methods Individuals with a multi-disciplinary diagnosis of interstitial lung abnormality (ILA) and healthy controls underwent BAL as part of the RETAIN Study (REC:23/EE/0221) at the Royal Brompton Hospital. BALf samples were processed using a fixation-based GEM-X Flex workflow (10x Genomics) and subjected to scRNA-seq. Libraries were sequenced on NovaSeq X Plus (150 bp paired-end) at approximately 10,000 reads per cell (Novogene). Raw sequencing data were processed using Cell Ranger (10x Genomics) and analysed in R using Seurat v5. Standard quality-control filtering was applied to remove low-quality cells. Cell-type annotation was performed using canonical marker genes and validated with CellTypist automated annotation. Differential compositional analyses were conducted to compare cell populations across study groups.

Results scRNA-seq identified diverse immune cell populations in BALf, including multiple subtypes of macrophages, T cell subtypes, B cells, dendritic cell subtypes, mast cells, and NK cells. Macrophages were the dominant population, with marked inter-individual and inter-group heterogeneity. Progressive ILAs were characterised by enrichment of lymphocyte subsets and CHIT1+ macrophages suggesting these may drive early fibrotic progression.

Conclusion This study reveals distinct immune cell landscape alterations in the distal airspace associated with ILA progression. Enrichment of CHIT1+ macrophages and regulatory lymphocyte populations in progressive disease implicates early immune dysregulation preceding overt fibrosis. These findings lay the groundwork for identifying predictive biomarkers and will require validation in a larger cohort.

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S113 INITIATORS AND DRIVERS OF LUNG FIBROSIS – INSIGHTS FROM SPATIAL BIOLOGY AND IN VITRO STUDIES USING ALVEOLAR ORGANOIDS

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10.1136/thorax-2026-BTSabstracts.119

Background and Aims Alveolar epithelial cell (AEC) dysfunction is thought to have a role in initiating and driving aberrant repair responses in Idiopathic pulmonary fibrosis (IPF). Current treatment only slow disease progression in established fibrosis, highlighting the continued need to understand pathogenic pathways in IPF, particularly in earlier stages of fibrotic process.

We hypothesised that T1-IFN–activated myeloid cells co-localise with and are activated by dysfunctional AECs and contribute to alveolar injury.

Methods To investigate this, we examined the spatial distribution and co-localisation of T1-IFN responsive myeloid cells in IPF lung tissue using spatial transcriptomic and imaging mass cytometry (IMC). A hypothesis-driven IMC panel was applied to lung tissue from IPF patients with histologically confirmed usual interstitial pneumonia. Data were analysed using the Spatial Omics Oxford analysis pipeline (SpOOx) v2.0[1].

For in vitro studies, primary AT2 cells isolated from excess healthy lung tissue were used to generate alveolar organoids (AOs). Bleomycin induced injury was used to model AEC dysfunction. Injured AOs were co-cultured with wild-type (WT) and IFNAR deficient monocyte-like THP-1 cells to assess epithelial-myeloid interactions and T1-IFN signalling.

Results Regional transcriptomic analyses of IPF lung tissue identified increased T1-IFN signalling in IPF alveolar macrophages and monocytes localised to the alveolar barrier in early disease. From IMC analysis, intermediate AEC subsets were identified and exhibited markers of senescence together with increased cytoplasmic IFNβ and TNFα expression. Amongst alveolar epithelial subtypes, T1-IFN activated macrophages were co-located only with dysfunctional AECs.

Bleomycin-induced injury of AOs recapitulated key features seen in situ. Addition of WT but not IFNAR deficient THP-1 cells to injured AOs increased expression of genes associated with epithelial senescence and inflammation. Interferon stimulated gene expression in monocytes was increased in co-culture with injured AOs, with reduced expression in uninjured AOs, compared to unstimulated THP-1 cells.

Conclusions These findings suggest that T1-IFN activated monocytes and macrophages are linked to dysfunctional and senescent alveolar epithelial cell states and appear involved in early stages of fibrotic processes where they amplify AEC inflammation and cellular senescence. Targeting this pathway may offer an opportunity to intervene in early disease processes prior to irreversible fibrosis.

S114 A 3D MULTI-OMICS ATLAS OF HUMAN LUNG FIBROSIS DEFINES MOLECULAR–ARCHITECTURAL NICHES

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Introduction Human lung fibrosis is a progressive and fatal disease marked by spatially heterogeneous scarring, yet the complex three-dimensional (3D) tissue microenvironments in which fibrosis develops remain poorly defined. Conventional transcriptomic studies lack 3D architectural context, while imaging alone cannot dissect disease-relevant molecular changes.

Aims and Objectives To generate a 3D multi-omics atlas integrating lung microarchitecture with spatial gene and protein expression in human lung fibrosis.

Methods Micron-scale three-dimensional X-ray histology (XRH) was integrated with spatial transcriptomic and proteomic profiling of diagnostic fibrotic and control lung tissue. Molecular data were digitally co-registered within the 3D XRH volume, enabling direct integration of molecular expression with local structural metrics including tissue density and vascular proximity.

Results Integrated 3D analysis revealed marked structural heterogeneity in fibrotic tissue, with increased local tissue density, altered airway and vascular topology, and loss of normal alveolar geometry. Spatial transcriptomic clustering resolved discrete anatomical compartments, segregating fibrotic from control regions and defining alveolar, vascular and airway domains, as well as quantifying molecular changes surrounding and within regions of fibrotic remodelling. Integration of three-dimensional structural metrics with molecular expression patterns revealed coordinated remodelling of epithelial, stromal and vascular programmes, defining distinct molecular–architectural niches within the fibrotic lung.

Conclusions We have established a 3D multi-omics atlas of human lung fibrogenesis, enabling high-resolution mapping of molecular–structural relationships and providing a framework for mechanistic investigation and translational model benchmarking.

Focused pleural medicine: advances in diagnostic and therapeutics

S115 SMALL EFFUSION, BIG IMPACT: THE ROLE OF ULTRASOUND-GUIDED ASPIRATION OF MINIMAL PLEURAL EFFUSIONS IN CANCER DIAGNOSIS AND STAGING

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10.1136/thorax-2026-BTSabstracts.121

Introduction and Objectives Small pleural effusions detected on CT imaging are frequently regarded as insufficient for diagnostic aspiration. Previous studies suggest that minimal pleural effusions may represent early malignant pleural involvement and are associated with adverse prognosis in non-small-cell lung cancer.[1] We aimed to evaluate the diagnostic yield, staging implications and safety of ultrasound-guided aspiration of very small pleural effusions in patients undergoing investigation for suspected malignancy.

Methods We performed a retrospective review of patients undergoing ultrasound-guided pleural aspiration between 2009 and 2023. Eligible patients had a CT-detected pleural effusion measuring ≤10 mm in depth and confined to the costophrenic recess. Cytological findings were reviewed to determine their contribution to establishing a diagnosis of malignancy and influencing TNM staging. Procedural safety was also evaluated.

Results Thirty-three patients met the inclusion criteria. Pleural fluid cytology was positive for malignancy in 13 patients, corresponding to a diagnostic yield of 39.4%. Of these 13 patients, pleural fluid cytology provided the only pathological confirmation of malignancy in six (46.2%). Four patients (30.8%) were upstaged based on positive pleural fluid cytology findings. In the remaining three patients (23.1%), cytology contributed to the diagnosis alongside other investigative modalities. No immediate or delayed procedure-related complications were observed.

Conclusions Ultrasound-guided aspiration of very small pleural effusions is safe and provides a clinically meaningful diagnostic yield. Pleural fluid cytology can establish a diagnosis of malignancy and influence tumour staging, with important implications for subsequent management. These findings suggest that small pleural effusions should not be dismissed as diagnostically insignificant and support consideration of aspiration before proceeding to more invasive investigations in appropriately selected patients.

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S116 STANDARDISING PLEURAL MANAGEMENT IN SUSPECTED MALIGNANT PLEURAL EFFUSION USING UNIFIED PRE-TEST PROBABILITY AND THE RED SCORE: A QUALITY IMPROVEMENT PROJECT

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10.1136/thorax-2026-BTSabstracts.122

Background Malignant pleural effusion (MPE) causes significant symptom burden, repeated procedures, and unplanned hospital admissions. Many patients undergo repeated therapeutic aspirations despite a high pre-test probability of malignancy, delaying definitive intervention. The RED score predicts effusion recurrence timing but is not routinely used in pleural pathways.[1]

Aim To reduce emergency admissions and earlier-than-planned pleural reviews through a standardized pathway incorporating pre-test probability assessment and RED score–guided decision-making.

Methods A two-cycle quality improvement project was conducted in adults with known or suspected MPE. Cycle 1 (retrospective baseline, n=48) audited emergency admissions or



Abstract S115 Figure 1 Image A shows mediastinal thickening (blue arrow) and minimal pleural effusion (green arrow). Image B shows enlarged ultrasound image with blue arrow marking the needle in the fluid



Malignant Pleural Effusion – QIP Data Collection Proforma
(For use in patients with known or suspected malignant pleural effusion undergoing pleural assessment or intervention)

Patient Details
- Hospital number: _____
- Initials: _____
- Age: ____
- Date of assessment: ___ / ___ / ____
- Location: ☐ Pleural clinic ☐ Ward ( ) ☐ Other: _____

Section 1 – Pre-Test Probability of Malignancy
Clinical features (tick if present)
Section 1 – Pre-Test Probability of Malignancy
Clinical features (tick if present)
☐ Current smoker/or Ex-smoker
☐ Known malignancy
☐ Asbestos exposure
☐ Unintentional weight loss and/or systemic symptoms
Radiological features (CT or thoracic ultrasound)
☐ Pleural nodularity
☐ Pleural thickening >10 mm
☐ Pleural-based mass
☐ Diaphragmatic pleural involvement
☐ Mediastinal pleural involvement
☐ Chest wall invasion
Overall pre-test probability:
☐ **High** ☐ **Low / Indeterminate**

Section 2 – Breathlessness Assessment (VAS – last 24 hours)
Question to patient:
"On average, how breathless have you felt in the last 24 hours?"
Dyspnoea VAS score (cm): ____ /10

No breathlessness at all — Worst possible breathlessness

Section 3 – Clinical Observations
- Respiratory rate (breaths/min): ____
- Oxygen therapy:
  ☐ Room air ☐ Supplemental oxygen ☐ HFNC ☐ NIV

Section 4 – Thoracic Ultrasound Assessment
- Maximum pleural effusion depth (cm): ____
  (Measure from visceral to parietal pleura)

Section 5 – RED Score Calculation
Step 1
Effusion depth (cm) + Dyspnoea VAS (cm) = ____
☐ **<14 → Low RED score**
☐ **≥14 → proceed to Step 2**

Step 2
Respiratory rate ≥22 breaths/min?
☐ Yes ☐ No
(Add 1 point if Yes)

Final RED Score Category
(Tick one based on the final numerical score)
☐ **Low RED score**
Total score <14
→ Low risk of early recurrence
→ Unlikely to require another pleural procedure within 6 weeks
☐ **Medium RED score**
Total score 14–20
→ Moderate risk of recurrence
→ Likely to require another pleural procedure within 2–6 weeks
☐ **High RED score**
Total score ≥21
→ High risk of early recurrence
→ Likely to require another pleural procedure within 2 weeks
Important: RED score is used only in known or suspected malignant pleural effusion and predicts timing of recurrence, not diagnosis.

Section 6 – Cytology Status
- Cytology taken today: ☐ Yes ☐ No
- Cytology result:
  ☐ Positive ☐ Negative ☐ Pending
- Total number of pleural aspirations to date:
  ☐ 1 ☐ 2 or more

Section 7 – Performance Status
WHO / ECOG performance status:
☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Section 8 – Management Decision (QIP Intervention)
☐ Single diagnostic pleural aspiration only
☐ Early referral for definitive pleural intervention
☐ Medical thoracoscopy
☐ Indwelling pleural catheter (IPC)
☐ Symptom-focused management
Primary reason(s) for decision (tick all that apply):
☐ High pre-test probability
☐ RED score category
☐ Cytology result
☐ Performance status

Section 9 – Additional Clinical Notes / Comments
(Document any additional considerations, MDT advice, patient preference, or reasons for deviation from pathway)
_____
_____
_____
_____

Section 10 – QIP Outcome Tracking (Follow-up)
(To be completed at follow-up)
- Emergency hospital admission within **3 months** related to pleural effusion?
  ☐ Yes ☐ No

If yes, intervention required:
☐ Therapeutic pleural aspiration
☐ Chest drain insertion
- Date of definitive pleural intervention (*if performed*):
  ___ / ___ / ____

Completed by
- Name: _____
- Role: _____
- Signature: _____
- Date: ___ / ___ / ____

Abstract S116 Figure 1

earlier-than-planned pleural reviews within three months, the number of pleural procedures, and the achievement of definitive pleural management over six months. Cycle 2 was a prospective evaluation to May 2026 using a structured proforma capturing pre-test probability, dyspnea visual analog scale (VAS), thoracic ultrasound effusion size,[2] RED score, performance status, and predefined management strategy (n=18).

Results In Cycle 1, 16/48 patients (33%) had an emergency admission or earlier-than-planned pleural review within three months; 9/16 (56%) re-presented within 14 days. One-third of the patients required more than two pleural procedures, 73% achieved definitive management within three months, and 83% initially underwent therapeutic aspiration. In Cycle 2, 2/18 patients (11.1%) had recorded emergency readmissions or earlier-than-planned reviews. One event was primarily logistical: a patient with a high RED score admitted for chemotherapy incidentally developed symptomatic effusion recurrence. The clinically driven event rate was 1/18 (5.6%). Most patients required no more than two procedures, and the majority with completed follow-up achieved definitive management within three months or by data censoring criteria.

Conclusion A proforma-based pathway incorporating pre-test probability and RED score–guided decision-making was associated with a reduction in clinically driven emergency admissions from 33% to 5.6% and a trend towards earlier definitive pleural management.

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S117 MALIGNANT PLEURAL EFFUSION LIQUID BIOPSY EVALUATION (MAPLE): EXPLORING THE DIAGNOSTIC UTILITY OF PLEURAL FLUID CTDNA

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10.1136/thorax-2026-BTSabstracts.123

Introduction Pleural fluid (PF) cytology has an overall diagnostic yield of less than 60% in malignant pleural effusions (MPE), frequently necessitating invasive biopsy procedures for definitive diagnosis. Analysis of circulating cell-free DNA (cfDNA) in PF offers a promising minimally invasive approach for diagnosing MPE. Here, we evaluated the feasibility and diagnostic utility of PF cfDNA profiling using shallow whole-genome sequencing (sWGS).

Methods Matched PF and blood were collected from 14 patients with suspected MPE, alongside standard of care tests. Cell-freeDNA was extracted from matched PF and plasma, with 300pg analysed by sWGS. Copy number (CN) aberrations indicative of circulating tumour DNA (ctDNA) were identified using a customised ichorCNA pipeline, as described previously.[1] Sensitivity and specificity were calculated relative to final pathological outcomes using an intention-to-diagnose approach for all evaluable reference cases, with 95% confidence intervals (Clopper–Pearson).

Results Patients had a mean age of 72.9 ± 8.1 years, with pleural effusions classified as malignant in 10/14 (71%) and benign in 3/14 (21%); 1 case with an indeterminate pathological outcome was excluded. Among 10 patients with MPE, cytology was malignant in 6/10 (60%) and unavailable in one case who proceeded directly to thoracoscopy. The sensitivity and specificity of sWGS-based detection of PF ctDNA was 80% (8/10 CI 44–97%) and 100% (3/3; CI 29.2–100%) respectively, for plasma ctDNA this was 30% (3/10; 95% CI 7–65%) and 100% (3/3; 95% CI 29–100%) respectively. Importantly, ctDNA was identified in 3/10 (30%) MPE cases with negative cytology. CN aberrations in PF ctDNA involved regions containing key lung cancer-associated genes, including amplifications in *EGFR* and deletions in *RB1*, *SMARCA4*, and *CDKN2A*.

Discussion These findings suggest sWGS-based profiling of PF cfDNA is a feasible complimentary test to cytological examination in diagnosing MPE, offering a rapid, low-cost method of detecting genomic CN aberrations associated with malignancy. Larger studies are required to validate the technology and investigate the role of advanced diagnostics utilising ctDNA.

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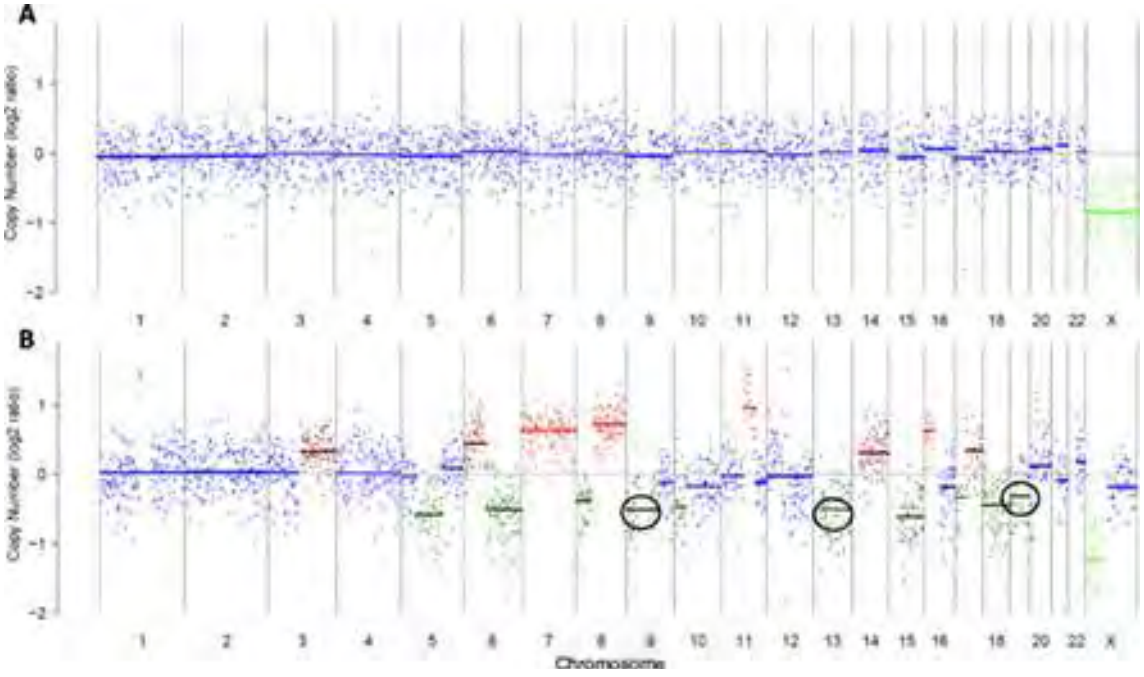
S118 PLEURAL COMPLICATIONS ASSOCIATED WITH TYROSINE KINASE INHIBITORS

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Introduction Tyrosine kinase inhibitors (TKI) are widely used in the treatment of solid tumours. However, clinical benefit is often limited by toxicity. Although TKIs are increasingly recognised as causing pleural complications in clinical practice, there is limited real-world evidence to substantiate this. We set out to investigate the features predictive of pleural complications associated with TKIs.

Methods All adult patients who received at least one cycle of a TKI for a solid tumour between 1 January 2020 and 31 January 2024 at a tertiary referral centre were included. Records were reviewed for pleural complications that occurred within



Abstract S117 Figure 1 sWGS profiles for Patient M1: (A) plasma cfDNA and (B) PF cfDNA, demonstrating the presence of ctDNA in PF only. CN amplification and deletion are shown in red and green, respectively. CN amplification present in key genes associated with lung cancer are illustrated by black circles

90 days of completing treatment. We used multivariable Cox proportional hazards models to determine the cumulative incidence of pleural events and identify independent risk factors. All models were stratified by primary tumour type to account for patients with primary lung cancers.

Results The final cohort consisted of 680 drug regimens across 587 patients (55.4% male). Mean patient age was 66.0 years (± 12.2). Overall, 26.4% (n=155) of patients experienced at least one pleural complication, including pleural effusion (n=155), iatrogenic pneumothorax (n=13), and spontaneous pneumothorax (n=3). The Cox proportional hazards model revealed that the presence of lung metastases was the only significant risk factor for pleural complications (HR 1.8, 95% CI 1.16-2.78, p<0.01). Age, smoking history, pulmonary co-morbidities, and drug mechanisms were not significant.

Spontaneous pneumothorax was the rarest pleural complication. Two cases of spontaneous pneumothorax occurred in patients receiving cabozantinib for osteosarcoma, constituting 33.3% of the total osteosarcoma cohort (n=6). Hollow parenchymal lesions such as bullae, cysts, or cavities preceded pneumothorax development. Cox proportional hazards model identified VEGF inhibition as the only independent predictor for developing hollow parenchymal lesions (HR 7.6, 95% CI 2.0-28.8, p<0.03). Lesions only progressed to spontaneous pneumothorax in patients receiving cabozantinib for osteosarcoma.

Conclusions TKIs are associated with pleural complications, including pleural effusion and pneumothorax. The risk of pleural complication is increased by the presence of lung metastases and hollow lung parenchymal lesions.

S119 SEVERITY MEETS VULNERABILITY: INTEGRATING FRAILTY AND RAPID FOR RISK STRATIFICATION IN PLEURAL INFECTION

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Introduction Risk stratification in pleural infection commonly relies on disease-specific tools such as the RAPID score, which predicts mortality but does not capture physiological reserve. Frailty assessment using the Clinical Frailty Scale (CFS) may provide additional prognostic information beyond conventional severity scores.

Methods We conducted a retrospective cohort study of adults managed for pleural infection at a UK tertiary centre (n=49). Baseline frailty was assessed using the Clinical Frailty Scale. Disease severity was classified using RAPID score categories. Associations between CFS and RAPID were explored, and outcomes including mortality and surgical escalation were compared across combined frailty–severity strata.

Results CFS data were available for 47 patients. No significant association was observed between baseline frailty and RAPID score category, with similar RAPID distributions across frailty strata. Surgical escalation occurred in 26% (5/19) of patients with low frailty (CFS 1–3) compared with 0% (0/15) of those with severe frailty (CFS ≥6), despite comparable RAPID scores. Mortality was higher among patients with greater frailty, including those classified as low or intermediate risk by RAPID. Combined stratification using CFS and RAPID identified clinically distinct outcome groups not apparent when either score was applied in isolation.

Conclusion CFS and RAPID provide complementary, non-overlapping prognostic information in pleural infection. While RAPID reflects acute disease severity, frailty captures baseline vulnerability influencing outcomes and treatment escalation. Incorporating frailty assessment alongside RAPID scoring may improve risk stratification and support more individualised clinical decision-making

S120 LIPIDS IN PARAPNEUMONIC PLEURAL EFFUSIONS DRIVE TISSUE FACTOR–DEPENDENT THROMBIN GENERATION IN MACROPHAGES

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Background Parapneumonic pleural effusions (PPE) range from simple exudates to complex fibrinous collections which fail standard drainage, requiring fibrinolysis or surgery. As recognised contributors to immunothrombosis, understanding the impact of the PPE microenvironment on macrophage thrombin generation is essential.

Aim To define how the PPE microenvironment impacts macrophage thrombogenicity.

Methods Pleural fluid (PF) collected from PPE and transudate (TPE) controls was centrifuged to obtain acellular supernatant. PF cytokines were quantified by multiplex ELISA and metabolites by mass spectrometry. Healthy-donor monocyte-derived macrophages (MDM) were incubated with PPE or TPE. Thrombin generation was measured using fluorogenic thrombin assays, sandwich ELISA, and chromogenic factor X (FXa) assays. Multiple linear regression evaluated associations between metabolites and macrophage thrombogenicity.

Results PPE fluid has a metabolomic profile distinct from transudate, with the top differentially enriched metabolites being lipids. PPE fluid contains higher levels of pro-inflammatory cytokines TNF-α, IL-6 and IL-1β. PPE fluid enhanced macrophage thrombin generation, with reduced lag time and increased peak thrombin and endogenous thrombin potential (p<0.01). This effect was abrogated in the presence of tissue factor neutralising antibody (p<0.05) indicating a macrophage tissue factor production in response to PPE as the driver of coagulation. Lipids were removed from PF using cleanascite™. When PPE was depleted of lipids, the thrombin response was abrogated (p<0.05). When lipid transporter CD36 is blocked, the thrombin production is diminished in response to PPE suggesting a functional role of lipids. Etomoxir also trends towards thrombin production in response to PPE (p=0.16).

Conclusion The PPE microenvironment drives a procoagulant macrophage phenotype which is driven primarily by macrophage tissue factor activity. Findings implicate a functional role for lipids and possibly metabolic reprograming towards fatty acid utilisation.

NIV: why outcomes matter

S121 DIAGNOSIS-SPECIFIC PATTERNS OF HEALTHCARE UTILISATION AND OUTCOMES FOLLOWING ACUTE NON-INVASIVE VENTILATION FOR HYPERCAPNIC RESPIRATORY FAILURE

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Background Acute non-invasive ventilation (NIV) is an established treatment for acute hypercapnic respiratory failure (AHRF). However, patients receiving NIV represent a heterogeneous population with differing underlying diagnoses, healthcare utilisation patterns and clinical outcomes. We aimed to compare healthcare utilisation and outcomes according to the primary cause of AHRF and identify predictors of key clinical outcomes.

Methods We analysed prospectively collected audit data from adults receiving acute NIV for AHRF at a tertiary centre between April 2024-March 2025. Patient-level analyses were performed for demographics; whereas admission-level analyses were used for recurrent admission patterns, acute NIV success, home NIV initiation, mortality outcomes. Multivariable logistic regression was performed to identify independent predictors of outcomes.

Results A total of 208 acute NIV admissions from 177 patients were analysed. Significant differences were observed between diagnostic groups in body mass index (BMI), frailty, baseline CPAP use, ventilator pressures, home NIV initiation, in-hospital mortality and one-year mortality. COPD accounted for most admissions and recurrent healthcare utilisation, while obesity-related respiratory failure (ORRF) was associated with favourable short- and long-term outcomes despite greater obesity and frailty. In contrast, chest wall disease demonstrated the poorest outcomes across multiple measures, whereas neuromuscular disease was characterized by high acute NIV success and frequent transition to long-term ventilation. In multivariable analyses, increasing frailty independently predicted lower acute NIV success (OR 0.75, p=0.017), while higher BMI predicted home NIV initiation (OR 1.05, p=0.029). Long-term oxygen therapy was the only predictor of in-hospital mortality (OR 2.86, p=0.036). One-year mortality was independently associated with lower BMI (OR 0.93, p<0.001) and greater frailty (OR 1.37, p=0.012).

Conclusion Underlying diagnosis and demographics strongly influence healthcare utilisation, need for home ventilation and outcomes following acute NIV for AHRF. COPD accounted for most admissions and recurrent healthcare utilisation, whereas ORRF demonstrated favourable short- and long-term outcomes despite substantial disease burden. Frailty emerged as an important determinant of acute NIV success, while obesity was associated with increased transition to home NIV and improved longer-term outcomes. Incorporating diagnostic phenotype and frailty assessment into acute NIV pathways may improve prognostication, guide follow-up strategies and support more effective allocation of home ventilation services.

Abstract S121 Table 1 Comparison of patient characteristics and outcomes by primary diagnosis

Variable	Total (n=208)	COPD (n=134)	ORRF (n=53)	NMD (n=4)	CWD (n=6)	Other (n=11)	p-value
Unique patients	177	114	42	4	6	11	-
Median age, years (IQR)	73 (16)	73 (15)	70 (18)	56 (12)	76 (15)	78 (16)	0.114
Gender male, n (%)	77 (43)	52 (45)	16 (38)	2 (50)	2 (33)	5 (45)	0.914
Median BMI, kg/m^2 (IQR)	29 (16)	27 (12)	42 (15)	24 (6)	25 (8)	28 (10)	**<0.001**
Median frailty score (IQR)	4 (2)	4 (2)	5 (2)	5 (2)	6 (1)	4 (0)	**0.031**
Admission trajectory:							0.213
1 admission, n (%)	154 (74)	97 (72)	37 (70)	4 (100)	6 (100)	10 (91)	
≥2 admissions, n (%)	54 (26)	37 (28)	16 (30)	0	0	1 (9)	
Home NIV at admission, n (%)	17 (8)	9 (7)	7 (13)	0	1 (17)	0	0.107
CPAP at admission, n (%)	9 (4)	2 (1)	7 (13)	0	0	0	**0.001**
LTOT at admission, n (%)	26 (13)	19 (14)	5 (9)	0	1 (17)	1 (9)	0.810
Median pH (IQR)	7.26 (0.10)	7.27 (0.10)	7.25 (0.10)	7.31 (0.20)	7.25 (0.12)	7.24 (0.10)	0.568
Median pCO2 (IQR)	10.3 (3.2)	10.0 (3.2)	10.6 (2.1)	11.5 (7.0)	11.6 (1.7)	9.9 (5.0)	0.432
Median maximum IPAP at 1st day	19 (7)	18 (6)	23 (5)	21 (6)	21 (6)	20 (10)	**<0.001**
Median maximum EPAP at 1st day	5 (2)	4(3)	7 (3)	5 (1)	6 (1)	6 (4)	**<0.001**
Outcomes							
Acute NIV success, n (%)	146 (70)	92 (69)	41 (77)	4 (100)	2 (33)	7 (64)	**0.051**
New home NIV setup, n (%)	30 (14)	9 (7)	13 (25)	4 (100)	1 (17)	3 (27)	**<0.001**
In-hospital mortality, n (%)	36 (17)	24 (18)	5 (9)	0	4 (67)	3 (27)	**0.009**
1-year mortality, n (%)	92 (44)	63 (47)	14 (26)	1 (25)	5 (83)	9 (82)	**0.003**

Other includes CPE (n=1), Substance/drug (n=8), Other diagnosis (n=2). BMI Body mass index; CPAP continuous positive airway pressure; EPAP and IPAP expiratory and inspiratory positive airway pressure; LTOT long-term oxygen therapy; NIV non-invasive ventilation. Categorical variables: Chi-square test, Continuous variables: Kruskal-Wallis test.

S122 THE IMPACT OF ADHERENCE TO LONG-TERM NIV IN PATIENTS WITH COPD

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Background Long-term non-invasive ventilation (LT-NIV) extends overall survival in patients with chronic hypercapnic COPD and may reduce the number of moderate-to-severe exacerbations. However, the impact of adherence to LT-NIV has not been fully explored.

Aim The aim of the study was to investigate the minimal hours of LT-NIV use that is related to improved clinical outcomes.

Methods 392 patients with COPD (65±9 years, 59% females), who were set up on LT-NIV between 2011 and 2022 were included. Overall-survival at five years (OS), exacerbation-free survival (EFS) and hospitalisation-free survival (HFS) were investigated with Cox-regression. Reduction in exacerbations and hospitalisation post-NIV were compared with Mann-Whitney test between the adherent and non-adherent groups. Different hours of usage (≥4h, ≥6 and ≥8h) were tested.

Results Of the tested cut offs, ≥6h of NIV use was associated with clinical benefits. Patients with high adherence (≥6h) had a tendency for higher OS (62% vs. 48% at 5 years, p=0.05). Whilst there was no difference in EFS (p=0.90), patients with high adherence had longer HFS (p=0.01). Although there was no difference in the reduction of moderate exacerbations after NIV setup (0.42±2.29/year vs. 0.51±3.21/year, high vs. low adherence groups, p=0.95), patients with high adherence had a greater reduction in hospital admissions post LT-NIV setup (0.86±1.81/year vs. 0.35±2.13/year, p=0.02). High adherence was not related to age, sex, BMI, lung function, symptoms, frailty, COPD medications, baseline blood gases or NIV settings, but was associated with being ex-smoker (p=0.03). The presence of asthma (p=0.04) and depression (p=0.02) were associated with low (<6h) adherence.

Conclusions At least 6h LT-NIV use was associated with a higher reduction of annual hospital admissions and longer hospital admission-free survival. Interventions to increase adherence to LT-NIV are necessary for patients with chronic hypercapnic COPD.

S123 BEYOND AGE AND ACIDOSIS: FRAILTY INDEPENDENTLY PREDICTS INPATIENT MORTALITY AFTER ACUTE NIV IN A TWO-SITE COHORT

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Introduction Inpatient mortality after acute non-invasive ventilation (NIV) for acute hypercapnic respiratory failure remains high.[1] Pre-NIV acidosis governs patient selection and risk assessment, yet may misclassify risk in older, frailer, obese and socioeconomically deprived populations.[2] Whether a bedside frailty measure predicts mortality better than the acidosis on which current decisions rest is unknown. We determined which routinely available characteristics independently predict mortality across two contrasting sites.

Methods Retrospective study of all consecutive acute NIV admissions at two sites (BHH, GHH), January–December 2025. The primary outcome, inpatient mortality, was defined identically at both sites. Categorical variables were compared using Chi-squared, continuous variables using Mann–Whitney U. Independent predictors were identified by multivariable logistic regression (complete-case) including Rockwood Clinical Frailty Score (CFS), pre-NIV pH, Body Mass Index (BMI), Index of Multiple Deprivation (IMD) decile, age and site (table 1).

Results Of 405 acute NIV episodes (Site1: 208, Site2: 197), inpatient mortality was 86/405 (21.2%), with no difference between sites (21.6% vs 20.8%, p=0.94). Mortality rose stepwise across CFS bands (0%, 11.8%, 17.6%, 50%; χ^2=16.1, p<0.001). CFS was the strongest independent predictor (adjusted OR 1.94/point, 95% CI 1.41–2.67, p<0.001; model AUC 0.78); age lost significance once frailty was included. Higher BMI independently predicted lower mortality (Adjusted Odds Ratio 0.97/unit, p≈0.05). Pre-NIV acidosis showed a univariable gradient (31.8% at pH<7.15 vs 14.3% at pH≥7.35) but was not independent after adjustment. Despite marked site differences in deprivation and length of stay, the most-deprived patients were younger and less frail, with no excess mortality.

Conclusion In a large, majority-deprived cohort, frailty was the dominant independent predictor of mortality after acute NIV,

Abstract S123 Table 1 Baseline characteristics and NIV outcomes by site

Characteristic	Site 1 (n=208)	Site 2 (n=197)	Combined (n=405)	p*
Age, years — median (IQR)	73.2 (64–79)	72.3 (64–79)	72.9 (64–79)	0.52
Female — n (%)	124 (59.6)	121 (61.4)	245 (60.5)	0.79
BMI ≥30 kg/m^2 — n (%)†	75/203 (36.9)	87/197 (44.2)	162/400 (40.5)	0.12
IMD decile 1–3 (most deprived) — n (%)	164 (78.8)	97 (49.2)	261 (64.4)	<0.001
COPD — n (%)	140 (67.3)	139 (70.6)	279 (68.9)	0.55
Obesity / OHS — n (%)	46 (22.1)	35 (17.8)	81 (20.0)	0.33
CFS ≥6 — n (%, of recorded)‡	60/137 (43.8)	94/139 (67.6)	154/276 (55.8)	<0.001
Pre-NIV pH — mean	7.244	7.252	7.248	0.19
Length of stay, days — median (IQR)§	9 (6–18)	5 (4–8)	6 (4–12)	<0.001
Inpatient mortality — n (%)	45 (21.6)	41 (20.8)	86 (21.2)	0.94

*IQR, interquartile range; IMD, Index of Multiple Deprivation; OHS, obesity hypoventilation syndrome; CFS, Clinical Frailty Scale. *χ^2 test for categorical variables, Mann–Whitney U test for continuous variables. †BMI recorded in 400/405 (BHH 203, GHH 197); percentages use recorded denominators. ‡CFS recorded in 276/405 (68%; BHH 137, GHH 139); CFS ≥6 expressed as a percentage of patients with a recorded score. §Length of stay recorded in 314/405 (BHH 135, GHH 179).*

outperforming both age and acidosis. A rapid bedside CFS at NIV initiation may stratify risk better than pH alone and support proportionate ceiling-of-care planning.

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S124 NONINVASIVE MONITORING FOR NONINVASIVE VENTILATION: USE OF TRANSCUTANEOUS CARBON DIOXIDE MONITORING TO GUIDE ACUTE NONINVASIVE VENTILATION IN PATIENTS WITH ACUTE HYPERCAPNIC RESPIRATORY FAILURE: REACT CO2, A MULTICENTRE STUDY

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Background Arterial Blood Gas (ABG) analysis remains the standard for monitoring patients with Acute Hypercapnic Respiratory Failure (AHRF) treated with Non-invasive Ventilation (NIV). ABGs are painful and provide snapshots of patient progress. Our previous observational study demonstrated that transcutaneous carbon dioxide (TcCO2) monitoring is concordant with arterial CO2 and offers live continuous data. This technique, however, needs validation to guide acute NIV.
Aim We aimed to determine whether using TcCO2 could reduce the number of ABGs by >30% when used to guide NIV for AHRF, as well as assessing its safety profile.
Methods This prospective, open-label RCT was conducted at four UK hospitals between November 2023 and April 2026. 284 patients were screened and 42 enrolled with AHRF due to COPD, OHS or NMD with pH 7.15-7.35 and bicarbonate (HCO3) >22. Participants were randomized 1:1 to TcCO2 monitoring or Standard Care (SC) with ABGs. The study duration was for 72 hours or until NIV cessation. Participants were followed up for 28 days. The primary outcomes were the total number of ABGs and safety of TcCO2. The secondary outcomes included pain scores.

Results 42 patients were randomized (mean age 67 years, 26 female), TcCO2: n=20, SC: n=22. COPD exacerbation was the most common cause of AHRF. Pre-NIV ABG values were comparable in both arms: mean pH 7.27, pCO2 9.3. The mean number of ABGs in the TcCO2 arm was 2.4 versus 5.86 in the SC arm (mean difference -3.46, 95% CI -4.67 to -2.26; p<0.001). 10 (50%) patients did not need any further ABGs after initiation of TcCO2. No safety concerns were identified in the TcCO2 arm. The mean pain score in the TcCO2 arm was 0.14 versus 3.14 in the SC arm (p<0.05). There was 1 death in the TcCO2 arm versus 3 in the SC arm.

Abstract S124 Table 1

	Standard care (*n22*)	$TcCO_2$ (*n20*)	95% CI for the difference	P-Value
Mean Age	67.55	66.75	-7.0266, 8.6166	0.8378
Mean pH (at NIV commencement)	7.27	7.28		0.855220
Mean CO2 (at NIV commencement)	9.496	9.069	-0.3631, 1.2171	0.281
Mean number of ABG's	5.864	2.4	2.2581, 4.6699	<0.0001
Mean Pain score	3.45	0.483	1.4311, 4.5029	0.0004

Conclusion TcCO2 monitoring reduced the number of ABGs by 60% and was less painful. No safety issues were identified, though this was a small study. The patients enrolled were representative of the UK population, making the results generalisable. Further work is required to assess implementation and safety issues in clinical practice.

S125 TRANSCUTANEOUS CO2 MONITORING VERSUS ARTERIAL PACO2 DURING ACUTE NIV IN ACUTE HYPERCAPNIC RESPIRATORY FAILURE: A SYSTEMATIC REVIEW

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Background Acute hypercapnic respiratory failure (AHRF) has a high morbidity and mortality with treatment with non-invasive ventilation (NIV)shown to improve outcomes. Arterial blood gas (ABG) analysis is the gold standard for monitoring carbon doxide and therefore both diagnosing AHRF and monitoring the response to NIV, but it is invasive, intermittent, and resource-intensive. Transcutaneous carbon dioxide ($TcCO_2$) monitoring is a continuous, non-invasive method of monitoring. We have reviewed the evidence to support the use of tcCO2 in the management of AHRF in adults requiring NIV.
Methods A systematic review was performed according to PRISMA -DTA and PRISMA -P guidelines. MEDLINE and CENTRAL were searched to June 2025. Data extraction included study design, correlation coefficients, Bland–Altman limits of agreement (LoA), device type, and clinical outcomes. Risk of bias was assessed using QUADAS-2. Owing to heterogeneity in devices and reporting metrics. Therefore, the primary focus of this paper is on concordance between $TcCO_2$ and $PaCO_2$. Secondary outcomes include tolerability of $TcCO_2$ devices, reduction of ABG sampling rate, impact on clinical decision-making, and usability in NIV for AHRF.
Results Fifteen studies were screened, and nine observational studies met inclusion criteria,comprising >381 adult patients. The included populations predominantly consisted of acutely unwell adults with AHRF, with mean ages from 66 to 74 years and baseline PaCO2 values of 5.9 -10.1kpa, indicating mild to moderate respiratory acidaemia. $TcCO_2$ demonstrated a consistently strong correlation with $PaCO_2$(r >0.85). Importantly, directional time-trend concordance between $TcCO_2$ and $PaCO_2$ in response to NIV therapy was observed. Agreement varied according to sensor site, device type, tissue perfusion, lactate levels, and measurement timing. $TcCO_2$ monitoring frequently reduced the need for repeated ABG sampling ranging from 4-24 hours and supported bedside ventilatory adjustments, including during overnight monitoring.
Conclusion In observational studies $TcCO_2$ was shown to be a safe, pain free and feasible trend-based replacement to ABG

monitoring during acute NIV, offering continuous assessment while reducing reliance on repeated arterial sampling and associated procedural discomfort. However, decisions regarding ventilator settings wee not made solely based on $TcCO_2$. There is no current basis for using $TcCO_2$ in place of $PaCO_2$, considering the wide variability seen in the LOA from different investigations and the lack of prospective evaluation on the utility of $TcCO_2$ in clinical decision-making. A prospective randomised controlled trial is required to evaluate it further.

S126 COST-EFFECTIVENESS OF ACCELERATED VERSUS STANDARD WEANING FROM NIV IN COPD: AN ECONOMIC EVALUATION WITHIN A RANDOMISED CONTROLLED TRIAL

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10.1136/thorax-2026-BTSabstracts.132

Introduction Acute NIV for exacerbation of COPD is an effective but resource-intensive intervention, commonly delivered in Respiratory Support Units (RSU) or Critical Care. However, evidence-based guidance on weaning is limited, with marked variation in clinical practice. We evaluated the cost and cost-effectiveness of accelerated versus standard step-wise weaning protocols within the 7-center NIV Outcomes Score directed weaning RCT (ISRCTN:64639614).[1]

Methods An RSU bed-day cost was estimated using staffing, capital, consumable and overhead costs at a typical site, aligned with BTS guidance on development and implementation of RSUs. The analysis assumes RSU provision of acute NIV. NHS resource utilisation was captured for 90 days following randomisation. Quality Adjusted Life Years (QALYs) were measured over 83 days using EQ-5D-5L. Results are presented as an incremental cost-effectiveness ratio. Uncertainty around estimates of cost-effectiveness were explored using cost effectiveness acceptability curves (CEACs), expressing uncertainty around willingness-to-pay for a unit of outcome.

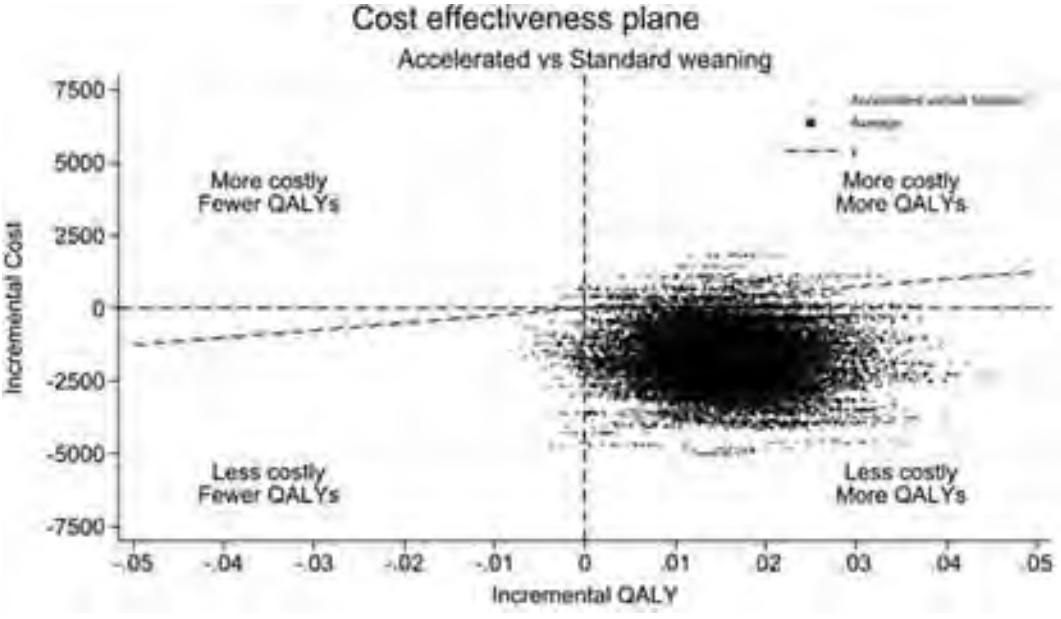
Results Between October 2022 and April 2025, participants were randomised to standard (n=83) or accelerated (n=81) weaning. Median (IQR) hours to successful weaning were: standard weaning=46 hours (41–70), accelerated weaning=6 hours (5–7), p<0.0001.

Total NHS costs over 90 days following randomisation were £1,685 lower in the accelerated weaning group than the standard weaning group (95%CI: -£3,922 to £552). For health-related quality-of-life, patients in the accelerated weaning group reported an additional 0.016 QALYs over the 83 days follow up from baseline, compared to standard weaning patients (95%CI: 0.001 to 0.030). The incremental cost effectiveness plane (figure 1) shows that accelerated weaning was both cost-saving and provided additional QALYs compared to standard weaning. The CEAC showed that accelerated weaning has a 97.16% probability of being cost-effective at a willingness-to-pay threshold of £25,000 per QALY.

Conclusions Accelerated weaning is clinically safe, effective, and cost-effective, compared to standard weaning. Cost-savings to the NHS are driven by patients requiring less time in the RSU. Accelerated weaning is associated with both lower mean costs and greater health benefit compared to standard weaning (a dominant health-economic model).

REFERENCE

1. Brewin K, *et al*. Standard versus accelerated weaning from NIV in COPD directed by the NIVO score:a RCT. *Thorax* 2025;**80**:A11.



Abstract S126 Figure 1 Cost-effectiveness plane to show accelerated weaning costs less and gains more QALYs than standard weaning

Eosinophilic exacerbations: what do we do?

S127 NOT ALL EOSINOPHIL-ASSOCIATED EXACERBATIONS ARE THE SAME : INFLAMMATORY PHENOTYPES OF UNTREATED COPD EXACERBATIONS

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10.1136/thorax-2026-BTSabstracts.133

Background Blood eosinophil count (BEC) is increasingly used to phenotype COPD exacerbations, guide corticosteroids use, and select patients for eosinophil-targeted therapies. However, exacerbations are heterogeneous inflammatory events, and blood eosinophilia may not uniformly reflect airway inflammation. We therefore characterised sputum inflammatory phenotypes during untreated COPD exacerbations and examined how type-2 biomarkers, BEC and fractional exhaled nitric oxide (FeNO), relate to airway inflammatory profiles.

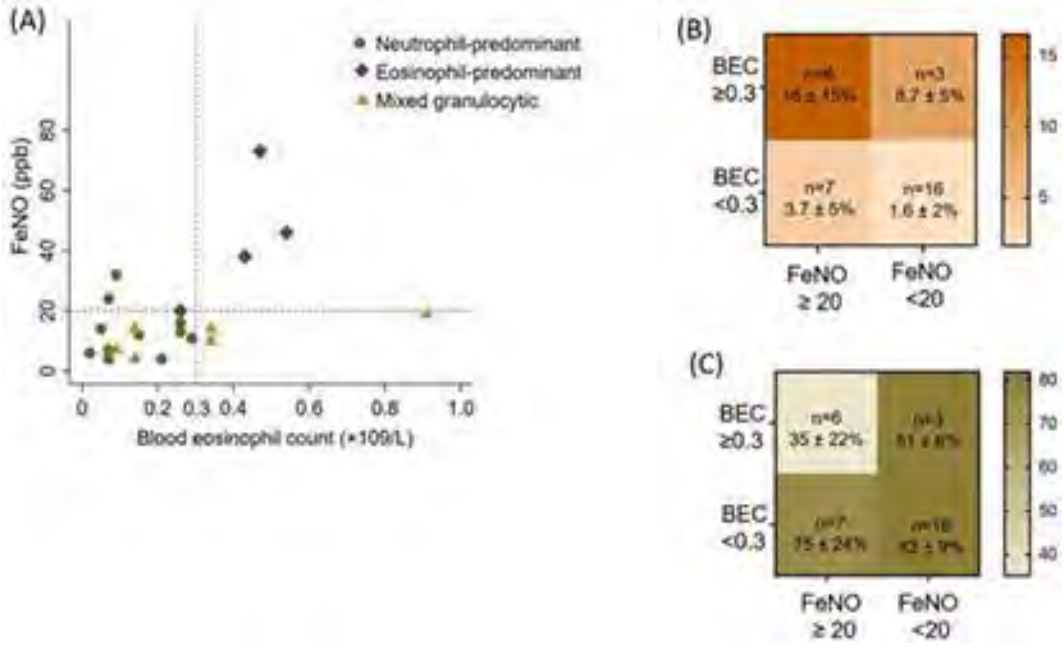
Methods Participants enrolled in the prospective INCLINE study (23/YH/0041) underwent assessment at exacerbation onset, prior to corticosteroids and/or antibiotics treatments. Exacerbations were classified according to sputum differential cell count: neutrophilic (eosinophils ≤3%, neutrophils >61%), eosinophilic (eosinophils >3%, neutrophils ≤61%), mixed granulocytic (eosinophils >3%, neutrophils >61%) or pauci-granulocytic (eosinophils ≤3%, neutrophils ≤61%) (PMID:33332995). Clinical and inflammatory characteristics were compared between exacerbation phenotypes.

Results Forty-two exacerbations (mean age 66±10 years; FEV_1 1.1±0.4 L) were characterised as neutrophilic (n=19, 45%), eosinophilic (n=5, 12%), mixed granulocytic (n=18, 43%) in the sputum (spontaneously collected).

Neutrophilic exacerbations demonstrated the greatest systemic inflammatory burden, with C-reactive protein levels (CRP 49.4±61.8 mg/L), increased neutrophil-to-lymphocyte ratio, and the highest frequency of bacterial or viral detection. Pure eosinophilic exacerbations demonstrated the highest FeNO (42 [29-60] ppb) levels, BEC (0.43±0.1 $\times 10^9$/L), and sputum eosinophil proportions (14.8±12.6%), with low CRP (4.3±0.5) mg/L.

Mixed granulocytic exacerbations showed elevated BEC comparable to eosinophilic exacerbations but FeNO remained low 14 [7-19] ppb with marked sputum neutrophilia (79.4±9.6%), indicating concurrent neutrophilic airway inflammation alongside eosinophilia. Unlike eosinophilic exacerbations, mixed granulocytic events were associated with significantly worsened CAT score, SGRQ score and FEV_1 from stable state (all $p<0.05$).

Among exacerbations with elevated BEC (≥0.3×10^9/L), mixed granulocytic inflammation was the predominant phenotype (10/16, 62.5%), whereas only four (25%) were purely eosinophilic. In contrast, FeNO-high exacerbations (≥20ppb) were predominantly eosinophilic, while BEC-high/FeNO-low exacerbations were predominantly mixed granulocytic. No



Abstract S127 Figure 1 Relationship between blood eosinophil count (BEC), fractional exhaled nitric oxide (FeNO), and sputum inflammatory phenotypes during COPD exacerbations (A) Scatter plot showing the distribution of sputum inflammatory phenotypes according to blood eosinophil count (BEC, x109 cells/L) and fractional exhaled nitric oxide (FeNO, ppb). Dashed lines indicate conventional thresholds for elevated BEC (20.3 x109 cells/L) and elevated FeNO (≥20 ppb). Neutrophilic exacerbations clustered predominantly in the BEC-low/FeNO-low quadrant, eosinophilic exacerbations in the BEC-high/FeNO- high quadrant, while mixed granulocytic exacerbations were predominantly observed in the BEC-high/FeNO-low quadrant; (B) Mean sputum eosinophil proportion (%) according to combined BEC (×109 cells/L) and FeNO (ppb) categories. The highest sputum eosinophil burden was observed in the BEC-high/FeNO-high group; (C) Mean sputum neutrophil proportion (%) according to combined BEC and FeNO categories. The lowest sputum neutrophil burden was observed in the BEC-high/FeNO-high group, whereas BEC-high/FeNO-low exacerbations retained marked neutrophilic airway inflammation

eosinophilic exacerbations occurred in the BEC-low/FeNO-low group (see figure 1). Further cellular immunophenotyping and cytokine analyses across exacerbation phenotypes are ongoing.

Conclusion Eosinophil-associated COPD exacerbations are biologically and clinically heterogeneous. Blood eosinophilia during COPD exacerbations frequently reflects mixed granulocytic rather than purely eosinophilic airway inflammation. FeNO may provide complementary information that further distinguishes BEC-high exacerbations into distinct inflammatory phenotypes.

S128 REPEATED BLOOD EOSINOPHIL COUNTS PREDICT EXACERBATION SUB-TYPES IN THE CHARACTERISATION OF COPD EXACERBATIONS STUDY (COPE)

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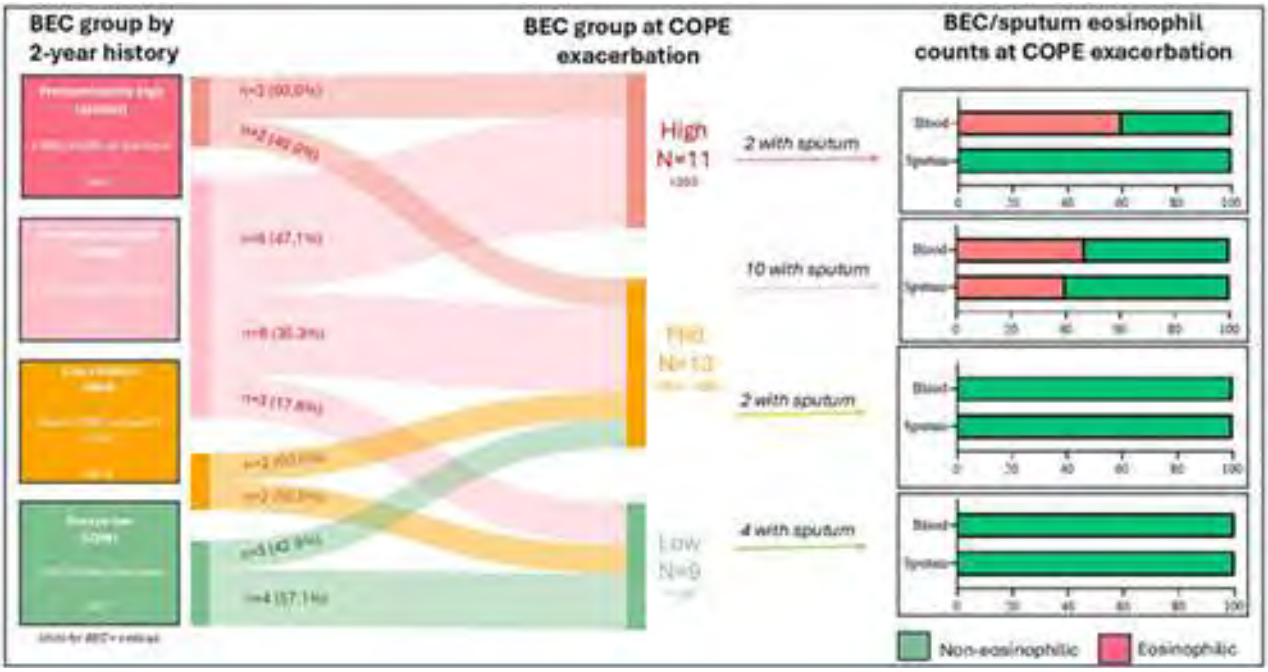
10.1136/thorax-2026-BTSabstracts.134

Introduction Eosinophilic inflammation in COPD is associated with an increased likelihood of response to inhaled corticosteroid and type 2 (T2)-targeting biologics. Higher blood eosinophil counts (BECs) are associated with increased T2 inflammation in the lungs. A previous study demonstrated that multiple BECs in the stable state can help identify individuals with a susceptibility for high BEC during exacerbation. We have now investigated the association between multiple BEC and pulmonary eosinophilia during exacerbations.

Methods Blood and sputum samples were collected from patients with COPD enrolled in the Characterisation of COPD Exacerbations study (COPE) during exacerbation, prior to acute treatment. Historic BEC (over 2 years), taken as part of routine care prior to this event, were retrospectively analysed; patients were grouped by multiple BEC: 'predominantly high (pHIGH)', 'intermittently high' (iHIGH), 'low variable' (VAR) and 'persistently low' (LOW) (definitions presented in figure 1). Exacerbations were classed as 'eosinophilic' if blood and sputum eosinophil counts were ≥300cells/uL and/or ≥3%, respectively.

Results A total of 53 COPD patients were enrolled; BECs were collected during exacerbation for n=33, 19 of whom also produced sufficient sputum for an eosinophil count. COPD patients were 73.0 (7.4) years of age, 60.4% female, 28.3% were current smokers and 96.2% ICS users, with an annual exacerbation rate of 4.0 (3.0). Using multiple historic BECs: pHIGH, iHIGH, VAR and LOW groups represented 15.2, 51.2, 12.1 and 21.5% of the population, respectively. High BEC at exacerbation was observed in 60.0 and 47.1% of pHIGH and iHIGH individuals, respectively. Elevated sputum eosinophil counts were significantly different between groups during exacerbation (p=0.047) and were only observed in the iHIGH group (40.0%). No patients in the VAR or LOW groups had high BEC or sputum eosinophil counts at exacerbation.

Conclusion We have demonstrated that eosinophilic exacerbations are most frequently detected in pHIGH and iHIGH groups, confirming previous observations that iHIGH individuals are susceptible to eosinophilic exacerbations. We now extend these findings to show that individuals with previous elevated BEC also show pulmonary eosinophilia at exacerbation. These findings have clinical relevance with respect to the identification of individuals most likely to respond to T2-targeted therapy.



Abstract S128 Figure 1 Exacerbation sub-types associated with groups defined using multiple BECs

S129 THE BURDEN AND CLINICAL CHARACTERISATION OF THE EOSINOPHILIC PHENOTYPE IN PATIENTS WITH RECURRENT SEVERE COPD EXACERBATIONS

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10.1136/thorax-2026-BTSabstracts.135

Background COPD exacerbations are a leading cause of hospital admissions with significant morbidity and mortality. Eosinophilic COPD phenotype as a treatable trait has gained significant attention with the approval of biologics. We evaluated the burden of eosinophilic COPD in patients with recurrent severe exacerbations and compared various clinical parameters to better define this population.

Methods This retrospective observational cohort study included patients with recurrent severe exacerbations (3 or more) between June 2024-May 2025), identified from electronic patient records. Patients without documented spirometry were excluded. The eosinophilic phenotype (≥ 300 cells/ μL). Historical eosinophil counts were reviewed and classified as isolated, recurrent or persistent cohort. Demographics, seasonal variation, pulmonary rehabilitation (PR), vaccination history, mucus plugging on imaging, sputum microbiology and exacerbation history were compared between patients with recurrent cohort versus isolated or persistent cohort.

Results 255 patients had recurrent severe exacerbations and 22.7% (n=58, mean age; 74+/-10, males; 59%, BMI; 24+/-7) had eosinophilic phenotype. Longitudinal tracking showed 17% (n=10) had a single isolate, 7% (n=4) had persistent and 76% (n=44) had recurrent Eosinophilia. The recurrent cohort had a significant variation in seasonal testing during winter (67% v/s 7%; P < 0.001), summer (71% v/s 12%; P < 0.001) and autumn (60% v/s 10%; P= 0.009.

The recurrent cohort demonstrated a trend toward higher PR completion (38% v/s 10%), imaging confirmed mucus plugging (48% v/s 14%) and positive sputum isolates (48% v/s 14%) although not statistically significant.

Influenza vaccination uptake was significantly higher in the recurrent cohort (47% v/s 7%; P= 0.026). Patients with recurrent eosinophilia also had a significantly higher rate of historical moderate exacerbations (P= 0.048), though no difference in severe exacerbations (figure 1).

Conclusion The eosinophilic phenotype is common among patients with recurrent severe COPD exacerbations and is predominantly recurrent over time. Recurrent eosinophilia is associated with seasonal variation and higher moderate exacerbations, supporting its role as a clinically relevant and potentially treatable trait. Higher rates of PR completion and vaccinations in this group may reflect the benefits of earlier clinical recognition and intervention.

S130 MOST HOSPITALISED COPD EXACERBATIONS LACK A DEFINABLE VIRAL OR EOSINOPHILIC ENDOTYPE: A REAL WORLD COHORT STUDY

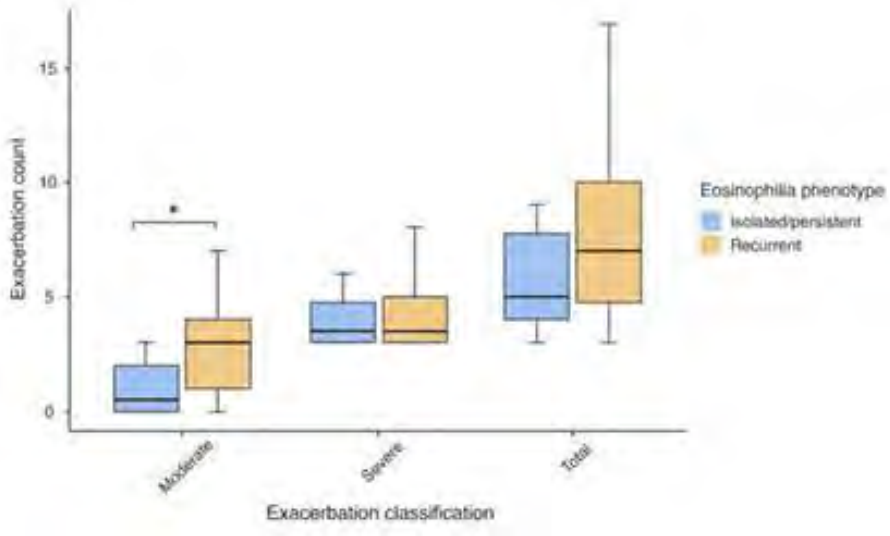
[1]C Morgan, [2]R Granell, [1]C Hyams, [1]JW Dodd. *[1]Academic Respiratory Unit, University of Bristol/North Bristol NHS Trust, Bristol, UK; [2]Centre for Academic Primary Care, Bristol Medical School, Bristol, UK*

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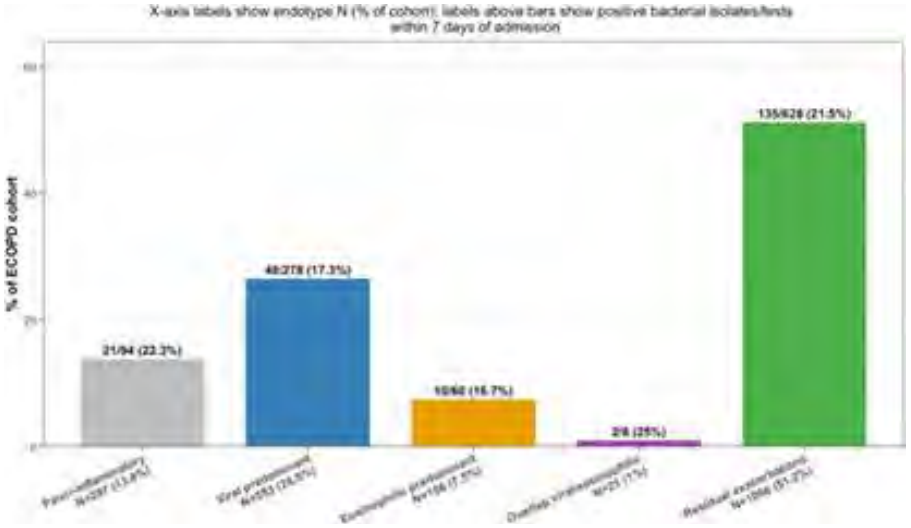
Introduction COPD exacerbations (ECOPD) are a leading cause of death worldwide. Predominant biological mechanisms (endotype) include viral (T1 inflammation), eosinophilic (T2), and pauci-inflammatory. A greater understanding of these endotypes may inform targeted therapeutics strategies.[1] However, the distribution and characteristics of these endotypes remain incompletely described in real-world hospitalised populations.

Methods We analysed 2,549 index ECOPD admissions (>45 years) from the AvonCAP UK infection surveillance study (2022–2024). Endotypes were defined as: viral (≥1 respiratory virus within 7 days of admission), eosinophilic (BEC $>0.30\times10^9$/L on admission), pauciinflammatory (CRP <20 mg/L and WCC $<11\times10^9$/L on admission, virus/eosinophil negative), and residual inflammatory (remaining cases). Endotype distribution and bacterial culture yield were assessed.

Results Complete data (viral testing, CRP, WCC, BEC) were available for 2,083/2,549 (79.2%) admissions; viral testing was not performed in 438 cases. The distribution of ECOPD endotypes was as follows: 553 (26.5%) viral predominant,



Abstract S129 Figure 1



Abstract S130 Figure 1 Distribution of ECOPD endotypes & bacterial culture yield in a real-world hospitalised cohort. X-axis labels show endotype N (% of cohort); labels above bars show positive bacterial isolates/tests within 7 days of admission

156 (7.5%) eosinophilic predominant, 21 (1%) mixed viral/eosinophilic, 287(13.8%) pauci-inflammatory and 1066 (51.2%) residual inflammatory (figure 1). Among viral cases, the most frequently identified pathogens were SARSCoV2 (44.6%, n=256), influenza A (20.4%, n=117), rhinovirus (12.5%, n=72), and respiratory syncytial virus (10.8%, n=62). There was no difference in sputum microbiological yield across endotypes ($\chi^2(4)=2.98$, p=0.56). The most commonly cultured organisms were *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Pseudomonas aeruginosa*. Potential confounders were explored: inhaled corticosteroid use at admission and antibiotic exposure within 14 days prior to admission did not differ between groups (ICS: $\chi^2(4)=2.14$, p=0.71; antibiotics: $\chi^2(4)=6.14$, p=0.19). Acute oral corticosteroid exposure was not captured and may have influenced endotype classification.

Conclusions Distinct ECOPD endotypes can be identified in hospitalised patients with minimal overlap; however, over half of exacerbations fall into a residual inflammatory group without evidence of viral or eosinophilic drivers, using routinely available biomarkers. This large uncharacterised group highlights a major gap in current biological stratification and underscores the need for improved mechanistic understanding. Current biomarkerbased classification fails to characterise the majority of hospitalised ECOPD, limiting the immediate clinical utility of precision treatment strategies.

REFERENCE

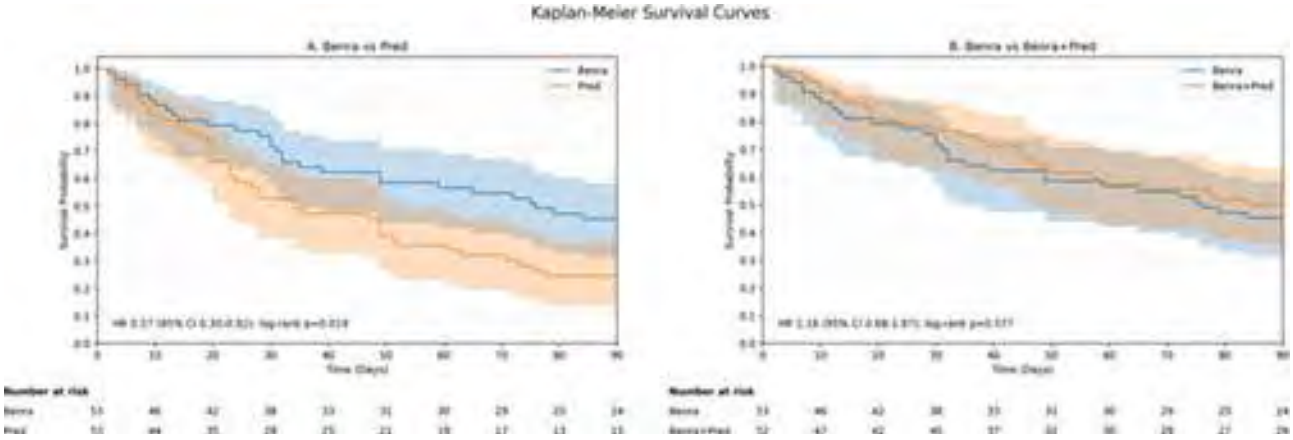
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S131 ROBUSTNESS OF TIME-TO-EVENT BENEFITS ASSOCIATED WITH BENRALIZUMAB-CONTAINING TREATMENT IN EOSINOPHILIC ASTHMA AND COPD: SENSITIVITY ANALYSES FROM THE ABRA TRIAL

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10.1136/thorax-2026-BTSabstracts.137

Background In the ABRA trial, benralizumab-containing treatment (benralizumab alone or in combination with prednisolone) reduced the risk of treatment failure at 90 days following eosinophilic asthma and COPD exacerbations, compared with prednisolone alone.[1] However, it is unclear whether this effect



Abstract S131 Figure 1 Kaplan-Meier plot of time to first treatment failure event

was driven by benralizumab or influenced by concomitant prednisolone use. This study assessed the consistency of treatment effects across individual treatment groups using sensitivity analyses.

Methods Post-hoc Cox proportional hazards models were used to estimate pairwise hazard ratios (HRs) with 95% confidence intervals (CIs) across the original treatment groups: benralizumab alone, benralizumab plus prednisolone, and prednisolone alone. Sensitivity analyses evaluated consistency of treatment effects within benralizumab-containing regimens and potential heterogeneity between treatment strategies.

Results A total of 158 patients were randomised in the eosinophilic exacerbation cohort, with 53 allocated to benralizumab alone, 52 to benralizumab plus prednisolone, and 53 to prednisolone alone. Group-specific analyses showed that benralizumab monotherapy was associated with improved outcomes compared with prednisolone (figure 1A; HR 0.57, 95% CI 0.35-0.92; p=0.019). There was no evidence of a difference between benralizumab monotherapy and benralizumab plus prednisolone combination therapy (figure 1B; HR 1.16, 95% CI 0.68-1.97; p=0.577). Overall, treatment effects appeared consistent across benralizumab-containing regimens.

Conclusion Sensitivity analyses demonstrated consistent treatment benefit across benralizumab-containing regimens, with no meaningful difference associated with the addition of prednisolone. These findings support the robustness of the pre-specified benefit of benralizumab-containing treatment with no evidence of benefit from prednisolone alone or in combination.

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S132 VALIDATION OF THE BAT RISK SCORE IN A MULTI-CENTRE COHORT OF HOSPITALISED COPD EXACERBATIONS

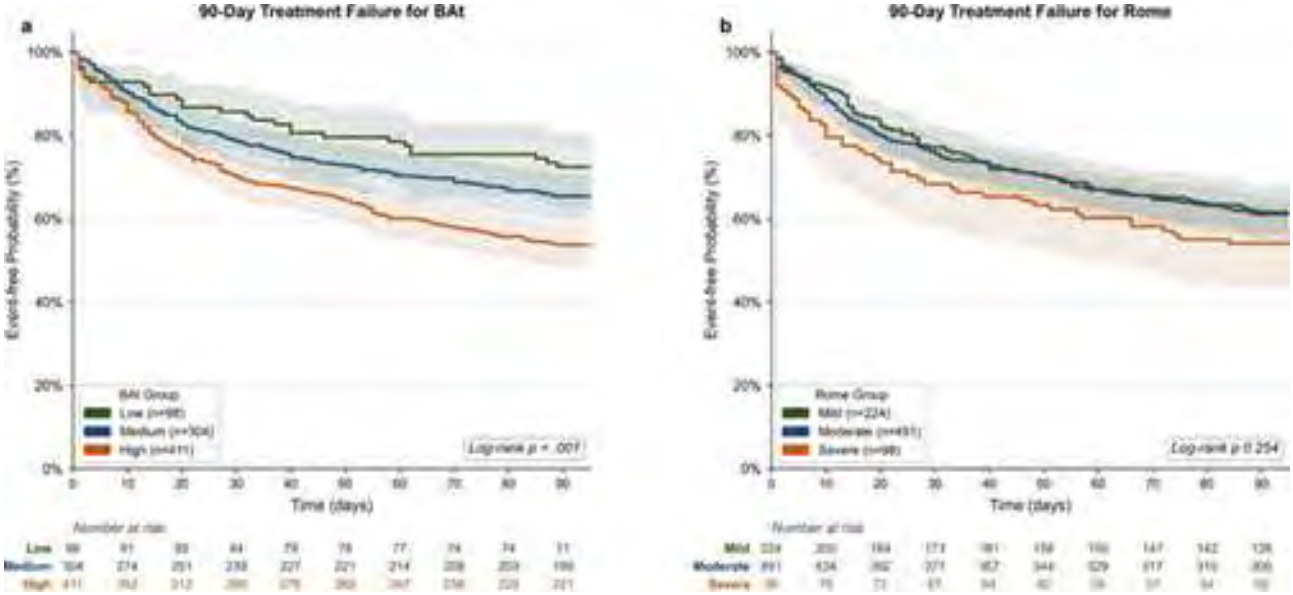
[1]A Beersaerts, [2,3]HWW Aung, [4]N Smallcombe, [4]E Dogu, [1]K Vermeersch, [1]P Moghames, [2,3]HJC McAuley, [2,3]CA Flynn, [2,3]CE Brightling, [2]S Everaerts, [4]S Cass, [4]REK Russell, [1,5]W Janssens, [2,3]NJ Greening, [4]M Bafadhel. *[1]Research group BREATHE, KU Leuven, Leuven, Belgium; [2]Institute for Lung Health, National Institute for Health and Care Research Leicester Biomedical Research Centre (Respiratory), Glenfield Hospital, Leicester, UK; [3]School of Medical Sciences, Division of Respiratory Sciences, University of Leicester, Leicester, UK; [4]King's Centre for Lung Health, School of Immunology and Microbial Sciences, King's College London, London, UK; [5]Department of Respiratory Diseases, UZ Leuven, Leuven, Belgium*

10.1136/thorax-2026-BTSabstracts.138

Introduction Acute exacerbations of COPD (AECOPD) are a major contributor to morbidity and mortality worldwide. However, clinically, current severity nomenclature continues to be largely healthcare utilisation based. The Baseline, Acuity and trigger (BAt) classification has been developed as a risk score (low-, medium- and high-risk groups) to predict clinical outcomes following hospitalised AECOPD.

Aims The BAt classification was evaluated for its ability to predict treatment failure -a composite of readmission and all-cause mortality- at 90 days in patients hospitalised with AECOPD, using data across three European hospital sites in Leuven (Belgium), Leicester and London (UK). Secondary outcomes included 365-day treatment failure, readmission and mortality at 90 and 365 days and treatment escalation. Performance was compared with the Rome severity scoring.

Results In 813 patients admitted to hospital with an AECOPD (n=374, 46% female; mean (SD) age 70 (10) years), there were 39.6% (n=322/813) and 66.7% (n=542/813) treatment failure events at 90- and 365- days respectively. Outcomes differed



Abstract S132 Figure 1 Time to treatment failure by BAt and Rome group within 90-days

significantly across BAt risk groups (log rank p<0.001), with 90-day time to treatment failure occurring with increasing hazard by BAt risk group (high vs low risk HR 1.83, 95% CI 1.22–2.75; p=0.004, figure 1). No significance in time to treatment failure was observed across Rome severity groups.

At 365 days, time to treatment failure remained significantly associated with BAt risk group (log rank p<0.001) with increasing hazard by BAt risk group (high vs low risk HR 1.65, 95% CI 1.23–2.22; p<0.001). BAt also discriminated 90-day readmission and was associated with greater treatment escalation compared to Rome. Both BAt and Rome were significantly associated with 90- and 365-day mortality in hospitalised exacerbations of COPD.

Conclusion BAt can predict the risk of treatment failure (readmission and/or mortality) following an AECOPD requiring hospital admission. As a comprehensive tool that integrates baseline severity with clinical acuity, it could be routinely used in clinical practice and clinical trial settings.

ILDS: ILAS and familial fibrosis

S133 RISK STRATIFICATION AND LONGITUDINAL ASSESSMENT OF INTERSTITIAL LUNG ABNORMALITIES USING E-LUNG QUANTITATIVE CT BIOMARKERS

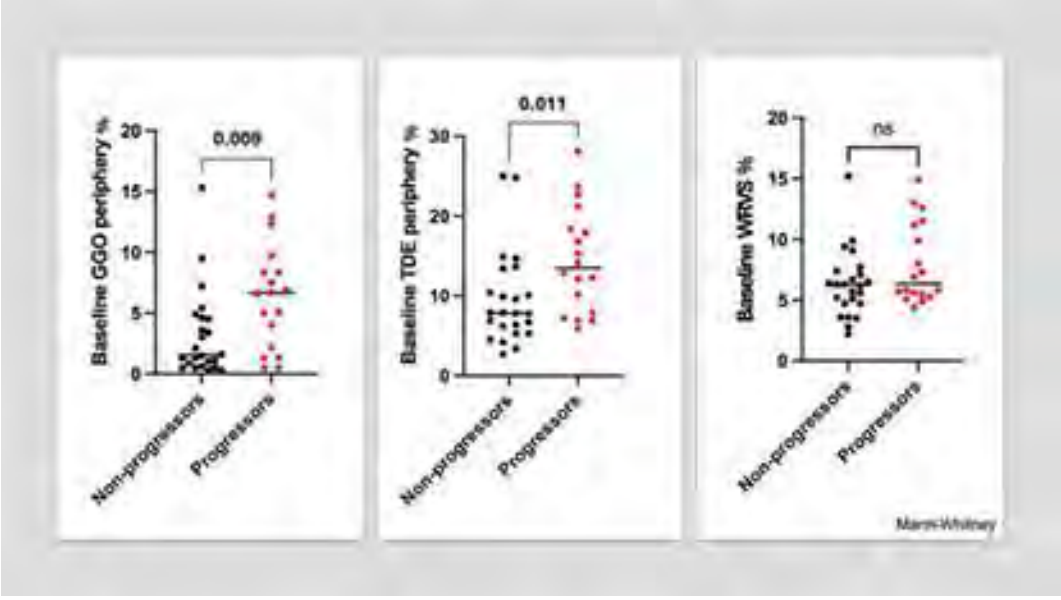
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10.1136/thorax-2026-BTSabstracts.139

Interstitial lung abnormalities (ILA) are increasingly identified as incidental findings on CT following lung cancer screening. A subset of ILAs will develop progressive fibrotic interstitial lung disease (ILD.) Accurately predicting which individuals progress remains a key unmet clinical need. We evaluated whether e-Lung (Brainomix), an AI quantitative CT (qCT) platform, could identify ILA progressors at baseline, or upon serial imaging.

Using an exploratory cohort of incidental ILAs referred to a tertiary ILD centre between 2018-2025, we retrospectively assessed qCT biomarkers in those who progressed to ILD compared with those who remained as ILA non-progressors. Progression was defined as: formal change in diagnosis to ILD following multidisciplinary meeting, documented progression to ILD in clinical correspondence, or clinician-initiated antifibrotics. e-Lung biomarkers included Weighted Reticulovascular Score (WRVS), peripheral ground-glass opacity (pGGO) and peripheral Total Disease Extent (pTDE), all defined as radiologic abnormalities in the peripheral 10mm of the lung. Baseline biomarkers were compared using non-parametric methods and univariate Cox regression. Linear mixed models were used to determine whether longitudinal biomarker trajectories differed between groups over time.

18/42 (43%) individuals with ILA progressed to ILD over a median of 3.88 years. 30/42 had ≥ 3 serial CTs. At baseline CT, progressors had significantly higher pGGO (6.7% vs 1.6%; p=0.009) and pTDE (13.5% vs 7.9%; p=0.011) than non-progressors; baseline WRVS did not significantly differ between groups (figure 1). In time-to-event analyses, all metrics were associated with time to progression. pGGO HR 1.13 per 1% increase, (1.03–1.25, p=0.010); WRVS HR 1.24 (1.06–1.45, p=0.009); and pTDE HR 1.09, (1.03–1.16, p=0.006). pTDE demonstrated the highest C-index (0.73). WRVS most clearly distinguished progressors from non-progressors longitudinally, increasing more rapidly in progressors (+0.6%/year vs



Abstract S133 Figure 1 Baseline quantitative CT-derived biomarkers in the peripheral 10 mm lung region using e-Lung in ILA progressors and non-progressors. Baseline peripheral ground-glass opacity, total disease extent, and weighted reticulovascular score were compared between ILA that progressed to ILD and non-progressors. Peripheral GGO and TDE were significantly higher in progressors, while WRVS did not differ significantly between groups. Abbreviations: CT, computed tomography; ILA, Interstitial lung abnormality; ILD, Interstitial Lung Disease; GGO, ground-glass opacity; TDE, total disease extent; WRVS, weighted reticulovascular score; ns, not significant

+0.05%/year; p<0.0001), while pTDE also rose faster in progressors (+1.1%/year vs +0.2%/year; p=0.0204.)

e-Lung distinguished ILA progressors from non-progressors with pTDE and pGGO emerging as promising baseline qCT predictors of progression, whilst WRVS trajectory best correlated with progression of fibrosis. e-Lung warrants further prospective investigation as a potential tool to predict ILA progression, and validation in a larger ILA cohort is warranted.

S134 MEDIASTINAL LYMPHADENOPATHY PREDICTS RADIOLOGICAL PROGRESSION OF INTERSTITIAL LUNG ABNORMALITIES (ILA) IN LUNG CANCER SCREENING PROGRAMME

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10.1136/thorax-2026-BTSabstracts.140

Background Lung Cancer Screening (LCS) programmes increasingly identify incidental interstitial lung abnormalities (ILA) on low-dose CT. However, progression is variable and difficult to predict. Previous studies have demonstrated that mediastinal lymphadenopathy (MLN) is associated with adverse outcomes in ILD cohorts. We investigated the prevalence of MLN in an LCS-derived ILA cohort and its relationship with radiological fibrosis progression.

Methods Single centre study of consecutive patients referred from a LCS programme to a tertiary ILD centre between August 2020 and August 2023 who had at least two computed tomography (CT) chest scans available for review. During the study period, referral criteria included ILA affecting ≥5% lung volume. Electronic patient records were reviewed. CTs were assessed by two independent radiologists for fibrosis progression (interval worsening of ILA), MLN (≥10 mm short axis) and fibrosis extent (>10%). Alternative causes of MLN were excluded. Risk factor analysis for time to progression was assessed by Cox proportional hazards regression.

Results 116 patients were included. Baseline demographics: mean (SD) age 70.8 years (5.4), sex (male) 58%, FVC% 96.1 (17.5), and TLCO% 69.7 (16.5). Median (IQR) time between CTs was 23 months (10–24). Radiological progression occurred in 47.4% (n=55). Baseline MLN ≥10 mm was present in 25% of patients. At baseline, progressors had a higher prevalence of MLN ≥10 mm (36% vs. 15%, p=0.01) and CT fibrosis >10%, as well as lower TLCO (67% vs. 72%, p=0.10), compared with non-progressors (table 1). In univariate analysis, MLN ≥10 mm and CT fibrosis >10% increased the risk of progression (HR 1.993, p=0.02 and HR 2.246, p=0.04, respectively; table 1). In multivariate Cox regression analysis (age, smoking pack-year history, fibrosis >10%, and TLCO%), baseline MLN ≥10 mm was a strong independent predictor of progression (HR 2.439, p=0.010).

Conclusion In this LCS-ILA cohort, baseline MLN≥ 10mm independently predicted fibrosis progression. This observation could aid clinicians identifying patients requiring closer surveillance and earlier intervention. External validation of these findings is warranted.

S135 CHARACTERISATION OF INTERSTITIAL LUNG ABNORMALITIES IN A UK MULTICENTRE ILA CLINIC COHORT: EARLY FINDINGS FROM THE ILARC-UK PILOT

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Introduction Interstitial lung abnormalities (ILAs) are non-dependent parenchymal abnormalities identified on CT imaging which, in a subset of individuals, represent a pre-clinical stage of interstitial lung disease. The Fleischner Society Position paper described three radiological subtypes of ILA, including fibrotic and non-fibrotic patterns, which have differing prognostic implications. The ILARC-UK consortium is piloting a standardised model of care through dedicated ILA clinics across

Abstract S134 Table 1 Baseline Demographics Progression vs. Non-Progression of Interstitial lung abnormality and Cox Univariate analysis for CT Progression

Variable (at baseline)	Demographics (Mean (SD)/% (n))			Cox - Univariate Analysis (CT Progression Fibrosis)	
	Progressors (n=55)	Non-Progressors (n=61)	p	Hazard Ratio (95% CI)	p
Age	70.9 (4.7)	70.8 (5.9)	0.49	0.623 (0.94-1.04)	0.62
Sex (male)	48% (32)	52% (35)	0.93	0.795 (0.46-1.38)	0.41
Smoking (PYH)	41.5 (30.3)	37.8 (16.2)	0.45	0.418 (0.99-1.02)	0.42
FVC% predicted	94.5 (2.5)	97.5 (2.2)	0.19	0.995 (0.98-1.01)	0.53
TLCO% predicted	66.7 (16.8)	72.2 (15.9)	0.10	0.987 (0.97-1.01)	0.15
CT Fibrosis extent (>10%)	16% (9)	5% (3)	0.04*	2.246 (1.047-4.817)	0.04
MLN≥10mm	36% (20)	15% (9)	0.01*	1.993 (1.13-3.52)	0.02
Gasto-oesophageal reflux	43% (24)	44% (27)	0.94	0.878 (0.506-1.523)	0.64

PYH= pack year history, FVC=Forced vital capacity, TLCO transfer co-efficient for carbon monoxide, MLN = mediastinal lymphadenopathy

multiple UK centres to improve phenotyping, risk stratification, and longitudinal follow-up. We report early findings from this multicentre cohort.

Methods Patients referred to dedicated ILA clinics across five UK NHS tertiary centres between August 2023 and November 2025 were prospectively assessed. Clinical, demographic, radiological, and physiological data were collected using a standardised protocol. Baseline characteristics and comorbidities were compared across ILA subgroups using parametric and non-parametric statistical tests.

Results A total of 266 individuals with ILAs were included, comprising 122 (45.9%) subpleural fibrotic ILAs, 134 (50.4%) subpleural non-fibrotic ILAs, and 10 (3.7%) non-subpleural non-fibrotic ILAs. ILAs were most commonly identified during evaluation of respiratory symptoms (51.1%), followed by incidental detection during investigation of non-respiratory symptoms (26.3%) and lung cancer screening (22.6%). Patients with fibrotic ILAs were older than those with non-fibrotic ILAs (73 ± 6.8 vs 69 ± 8.8 years, $p<0.0001$) and had higher rates of smoking exposure (69.7% vs 59.5%). There was no significant difference in FVC between groups, however TLCO was significantly reduced in subpleural fibrotic ILAs compared to non-fibrotic; 71.5 ± 18.2 vs. 77.9 ± 18.8 ($p=0.0094$). Cardiovascular comorbidity was common across the cohort; ischaemic heart disease was significantly more prevalent in fibrotic ILAs compared with non-fibrotic ILAs (OR 2.32, 95% CI 1.19–4.76; $p=0.017$).

Conclusion In this multicentre UK ILA clinic cohort, subpleural fibrotic ILAs are associated with older age, increased smoking exposure, lower baseline gas transfer and higher burden of ischaemic heart disease, compared with non-fibrotic ILAs. These findings highlight clinically meaningful differences between radiological subtypes and support the role of dedicated ILA clinics in enabling systematic characterisation and risk stratification. Continued longitudinal follow-up will be essential to determine progression rates and prognostic implications across ILA subtypes within the ILARC-UK framework.

S136 CLINICAL CHARACTERISTICS ASSOCIATED WITH PROGRESSION OF INTERSTITIAL LUNG ABNORMALITIES TO INTERSTITIAL LUNG DISEASE: A SINGLE-CENTRE COHORT STUDY

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Background Interstitial lung abnormalities (ILA) are subclinical CT findings increasingly identified through lung cancer screening and investigation of respiratory or cardiac symptoms. Although a proportion of patients progress to interstitial lung disease (ILD), predictors of progression remain poorly defined, limiting risk stratification, surveillance, and opportunities for early intervention. We aimed to determine whether baseline demographic, physiological, and socioeconomic factors were associated with progression to ILD.

Methods We conducted a retrospective cohort study of patients with ILA referred to our tertiary centre between 2019 and 2025. Patients with a diagnosis of ILD at baseline or fewer than two clinic visits were excluded. Progression to ILD was defined as a documented change in diagnosis following multidisciplinary team review integrating symptoms, CT findings, and pulmonary function trends, or initiation of antifibrotic therapy. Baseline demographics, physiological parameters, comorbidities, and socioeconomic deprivation measured using the Index of Multiple Deprivation (IMD) decile were compared between progressors and non-progressors. Continuous variables were analysed using non-parametric tests, while comorbidities and ethnicity were summarised descriptively.

Results Nineteen of 50 patients with ILA (38%) progressed to ILD during follow-up, with a median time to progression of 39.8 months (IQR 22.3–55.9). There were no significant differences between progressors and non-progressors in age, smoking status, family history of ILD, or autoimmune serology. Baseline FVC was similar between groups; however, TLCO was significantly lower in progressors than non-progressors (70% vs 106% predicted, $p<0.0001$). Subpleural fibrotic ILA were more common among progressors than non-progressors (63% vs. 37%). Hypertension was more frequent in progressors (42% vs 19%). Socioeconomic deprivation did not differ significantly between groups (IMD decile 6 vs 7, $p=0.64$). The cohort was predominantly White (86%), precluding ethnic subgroup analysis.

Conclusion More than one-third of patients with ILA progressed to ILD during follow-up. Reduced baseline TLCO was the principal feature distinguishing progressors from non-progressors, suggesting that impaired gas transfer may identify individuals at increased risk of progression. The higher prevalence of hypertension among progressors warrants further investigation. The limited prognostic utility of conventional demographic and clinical characteristics underscores the need for improved biomarkers and prospective longitudinal studies to enhance risk stratification in ILA.

S137 AN OVERVIEW OF THE CLINICAL CHARACTERISTICS OF FAMILIAL PULMONARY FIBROSIS PATIENTS IN THE UK: DATA FROM THE BRITISH THORACIC SOCIETY ILD REGISTRY 2013-2026

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Background Interstitial lung diseases (ILDs) are a heterogeneous group of disorders with variable prognosis. Family history is one of the strongest risk factors. Family history or an unexplained diagnosis of a fibrotic-ILD before age 50 may suggest an

underlying genetic contribution and a more progressive phenotype. Up to 40% of familial cases are thought to have a genetic component, including telomere-related genes. Knowledge in this area is rapidly evolving.

Aims To assess the baseline demographics and clinical characteristics among familial ILD patients in comparison to those with no family history in the UK.

Methods Data from the BTS UK ILD Registry (January 2013-April 2026; table 1a) were analysed. Data were available for 11027 patients and screened for documentation of family history. 7331 patients were included in the study, and 3696 were excluded due to blank or inconclusive family history data. Family history was self-reported and included first and/or second-degree relatives.

Results 564 (7.7%) patients were documented to have a relative with ILD. Familial patients were younger (70 y/o vs 74 y/o, p<0.001) and less likely to be male (61% vs 74%, p<0.001) than those with no family history. Interestingly, a lower proportion of patients with a family history were diagnosed with IPF than in the non-familial group (79% vs 83.4%, p=0.012), despite similar baseline FVC, DLCO, and radiological patterns. Smoking rates were similar in both groups. Antifibrotic prescription rates were similar between the familial and non-familial ILD groups.

Abstract S137 Table 1a Baseline characteristics of patients with familial ILD compared to those with sporadic ILD. Data from the British Thoracic Society ILD Registry 2013-2026[#]

Baseline characteristics	Family history of ILD N=564	No family history of ILD N=6,767	P value
Demographic			
Age in years, median (IQR)	70 (63-76)	74 (67-79)	<0.001
Male, n (%)	354 (60.9)	5,101 (73.8)	<0.001
Body mass index in Kg/m^2, median (IQR)	28.6 (25.4-32.0)	28.3 (25.2-31.6)	0.284
Clinical			
Smoking history, n (%)			0.457
Never	180 (36.6)	1,953 (34.2)	
Ex-smoker	291 (59.1)	3,545 (62.0)	
Current	21 (4.3)	221 (3.9)	
Diagnosis			
Idiopathic Pulmonary Fibrosis (IPF), n (%)	390 (79.0)	4,607 (83.4)	0.012
Radiological pattern*			
Alternative pattern/ Indeterminate UIP, n (%)	7 (2.0)	103 (2.6)	0.589
Probable UIP, n (%)	191 (55.7)	2131 (53.1)	
Definite UIP, n (%)	145 (42.3)	1781 (44.4)	
Lung function*			
FVC - percent predicted, median (IQR)	79.0 (68.1-94.2)	78.1 (67.2-91.8)	0.372
DLCO - percent predicted, median (IQR)	52.2 (42.8-64.0)	51.0 (39.8-64.4)	0.240
Pharmacological treatment			
Antifibrotic treatment, n (%)	253 (55.2)	2,742 (52.6)	0.280

Legend: ILD=interstitial lung disease; IQR=interquartile range; UIP=usual interstitial pneumonia; FVC=forced vital capacity; DLCO=diffusing capacity of the lungs for carbon monoxide. Excluded cases: occupational lung diseases, drug-induced ILD.
*Lung function and radiological pattern data are available in a subset of patients.
[#]Cut-off date: 24th April 2026.

Abstract S37 Table 1b Secondary analysis: Clinical comparison between patients with sporadic ILD diagnosed before age 50 and familial ILD of all ages. Data from the British Thoracic Society ILD Registry 2013-2026[#]

Baseline characteristics	Sporadic ILD <50y/o N=323	Familial ILD All ages N=564	P value
Idiopathic Pulmonary Fibrosis (IPF), n (%)	25 (23.8)	390 (79.0)	<0.001
Lung function*: FVC - percent predicted, median (IQR)	91.1 (71.0-104.7)	79.0 (68.1-94.2)	<0.001
Lung function*: DLCO - percent predicted, median (IQR)	78.7 (64.1-91.2)	52.2 (42.8-64.0)	<0.001
Pharmacological treatment: Antifibrotic treatment, n (%)	21 (24.1)	253 (55.2)	<0.001

*Lung function data available in a subset of patients. [#]Cut-off date: 24th April 2026. Legend: ILD=interstitial lung disease; FVC=forced vital capacity; IQR=interquartile range; DLCO=diffusing capacity of the lungs for carbon monoxide. Familial ILD definition: at least one first- or second-degree relative with ILD.

Secondary analysis of 323 patients with familial ILD (including sarcoidosis) diagnosed before age 50 showed a distinct phenotype compared to the whole familial group, with significantly higher baseline FVC (91% vs 79%) and DLCO (78.7% vs 52.2%), lower rate of IPF diagnosis (23.8% vs 79%), and lower antifibrotic use (24.1% vs 55.2%) (table 1b).

Conclusion Familial patients represent approximately 8% of the BTS registry population. A detailed family history is essential in ILD, helping to identify and screen those at risk of an underlying genetic risk, precipitating appropriate genetic screening in a specialised centre. Familial patients may have distinctive features that warrant personalised diagnosis and management.

S138 TELOMERE LENGTH ANALYSIS AND NEXT GENERATION SEQUENCING RESULTS IN PATIENTS WITH FIBROTIC INTERSTITIAL LUNG DISEASE

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Background Telomere biology disorders are a heterogeneous group of diseases, with pulmonary fibrosis being the most common in adults. Short telomere length (TL) is associated with a poorer prognosis in interstitial lung disease (ILD), can indicate the presence of a telomere-related gene (TRG) variant, and may influence clinical care decisions such as immunosuppression.

Antifibrotic therapies slow disease progression in progressive pulmonary fibrosis and idiopathic pulmonary fibrosis (IPF); however, lung transplant remains the only cure. New ISHLT guidelines recommend TL measurement for all ILD patients referred for transplant.

Aims To determine the prevalence of short TL in ILD and assess its association with TRG variants.
Methods Patients were recruited to Precise-PF, a prospective observational cohort study. Individuals with both TL measurement and next-generation sequencing (NGS) panel results were included. Recruitment occurred at a national referral clinic for familial pulmonary fibrosis (FPF). TL was measured using high-throughput single telomere length analysis (HT-STELA). Short TL was defined as TL <10th age-adjusted percentile. Clinical data includes age, sex, comorbidities and PFTs.
Results 203 participants were included: 91 FPF, 59 IPF and 53 systemic autoimmune rheumatic disease-associated ILD (SARD-ILD). Short TL was identified in 46 (22.6%) participants: 24(26.4%) FPF, 12(20.3%) IPF, and 10(18.9%) SARD-ILD. In the FPF cohort, 11(12.1%) had a likely pathogenic or pathogenic TRG variant, and 4(4.4%) had a variant of unknown significance (VUS). 8(13.6%) IPF and 2(3.4%) SARD-ILD participants had a VUS. There was a significant association between the presence of a TRG variant and short TL (p=0.045). However, 36(74%) of those with short TL had no identifiable TRG variant; in the familial cohort, and 10(55%) had a TL ≥10th percentile.
Conclusion We show that short telomeres were present in greater than 1 in 5 patients with FPF, IPF and SARD-ILD; however, most cases could not be explained by TRG variants. A recent ISHLT statement on TL testing in patients pre-lung transplant will likely increase measurement, but our data suggest that much remains unknown about the causes and implications of short telomeres. Further study and implementation strategies for subsequent genetic testing and risk assessment need to be developed.

Pulmonary vasculature: aims and imaging

S139 GENETIC ASSOCIATIONS OF RARE DISEASE COMPLICATIONS - BRAIN ABSCESS AND MIGRAINE IN PATIENTS WITH PULMONARY ARTERIOVENOUS MALFORMATIONS

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Introduction Pulmonary arteriovenous malformations (PAVMs) increase risks of brain abscess which is treated by emergency neurosurgery, and migraines as shown by published improvement in prevalence rates after complete obliteration of PAVMs, usually by embolisation.[1] Pre-2017 data identified dental bacteraemias and high iron as risk factors for brain abscess,[2] and dietary factors as risk for migraines. The current study objectives were to identify risk-modifying genes, and changes in expression of risk-modifying genes to potentially personalise advice and better understand mechanisms and preventative options.
Methods PAVMs and the often-associated hereditary haemorrhagic telangiectasia (HHT) were separately nominated as recruiting diseases for the 100,000 Genomes Project, specifying 'brain abscess' and 'migraine' in the original 2015 Data Models. Participants with these phenotypes were identified by custom Python scripts extracting standardised ICD10 and HPO diagnostic codes from linked NHS records within the Genomics England Research Environment. Rates were validated by a pseudonymised, database of routinely-captured NHS data for patients with radiologically-confirmed PAVMs in an institutional Rare Disease database. For 100,000 Genomes Project participants, genome-wide association (migraine), and aggregate variant analysis using SAIGE-GENE+ (both phenotypes) were performed, focussing on endothelial function, blood-brain barrier integrity, and immune function gene variants
Results The 100,000 Genomes Project cohort was confirmed as representative of the wider population by similar rates of brain abscess (8/93 [0.028, 0.14] and reported migraine (19/93 [0.12, 0.28]) to 1,149 institutional database patients (107/1,149 [0.08,0.11] brain abscess; 291/1,149 [0.23, 0.28] migraines), p-values >0.99 Fisher's exact test. No DNA variants reached genome-wide significance for migraine. AVT revealed a significant association between *TJP3* loss-of-function gene variants and migraine (p=0.005), with minor allele frequencies 0.19 (cases), 0.02 (controls, p=0.001). For brain abscess, SAIGE-GENE+ indicated potential associations with loss-of-function variants in *TJP3*, *NOD2*, and *SELE*. All three genes were expressed in PBMCs, two in endothelial cells, with no change in *THP3* or *NOD2* expression, but increased endothelial *SELE* expression after FSL-1 (bacterial) or iron treatments.
Conclusion This study identifies a candidate gene for migraine susceptibility and suggests potential protective effects of loss-of-function *TJP3* and *NOD2* variants against brain abscess.

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S140 ISCHAEMIC STROKE IN PATIENTS WITH PULMONARY ARTERIOVENOUS MALFORMATIONS: NEW ASSOCIATIONS WITH FRACTIONAL EXHALED NITRIC OXIDE AND ATRIAL FIBRILLATION

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Introduction and Objectives Pulmonary arteriovenous malformations (PAVMs) are abnormal blood vessels in the lungs providing a direct connection between pulmonary arteries and veins. PAVM patients' increased risk of ischaemic stroke is attributed to systemic venous blood bypassing pulmonary capillaries, reducing filtration of small emboli. Routine care includes pulmonary function and measurement of fractional exhaled nitric oxide (FeNO) to guide management of any concurrent airflow obstruction. Hypothesising that NO could act on PAVMs to further reduce filtering capability, this study investigated risk factors for ischaemic stroke in PAVM patients.

Methods Between February-April 2026, additional data from pulmonary function records were added to an institutional Rare Disease database that had been constructed with ethical approval (21/SW/0120), to hold deidentified, routinely-captured NHS data of all patients with radiologically-confirmed PAVMs. Following PAVM Project access approval by the January 2026 Data Access and Prioritisation Committee Meeting, pseudonymised data were examined for patients with radiologically-confirmed PAVMs and at least one FeNO measurement.
Results 27 (8.3%) of the 350 patients analysed suffered ischaemic stroke. Conventional risk factors such as smoking, hypertension, diabetes, hypercholesterolaemia and BMI were no different between stroke and non-stroke patients. Serum iron levels were lower in stroke patients (medians: 8.0μmol/L vs 14.0μmol/L, p_{adj}=0.020). Age and sex-adjusted logistic regression identified serum iron (odds ratio (OR)=0.92), FeNO (OR=0.95), and atrial fibrillation (AF; OR=3.85) as significant predictors of ischaemic stroke. Patients were separated by AF status for subgroup analyses. In non-AF patients, serum iron was lower in stroke patients (medians: 10μmol/L vs 14μmol/L, p_{adj}=0.090). In AF patients, differences between stroke and non-stroke patients were significant for FeNO (medians: 8ppb vs 22.5ppb, p_{adj}=0.090), haemoglobin (114g/L vs 155g/L, p_{adj}=0.090) and fibrinogen (4.6g/L vs 2.3g/L, p_{adj}=0.090).
Conclusion This real-world study validated previous research: low serum iron increases the risk of PAVM stroke while many conventional risk factors do not. Different risk factors for AF and non-AF patients support different ischaemic stroke aetiologies in patients with PAVMs who do and do not have AF. AF-related risk factors identified in this population such as FeNO might represent an ischaemic stroke risk factor in the wider AF population, and further investigation is suggested.

S141 TARGETING 5-HT TO PREVENT ISCHAEMIC STROKE DUE TO UNTREATABLE PULMONARY ARTERIOVENOUS MALFORMATIONS WITHOUT RISKING HAEMORRHAGE FROM CONVENTIONAL ANTI-PLATELET AGENTS

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Background Pulmonary arteriovenous malformations (PAVMs) are a rare disease estimated to affect 3 million people worldwide. Only a proportion have access to interventional treatments. PAVMs prevent pulmonary capillary filtration of bloodborne platelet emboli, causing ischaemic stroke. Stroke risk is higher in iron deficiency which augments platelet aggregation to 5-HT.[1] Usual antiplatelet agents can precipitate bleeds that aggravate iron deficiency and may prevent use, especially where PAVMs are associated with underlying hereditary haemorrhagic telangiectasia (HHT). In 2025, the 5-HT2A receptor antagonist, and antiplatelet agent Sarpogrelate demonstrated equivalent efficacy in ischaemic stroke prevention compared to aspirin in high-risk patients with diabetes in South East Asia, without haemorrhagic side-effects.[2] Sarpogrelate is not marketed in Europe or the US.
Methods To define levels of unmet need, we generated a pseudonymised Rare Disease database with automated feeds from routine clinical care. To facilitate personalisation to augment trial outcomes, we established gene variant burdens relevant to 5-HT levels/activity and proteomics-compatible biosampling methods, delivering a dynamic platform for gene expression in patient-derived blood cells. Alternate peripherally-selective 5-HT2A receptor antagonists were sought.
Results By 09/04/2026, of 1,149 consecutive patients with imaging-proven PAVMs (median age 47y; 735 [95% CI 0.61,0.67] female), 125/1149 [0.09, 0.13] had an ischemic stroke but 47/1,149 [95% CI 0.02, 0.04] had massive haemoptysis, haemothorax or brain haemorrhage that were fatal or required emergency treatment. Following maximal interventions that improved oxygenation and symptoms where present, 627 [0.52,0.57] patients had residual PAVMs, but only 51/109 [0.37, 0.56] ischaemic stroke patients received antiplatelet treatments for secondary prevention. Reasons included local professional withholding due to perceived bleeding risk, increased mucosal bleeding, and patient preference. Genes influencing 5-HT pathways/receptors were inactivated no more commonly in PAVM/HHT patients than in the general population. Functional studies in freshly-derived platelets demonstrated comparable expression of 5-HT2A receptor and platelet genes. Naftidrofuryl oxalate was identified as a well-tolerated, NICE-approved treatment for peripheral vascular disease where symptomatic benefits for ischaemic symptoms (intermittent claudication) had been ascribed to vasodilation, overlooking 5-HT's role in platelet aggregation.
Conclusions We propose a real-world trial of naftidrofuryl oxalate for patients with PAVMs.

S142 RETHINKING HYPOXAEMIA IN PULMONARY ARTERIOVENOUS MALFORMATIONS: ASSOCIATIONS WITH PULMONARY ARTERY PRESSURE AND GAS TRANSFER

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Background Pulmonary arteriovenous malformations (PAVMs) cause arterial hypoxaemia because blood bypasses the pulmonary capillary bed through right-to-left shunts, impairing gas exchange. Hypoxaemia therefore occurs in the absence of alveolar hypoxia. Although the 2017 British Thoracic Society Clinical Statement does not recommend long-term oxygen therapy as prophylaxis against pulmonary hypertension,[1] in current UK respiratory practice, some patients receive long-term oxygen prescribed for this indication. This suggested that either new data had been published or previous data/guidance was being overlooked.
Methods Post-2017 publications were identified through PubMed to establish whether new evidence supported the apparent change in practice. Between February-April 2026, pulmonary function measurements including spirometry and the carbon monoxide transfer coefficient K_{CO} were added to an institutional Rare Disease database constructed with ethical

approval (21/SW/0120), to hold deidentified, routinely captured NHS data of all patients with radiologically confirmed PAVMs. Data included standing serial oxygen saturation (SaO_2) measurements that are a validated biomarker for right-to-left shunting and pulmonary artery pressure (PAP) measurements at angiography immediately before PAVM treatment by embolisation. PAVM Project access approval was by the January 2026 Data Access and Prioritisation Committee Meeting. Pseudonymised data on patients with at least one recorded PAP measurement were examined.

Results No new data publications were identified examining oxygenation and PAP in patients with PAVMs. Of 793 recorded PAP measurements, pre-embolisation, 715 (90.2%) were below the pulmonary hypertension threshold of mean PAP>20mmHg. Oxygen saturation (SaO_2) ranged from 61-100%. Lower SaO_2 was not associated with higher mPAP/pulmonary hypertension. Instead, SaO_2 showed a weak positive association with mPAP (Spearman's rho=0.103, p=0.002). In multivariate linear regression, higher SaO_2 remained associated with higher log(mPAP) after adjustment for age and sex. The model was statistically significant and explained 12% of variation in log(mPAP) (n=661, R^2=0.12, p<0.0001) The K_{CO} was reduced where SaO_2 was lower (regression coefficient β=0.85 [95% CI 0.61, 1.08], p<0.0001).

Conclusion PAVM-related hypoxaemia is not associated with elevated mPAP. The data reinforce that shunt-related hypoxaemia is independent of alveolar hypoxia and should not lead to concerns about later development of hypoxic pulmonary hypertension.

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S143 USING HYPERPOLARISED 129XENON AND PROTON MRI TO DETECT IMPAIRED GAS EXCHANGE IN STRUCTURALLY NORMAL LUNG REGIONS IN SYSTEMIC SCLEROSIS PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION

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10.1136/thorax-2026-BTSabstracts.149

Introduction Pulmonary arterial hypertension (PAH) is a leading cause of mortality in systemic sclerosis (SSc). Conventional imaging cannot reliably detect the vascular gas-exchange dysfunction that precedes or co-exists with parenchymal lung disease in SSc. Ultra-short echo time (UTE) MRI enables structural imaging of the lung parenchyma. Hyperpolarised 129Xe MRI provides regional gas-exchange metrics by quantifying the 129Xe signal in the alveolar gas (GAS), membrane (M), and red blood cell (RBC) compartments. We hypothesised that, by restricting analysis to lung regions appearing structurally normal on UTE MRI, any gas-exchange impairment detected in SSc-PAH would reflect vascular rather than parenchymal dysfunction.

Methods Patients with SSc and suspected PAH were prospectively recruited; those with extensive ILD (>20% on CT) were excluded. Patients underwent right heart catheterisation, UTE MRI, and hyperpolarised dissolved-phase 129Xe-MRI at 1.5T. UTE images were normalised, and a threshold based on healthy volunteer data was applied to segment regions of normal and abnormal signal intensity. A co-registration pipeline was applied to calculate 129Xe gas-exchange metrics (RBC:M, M:GAS, and RBC:GAS) within normal UTE regions.

Results Thirty-six patients were included (SSc-noPAH n=14; SSc-PAH n=22); mean age was 67±7.7 years in SSc-noPAH and 64±6.7 years in SSc-PAH cohorts, with 34 (94%) females overall. Within regions of normal UTE MRI signal intensity, patients with confirmed SSc-PAH demonstrated significant impairment in gas-exchange compared with SSc patients without PAH. Median RBC:M was significantly lower in the SSc-PAH cohort (0.111 (0.081-0.180) vs 0.22 (0.17-0.25), p=0.001, Mann-Whitney test). Similarly, RBC:GAS was significantly lower in those diagnosed with SSc-PAH (0.0014 (0.00095-0.0019) vs 0.0020 (0.0015-0.0023), p=0.026). Conversely, M:GAS was significantly elevated in SSc-PAH patients (0.0111 (0.0100-0.0137) vs 0.0095 (0.0081-0.0104), p=0.005).

Conclusions In SSc-PAH, hyperpolarised 129Xe-MRI detected impaired gas-exchange in lung regions that appeared structurally normal on UTE MRI. These findings suggest that combined UTE and 129Xe-MRI may help differentiate vascular dysfunction from parenchymal pathology, with potential utility in monitoring of SSc and diagnosis of SSc-PAH.

S144 ANTHROPOMETRY, SEX AND AGE AT DIAGNOSIS INFLUENCE AI-DERIVED PULMONARY BLOOD VOLUMES FROM CT PULMONARY ANGIOGRAPHY IN PULMONARY HYPERTENSION

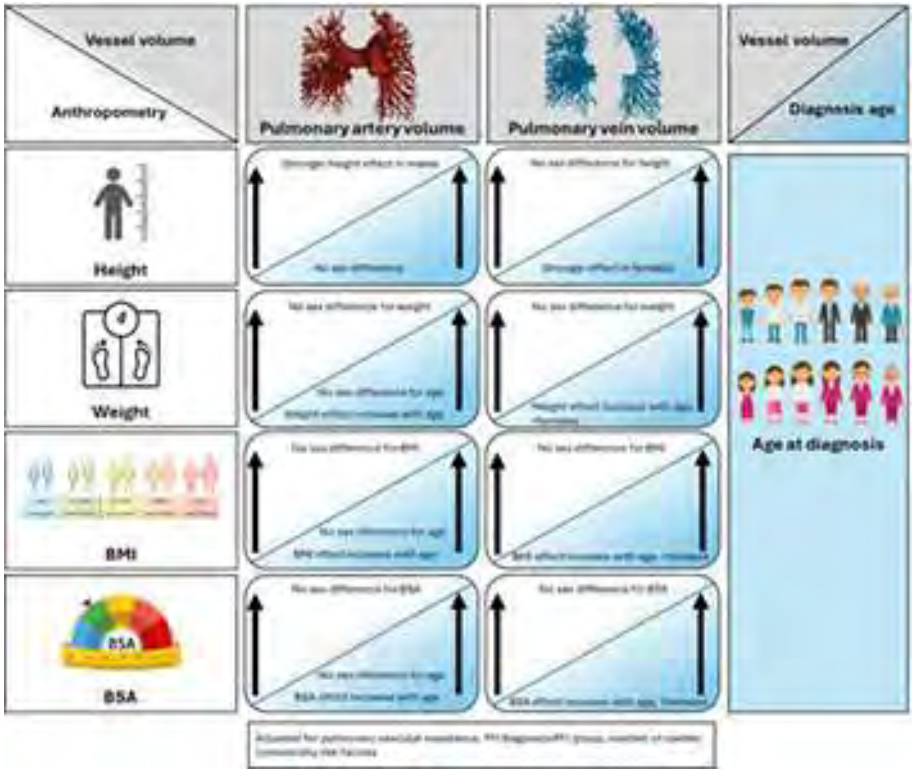
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10.1136/thorax-2026-BTSabstracts.150

Introduction Artificial intelligence (AI) enables automated quantification of pulmonary blood volumes (PBV) from CT pulmonary angiography (CTPA), offering potential imaging biomarkers for pulmonary hypertension (PH). However, the influence of biological characteristics on these metrics and their impact on prediction models remain unclear. We investigated the effects of anthropometrics, sex, and diagnosis age on AI-derived PBV, assessed whether normalization reduced sex-related PBV differences, and evaluated the incremental value of anthropometrics in predicting pulmonary vascular resistance (PVR) and cardiac output (CO).

Methods An AI-based segmentation model quantified pulmonary artery and vein volumes in a secondary CTPA analysis from the Cambridge PH Registry. Multivariable regression examined relationships between PBV and demographic factors, accounting for PH diagnostic group, PVR, and cardiac comorbidity burden. Associations between PBV and invasively measured PVR and CO were evaluated, assessing the incremental predictive value of anthropometric variables.

Results 376 patients were included: 120 pulmonary arterial hypertension, 30 group 2 PH, 79 group 3 PH, 102 chronic thromboembolic PH, and 45 without PH. Pulmonary artery volume increased with height, weight, body mass index (BMI), and body surface area (BSA) (all $p < 0.001$), with a stronger height-related effect in males. Older age at diagnosis was associated with larger pulmonary artery volume, especially in patients with higher weight, BMI, and BSA (all $p < 0.001$). Pulmonary vein volume also increased with anthropometrics. Older diag-



Abstract S144 Figure 1

nosis age disproportionately increased pulmonary vein volumes in females with higher weight, BMI, and BSA. Sex-related differences in PBV were substantially reduced following normalization to height, weight, or BSA (all $p < 0.001$).

Larger pulmonary artery volume and lower pulmonary vein volume were independently associated with higher PVR ($p < 0.001$). Larger pulmonary vein volume was independently associated with higher CO ($p < 0.001$). Inclusion of anthropometrics, particularly BSA, significantly improved prediction of PVR ($F = 12.8$, $p < 0.001$) and CO ($F = 23.8$, $p < 0.001$).

Conclusion AI-derived CTPA PBV are strongly influenced by anthropometrics, sex, and diagnosis age. Inclusion of anthropometric measures as covariates improves haemodynamic prediction from PBV. Anthropometric adjustment should therefore be incorporated into future AI-derived pulmonary vascular biomarkers for PH prediction and phenotyping.

From defence to damage: the phagocyte

S145 PLEURAL FLUID MULTI-OMICS REVEALS HETEROGENEOUS NEUTROPHIL POPULATIONS IN PLEURAL INFECTION

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10.1136/thorax-2026-BTSabstracts.151

Background Pleural infection is a severe and complex disease with a rising incidence worldwide. Previous studies have demonstrated substantial heterogeneity in disease biology, particularly in neutrophil activation states. However, knowledge of the immune response to pleural infection remains poorly understood.

Aim To deeply characterise the immune response and investigate its association with clinical outcomes.

Methods Paired whole blood and pleural fluid samples were collected from patients with pleural infection (n=4). Samples were analysed using cellular indexing of transcriptomes and epitopes by sequencing (CITE-seq). An independent validation cohort comprising pleural infection patients (n=13) and healthy controls (n=4) underwent bulk RNA sequencing.

Results Single-cell analysis identified distinct neutrophil subsets, including an immature population confined to blood, an activated population enriched in pleural fluid, and a mature population present in both compartments. Pleural fluid neutrophils exhibited more transcriptional and compositional changes compared to circulating blood neutrophils. A hypoxia-associated neutrophil subset, characterised by high expression of HIF1A and glycolysis-related genes, was exclusively detected in pleural fluid. Trajectory analysis indicated that these hypoxia neutrophils represent an intermediate cellular state during neutrophil differentiation within the pleural cavity. Their transcriptional reprogramming may therefore be critical for transitions to other neutrophil phenotypes. Bulk RNA sequencing deconvolution independently validated the preferential distribution of neutrophil subsets between blood and pleural fluid. Consistently, hypoxia-related gene signatures were significantly enriched in pleural fluid samples.

Conclusion This study diverse subsets of neutrophils in pleural infection. A distinct hypoxia intermediate neutrophil subset may play a key role in neutrophil fate transitions.

S146 ACUTE SYSTEMIC HYPOXIA INCREASES CIRCULATING NEUTROPHIL ACTIVATION AND EXTRAVASATION POTENTIAL

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10.1136/thorax-2026-BTSabstracts.152

Background Neutrophils are essential for pathogen control in respiratory infection. Hypoxia alters neutrophil function *in vitro*: most studies show increased phagocytosis but reactive oxygen species (ROS) production is variable. Patients with respiratory infection are often hypoxic but the impact of systemic hypoxia on neutrophil function is less certain. We evaluated the short-term systemic hypoxia effect on neutrophil phagocytosis, ROS generation and cell surface expression of neutrophil migration (CXCR2), adhesion (ICAM1), activation (CD11b) and pathogen recognition (CD16) markers.

Methods Healthy volunteers were exposed to 13kPa O_2 for 2 hours. Venous blood was taken pre- and post-exposure (without participant re-oxygenation). Neutrophil immunomagnetic isolation and assays were performed under 5% O_2, maintaining venous oxygen tension. Phagocytosis of *Escherichia coli* (5μg/ml) (n=6) and *Staphylococcus aureus* (10μg/ml) (n=3), intracellular ROS production (CellROX, 500nM) (n=5), and expression of CXCR2, ICAM1, CD11b and CD16 (n=5-12) were assessed by flow cytometry in neutrophils treated with/without TNF (20ng/ml).

Results Mean minimum peripheral oxygen saturation was 77.7 +/- 3.1%. There was a significant hypoxia-dependent 1.3-fold increase in neutrophil CXCR2 (unstimulated p=0.003; TNF-treated p=0.016) and 1.2-fold increase ICAM1 (unstimulated p=0.001; TNF-treated p=0.002) expression, suggesting enhanced extravasation ability. CD11b (1.3-fold) and CD16 (1.1-fold) were similarly increased by hypoxia in both unstimulated (CD11b p=0.022; CD16 p=0.28) and TNF-treated (CD11b p=0.002; CD16 p=0.006) neutrophils, indicating enhanced activation. This translated to a small but non-significant hypoxia-dependent increase in ROS generation (4 of 5 donors), and phagocytosis of *E. coli* (4 of 6 donors) and *S. aureus* (2 of 3 donors) in unstimulated neutrophils. Phagocytosis and ROS responses in TNF-treated neutrophils were more variable.

Discussion Acute systemic hypoxia acts synergistically with TNF priming to promote an activated circulating neutrophil phenotype with increased extravasation potential, which may enhance pathogen control. Systemic hypoxia likely also increases neutrophil intracellular ROS production and phagocytosis in some donors. Here, hypoxia may partially activate neutrophils in a similar manner to TNF, indicated by the lack of additional enhancement in TNF-primed neutrophils. Taken together, these findings indicate that hypoxia alone can promote neutrophil activation. Variable inter-donor neutrophil functional responses to hypoxia may influence pathogen control in patients with co-existing infection and systemic hypoxia.

S147 DISTINCT PATHOLOGICAL NEUTROPHILS IN IDIOPATHIC PULMONARY FIBROSIS IDENTIFY DISEASE ENDOTYPES

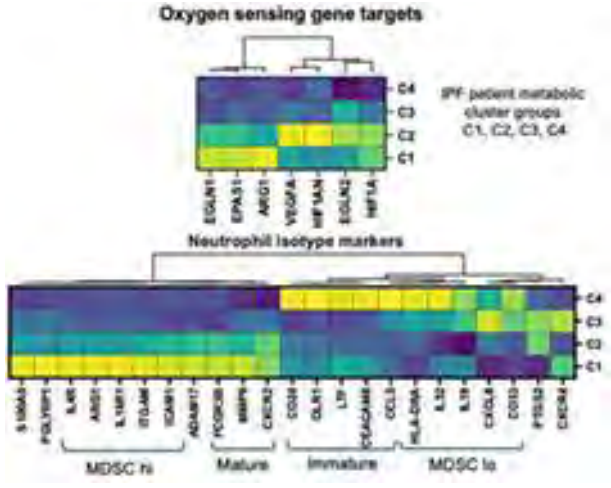
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Background Idiopathic Pulmonary Fibrosis (IPF) is a chronic interstitial lung disease associated with impaired gas transfer and systemic hypoxia. IPF pathophysiology can partially be attributed to neutrophil dysfunction. However, there is limited understanding of the role of neutrophils in IPF disease progression. Clinically, increased neutrophil counts in blood and bronchoalveolar lavage fluid strongly correlate with increased morbidity and early mortality in IPF. We used biomechanics and transcriptomics to characterise neutrophil subpopulations in IPF.

Methods Real-Time Deformability Cytometry was used to measure biomechanical parameters of neutrophils in whole blood from patients with IPF (N=37) and age-matched healthy controls (HC) (N=20). Gene profiles of isolated neutrophils were analysed by RNA-sequencing.

Results Neutrophils from patients with IPF were larger (p=0.0148) and stiffer (p=0.0197) than HC. Neutrophil size correlated with disease severity: IPF patients with larger neutrophils had lower percentage predicted forced vital capacity (p=0.0299 R^2=0.1277) and lower oxygen saturation (p=0.05, R=-0.3546). Gene-set-enrichment analysis identified significant changes in metabolic processes and pathways associated



Abstract S147 Figure 1 Hierarchical cluster analysis on RNA-seq neutrophil metabolic targets identified 4 distinct clusters. Aligning these clusters with (A) oxygen sensing targets (B) distinct neutrophil isotypes reveals clear endotypes

with pathological responses. Hierarchical cluster analysis on neutrophil metabolic targets identified 4 distinct clusters (figure 1). Aligning these clusters with patient disease severity, distinct neutrophil isotypes and oxygen sensing targets reveals clear endotypes.

Conclusion Neutrophils from patients with IPF have a distinct biomechanical profile, suggesting increased cell activation and subpopulation diversity. The larger, stiffer phenotype may cause neutrophil trapping in the extensive pulmonary microvasculature, increasing opportunity for neutrophil-driven lung injury. Metabolic dysregulation and hypoxia signalling in a subset of younger patients could underpin IPF endotypes and be exploited for precision medicine.

S148 EFFECTS OF HYPERCAPNIA LEAD TO DIFFERENTIAL PHENOTYPES IN COPD

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10.1136/thorax-2026-BTSabstracts.154

Introduction COPD patients can develop acute or chronic hypercapnic respiratory failure. Neutrophils are abundant inflammatory cells, with increased numbers in COPD. This research aims to define neutrophil phenotypes in an *in vitro* model of normocapnia, uncompensated hypercapnia, and following pH correction in buffered hypercapnia.

Methods Neutrophils from healthy volunteers (HV, n=5) and COPD (n=6) patients were isolated from whole venous blood using Percoll gradients and incubated in 5%CO_2 (normocapnia) and 15%CO_2 (hypercapnia) +/- pH buffering correcting acid balance (buffered hypercapnia). Neutrophil phenotype was assayed by flow cytometry using adhesion (CD11b, CD62L) and degranulation (CD63) markers after 4 hours incubation in hypercapnia +/- stimulation with platelet activating factor (PAF) and N-Formylmethionine-leucyl-phenylalanine (fMLP). Data is presented as median MFI [IQR]. Data was analysed with FlowJo v11.0.

Results Neutrophils from HV incubated in hypercapnia trended towards higher baseline CD11b expression (MFI: 1801 [1206-2998]) vs normocapnia (MFI: 901 [877-2257]), but no changes were observed in COPD subjects. Baseline CD62L levels showed the opposite- in hypercapnia a significant decrease was observed (MFI: 212 [82-419] vs 767 [593-1038], p=0.008), with a similar non-significant trend in COPD (MFI: 634 [182-964] vs 937 [261-1594], p=0.180). Baseline CD63 levels were comparable between hypercapnia vs normocapnia in HV (MFI: 502 [405-642] vs 518 [465-767] p=0.595) and COPD (MFI 546 [453-634] vs 569 [512-722] p=0.589).

After stimulation, neutrophils in hypercapnia and normocapnia shared similar trends of increased CD11b and CD63 expression in HV and COPD (figure 1). For CD62L, a significant decrease in expression was observed in HV in hypercapnia (p=0.008), with a small non-significant reduction in COPD (p=0.132) (figure 1).

Neutrophils from HV and COPD displayed blunted, significant reductions in delta MFI upon stimulation in all markers when incubated in buffered hypercapnia (figure 1). CD63 expression was significantly blunted compared to hypercapnia in HV (p=0.032) and COPD (p=0.041); but with trends to reduced expression of CD11b and CD62L (figure 1).

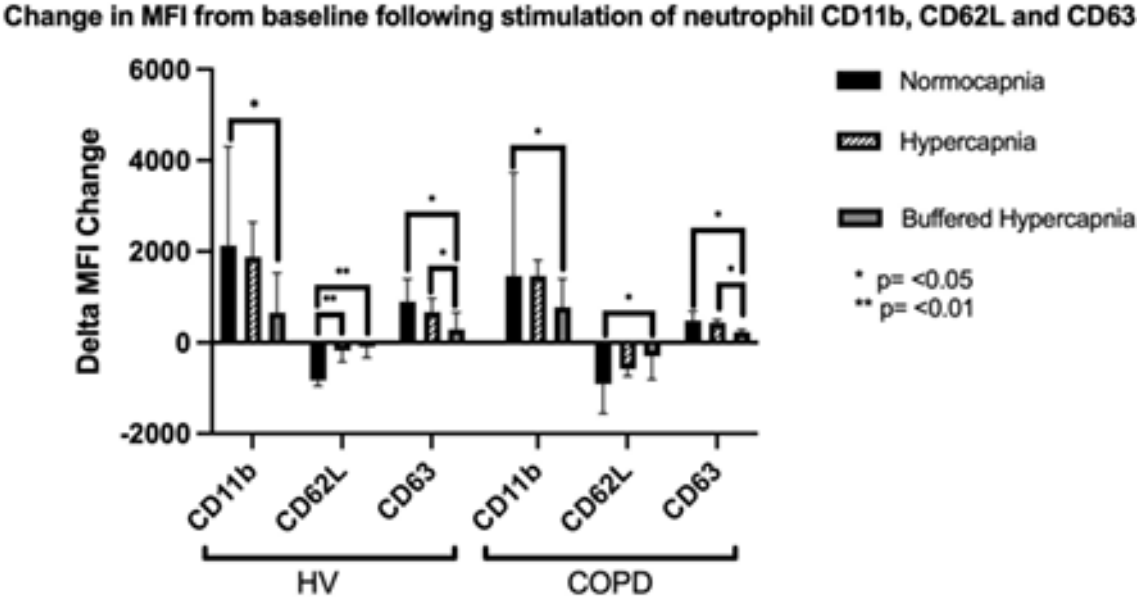
Conclusion In health, neutrophil adhesion, but not degranulation, is affected by hypercapnia. Upon stimulation, there is blunted neutrophil adhesion and degranulation responses both in health and COPD across normocapnia and buffered hypercapnia. Further cell responses in this model are required.

S149 HUMAN INHALED LIPOPOLYSACCHARIDE CHALLENGE INDUCES COORDINATED PULMONARY AND SYSTEMIC INFLAMMASOME ACTIVATION

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10.1136/thorax-2026-BTSabstracts.155

Background Inflammasomes are intracellular innate immune complexes that drive inflammatory responses and contribute to



Abstract S148 Figure 1 Delta MFI change of neutrophil adhesion and degranulation markers from baseline following stimulation

the pathogenesis of acute respiratory distress syndrome (ARDS), asthma and COPD. Despite increasing interest in inflammasome-targeted therapeutics, translational human models of inflammasome activation remain limited. Lipopolysaccharide (LPS), a component of gram-negative bacteria, activates multiple inflammasome pathways, including NLRP3 and inflammatory caspases. We hypothesised that inhaled LPS would induce measurable inflammasome activation in humans.

Methods Bronchoalveolar lavage (BAL) fluid, BAL cells and whole blood were collected from healthy volunteers undergoing inhaled LPS challenge in a controlled human model of acute lung inflammation. Inflammasome responses were assessed using cytokine quantification, bulk RNA sequencing of whole blood and BAL cells, single-cell RNA sequencing of BAL cells, and protein analyses of BAL fluid.

Results LPS inhalation significantly increased BAL concentrations of the inflammasome-dependent cytokines IL-1β and IL-18, whereas plasma concentrations were unchanged. Single-cell RNA sequencing identified pulmonary monocytes as the predominant source of IL-1β expression following LPS exposure and demonstrated enrichment of inflammasome-related transcriptional programmes within this population, including increased expression of NLRP3, MEFV and NINJ1. BAL-cell transcriptomics similarly revealed increased expression of inflammasome-associated genes, including NLRP3, AIM2, MEFV, CASP5, IL1β and IL18. Whole-blood transcriptomics demonstrated parallel systemic induction of inflammasome-related genes, including IL-1β, NLRP3, NLRC4, CASP4 and CASP5, indicating coordinated local and systemic responses to inhaled LPS. Consistent with inflammasome activation, exploratory Western blot analyses of BAL fluid demonstrated inflammasome-associated protein processing, with cleaved GSDMD detected in 18/21 LPS samples versus 1/3 controls and pro-IL-1β detected in 19/21 LPS samples whilst absent in controls.

Conclusions Inhaled LPS induces coordinated local and systemic inflammasome responses in humans, with pulmonary monocytes emerging as a major source of IL-1β expression. These findings demonstrate inflammasome activation in vivo and establish the human LPS inhalation challenge as a clinically relevant translational model of early inflammasome-driven lung inflammation. This model may facilitate mechanistic studies and evaluation of inflammasome-modulating therapies in respiratory disease.

S150 IMPAIRED ALOX15/5/FPR2 AXIS IN COPD MACROPHAGES

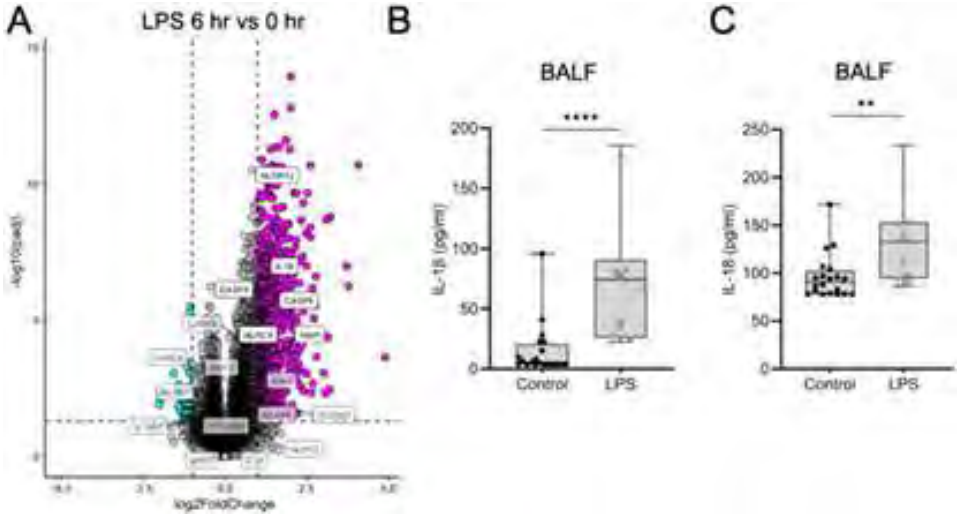
JSY Mah, J Devulder, A Lobosco, P Fenwick, Y Guo, S Bartlett-Pestell, R Lopez, D Gaboriau, L Donnelly, RJ Snelgrove, JA Wedzicha, LJ Finney. *Imperial College London, London, UK*

10.1136/thorax-2026-BTSabstracts.156

Background Chronic Obstructive Pulmonary Disease (COPD) is characterised by persistent airway inflammation, which may reflect defective resolution pathways rather than excessive inflammation alone. Resolvin D1 (RvD1) is a specialised pro-resolving mediator generated from docosahexaenoic acid via the arachidonate 15-lipoxygenase (ALOX15)–arachidonate 5-lipoxygenase (ALOX5) enzymatic pathway and subsequently signals through formyl peptide receptor 2 (FPR2). We previously showed that lower RvD1 levels were associated with prolonged recovery and worsened symptoms during COPD exacerbations.[1] As alveolar macrophages are essential for pathogen clearance and resolution of inflammation but are dysfunctional in COPD, we investigated whether COPD macrophages show altered capacity to generate and respond to RvD1 during bacterial stimulation.

Aim To assess the ALOX15–ALOX5–RvD1–FPR2 axis in COPD macrophages and determine whether exogenous RvD1 modulates inflammatory responses during bacterial challenge.

Methods Monocyte-derived macrophages from COPD patients (n=21) were pre-treated with vehicle or 1/10 nM RvD1 and then stimulated with heat-killed *H.influenzae*. IL-6, CXCL8 and IL-10 release were measured by ELISA. ALOX15, ALOX5



Abstract S149 Figure 1 LPS inhalation modulates inflammasome sensor and component genes systemically and increases inflammasome-dependent cytokine release in the lung. (A) Volcano plot of inflammasome sensor and component gene expression in whole blood cell by bulk RNA sequencing of whole blood 6 hours (hr) post LPS inhalation compared to baseline. Significantly upregulated or downregulated gene labels are opaque, and non- significant gene labels are transparent. (B-C) Quantification of IL-16 and IL-18 in BAL fluid (BALF) of volunteers 6 hours post-LPS inhalation compared to non-LPS control BALF. Data displayed as floating bars (min to max) line at mean. Points represent individual volunteer samples. N = 20 control and N = 11 LPS BAL fluid. **** p <0.0001, ** p <0.01. Data were compared by Mann Whitney test

and FPR2 expression/localisation were assessed over time using qPCR, flow cytometry and confocal microscopy.
Results ALOX5 expression was detectable in COPD macrophages at baseline but was downregulated following bacterial exposure. Conversely, ALOX15 was undetectable at baseline and was not induced by bacterial challenge at 24, 48 or 72 h, suggesting limited activation of the upstream enzymatic machinery required for endogenous RvD1 biosynthesis. Surface FPR2 expression by flow cytometry (n=6) was low overall, with some upregulation at 24h. Confocal microscopy (n=3) of permeabilised cells demonstrated FPR2 signal at 24 h, particularly in macrophages that had internalised *H.influenzae*, suggesting possible intracellular receptor localisation or receptor cycling following bacterial exposure. Functionally, 10 nM RvD1 significantly reduced IL-6 compared with vehicle at 24 h (180.2 vs 592.5 pg/mL, p=0.02) and reduced CXCL8 (828.4 vs 1074.9 pg/mL, p=0.02). RvD1 also increased IL-10 production (327.8 vs 169.1 pg/mL, p=0.1).
Conclusion These findings identify a limitation in COPD macrophage resolution capacity, characterised by absent ALOX15 induction and reduced ALOX5 following bacterial challenge. Despite low surface FPR2 expression, exogenous RvD1 retained cytokine-modulating activity, supporting further investigation of RvD1 replacement as a therapeutic strategy.

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The fibrotic niche: epithelial fibroblast interactions

S151 EPITHELIAL CELL DETACHMENT AS AN ORIGIN MODEL THAT PROMOTES BASAL TO ABERRANT BASALOID TRANS-DIFFERENTIATION

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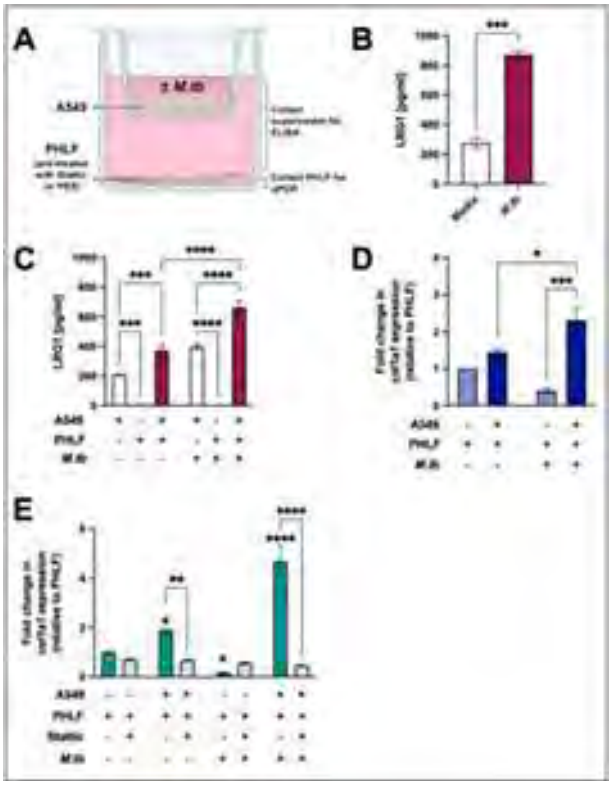
Background The epithelium is abnormal in IPF and is characterised by an increase in basal cells and the development of an aberrant basaloid epithelial cell type. Aberrant basaloid cells do not express Keratin 5 (KRT5) levels which causes loss of basal cell identity, altered regeneration after injury, and abnormal repair. Their origin remains unclear but are hypothesised to come from AT2 cells - which are thought to gain Keratin 17 (KRT17) as they transdifferentiate into aberrant basaloid cells. However, there is an unexplained expansion of KRT5+/KRT17+ airway basal cells that have become detached from the airway basal level and are observed at extremely high levels in IPF tissue. I hypothesise that detached small airway basal cells are another source of pro-fibrotic KRT5-/KRT17+ aberrant basaloid population which could be contributing to epithelial instability, fibrosis progression, and impaired tissue repair.
Methods KRT5 and KRT17 gene expression was evaluated in ihBECs (immortalised human bronchial epithelial cells) and primary basal cells (control and IPF) using real time qPCR in monolayer and detached cells. Cells were seeded at high density to induce overcrowding to promote detachment. Once 95% confluent, detached cells were collected from supernatant and dead cell removal was performed. The monolayer cells were also collected at the same time for RNA extraction. Fibrotic and transitional markers mRNA gene expression (MMP7, TP63, GDF15, ITGB6) was evaluated in ihBECs and validated in primary basal cells via qPCR.
Results Epithelial cell detachment leads to an aberrant basaloid gene expression pattern in the detached cells with loss of KRT5 and increase of KRT17 in ihBECs, which was replicated in primary basal cells. Detached ihBECs showed pro-fibrotic phenotype with up-regulation of MMP7, ITGB6 and GDF15 and down-regulation of TP63 (n=3). Detached primary basal cells also showed an increase in pro-fibrotic markers (n=2).
Conclusion Epithelial detachment leads to loss of KRT5 and increased KRT17 consistent with the phenotype of aberrant basaloid cells seen in IPF. Furthermore, detached airway basal cells demonstrate a pro-fibrotic phenotype which may indicate a role in promoting fibrogenesis.

S152 LUNG EPITHELIAL CELL-DERIVED LEUCINE-RICH ALPHA2 GLYCOPROTEIN-1 (LRG1) DRIVES THE DEVELOPMENT OF TUBERCULOSIS-INDUCED PULMONARY FIBROSIS

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Background Over half of Tuberculosis (TB) survivors develop pulmonary fibrosis after successful treatment of *Mycobacterium tuberculosis* (*M.tb*) infection, contributing to substantial morbidity and mortality. Fibrosis involves excessive collagen deposition from fibroblasts, but the mechanisms driving TB-induced fibrosis remain unclear. Leucine-rich alpha2 glycoprotein-1 (LRG1) promotes lung fibrosis via STAT3 signalling in animal models. Furthermore, LRG1 expression is elevated in active TB patients compared to controls. Whether LRG1 drives the development of fibrosis in TB is unknown.
Objectives We hypothesise that LRG1 expression following *M.tb* infection drives the development of pulmonary fibrosis. Therefore, the aim of this study is to investigate cellular sources of LRG1 during *M.tb* infection and assess the pro-fibrotic effects of LRG1.
Methods A lung epithelial cell line (A549) was infected with *M.tb* at multiplicity of infection (MOI) of 10. Secretion of LRG1 was quantified by ELISA. To assess *M.tb*-induced pro-fibrotic responses, a transwell system of *M.tb*-infected A549 and primary human lung fibroblasts (PHLF) was developed (figure 1A). Collagen gene expression in PHLF was measured by qPCR. In later experiments, PHLF were pre-treated with Stattic, a STAT3 inhibitor, before *M.tb* infection of A549 cells.
Results *M.tb*-infected A549 cells secrete significantly more LRG1 than controls after 24 h (872.2 ± 46.7 vs. 276.3 ±63.1pg/ml, $p<0.001$, figure 1B). Co-culture of *M.tb*-infected A549 cells with PHLF induced a 1.7-fold increase in LRG1 secretion compared to un-infected controls ($p<0.0001$, figure 1C). *M.tb* infection of PHLF alone induced minimal LRG1 secretion relative to A549 controls (figure 1C). Furthermore, gene expression of collagen, a fibrotic marker, was significantly upregulated by 1.6-fold in PHLF co-cultured with *M.tb*-infected A549 cells compared to un-infected controls ($p=0.027$, figure 1D). Lastly, blocking LRG1-induced activation of STAT3 significantly reduced collagen gene expression in PHLF co-cultured with *M.tb*-infected A549 cells compared to vehicle controls ($p<0.0001$, figure 1E).



Abstract S152 Figure 1 LRG1 expressed during M.tb infection drives pro-fibrotic responses. (A) Schematic of M.tb-infected A549 cells co-cultured with primary human lung fibroblasts (PHLF) using a transwell system; (B) Secreted LRG1 from M.tb-infected A549 cells at MOI of 10 for 24 h was quantified by ELISA (n=3 independent experiments, unpaired t-test, *** p<0.001); (C) Secreted LRG1 from PHLF (n=3 donors), grown below A549 cells infected with M.tb at MOI of 10 for 48 h, was quantified by ELISA. Experimental controls include A549 or PHLF only (two-way ANOVA, *** p<0.001, **** p<0.0001); (D) qPCR analysis of fold change in collagen (col1a1) gene expression from PHLF (n=3 donors), grown below A549 cells infected with M.tb at MOI of 10 for 48 h. Gene expression is relative to PHLF only controls (two-way ANOVA, *p<0.05, *** p<0.001). (E) A549 cells, grown on transwells above PHLF pre-treated with Stattic (a STAT3 inhibitor) or PBS, were infected with M.tb at MOI of 10 for 48 h (n=3 donors). qPCR of PHLF for col1a1 gene expression relative to PHLF only controls (two-way ANOVA, *p<0.05, ** p<0.01, **** p<0.0001)

Conclusion Overall, we show that *M.tb*-activated cellular networks drive pro-fibrotic responses via LRG1. Thus, targeting LRG1 may represent a novel host-directed therapy to reduce TB-induced pulmonary fibrosis in patients.

S153 TARGETING AKAP13 RHOGEF ACTIVITY AMELIORATES PRO-FIBROTIC PHENOTYPES DRIVEN BY THE IPF ASSOCIATED AKAP13 RISK VARIANT

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Background and Aims Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease with limited therapeutic options. The common risk variant rs62025270 lies near AKAP13, a multifunctional scaffold that integrates RhoA and protein kinase A signalling. The rs62025270 variant is associated with increased AKAP13 expression in human fibrotic lung tissue. We investigated how the rs62025270 variant alters epithelial signalling and evaluated the therapeutic potential of targeting AKAP13.

Methods CRISPR-Cas9 engineered immortalized human bronchial epithelial cells carrying rs62025270 were used to assess AKAP13 transcript architecture, fibrosis-related gene expression, RhoA activation, adhesion, proliferation, TGFβ signalling, and intracellular cyclic AMP. Mechanistic studies used isoforms overexpression, together with pharmacological inhibition of the AKAP13 Rho GEF domain using A13. Findings were examined in idiopathic pulmonary fibrosis precision-cut lung slices.

Results rs62025270-bearing iHBECs exhibited selective upregulation of AKAP13 isoforms, accompanied by increased cell adhesion and reduced proliferation. Transcriptomic profiling revealed upregulated fibrosis-related genes in rs62025270-bearing iHBECs, including *SAA1*, *FGF2*, *MMP1*, *CTSB*, *COL4A1*, and *CDKN1A*. rs62025270-bearing iHBECs also displayed increased RhoA activation and SMAD2 phosphorylation following LPA stimulation. Furthermore, cells harbouring the AKAP13 variant showed reduced intracellular cAMP levels. Pharmacological inhibition of AKAP13 with A13 reversed the pro-adhesive phenotype and reduced RhoA activation in iHBECs. Moreover, A13 suppressed SERPINE1, CCN2, and MMP7 expression, reduced SMAD2 nuclear translocation, and decreased hydroxyproline release in IPF-derived PCLS.

Conclusions Presence of the rs62025270 variant increases aberrant AKAP13 expression, disrupts epithelial homeostasis and promotes pro-fibrotic signalling. Inhibition of AKAP13's RhoGEF domain with A13 restores epithelial function and attenuates fibrotic activation, supporting targeting AKAP13 in IPF.

S154 GαQ/11 MODULATES THE EXTRACELLULAR MATRIX AND FIBROBLAST-EPITHELIAL INTERACTIONS TO CONTROL ALVEOLAR REPAIR

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Introduction Efficient repair is essential to maintain lung architecture and function. The extracellular matrix (ECM) provides repair signals and is altered in lung diseases such as fibrosis and emphysema. Mesenchymal $G\alpha_{q/11}$ inhibition alters lung ECM in vivo,[1] however the role of this ECM in controlling repair is unknown.

Aim Understand how fibroblast $G\alpha_{q/11}$ signalling influences ECM-driven lung repair and cell-cell interactions.

Materials and Methods Fibroblasts (mouse embryonic (MEFs), human lung (HLFs)) or epithelial cells (**mouse**: E10; **human**: A549, small airway (SAECs), induced pluripotent stem cell (iPSC)-derived type 2 (AT2)) were seeded upon ECM generated by species-matched fibroblasts (wild-type (WT) or *Gnaq*$^{-/-}$;*Gna11*$^{-/-}$ MEFs; HLFs cultured +/- YM-254890). ECM was static or stretched, with cells in direct contact or suspended above. Wound closure and cellular spread, viability, and proliferation were quantified.

EnrichGO and gseGO (ClusterProfileR package, R) were used for pathway analysis of RNASeq data.

For spheroids, cells were stained with CellTracker dyes, grown in mono- or co-culture in ultra-low adhesion plates, and morphology assessed.

Results In mouse and human models, epithelial (A549, SAEC, AT2) and fibroblast wound healing was attenuated on static ECM generated by $G\alpha_{q/11}$-inhibited fibroblasts (e.g. A549s: 6.4% on YM-254890-exposed ECM vs 16.5% on control, $p=0.01$). Stretched $G\alpha_{q/11}$-inhibited ECM impeded healing compared with control without cell-ECM contact (MEFs, $p<0.05$), implying stretch-induced release of repair regulators. Epithelial cell spread (32.9% vs 55.4% circular SAECs, $p<0.01$), but not proliferation or viability, were reduced on $G\alpha_{q/11}$-inhibited ECM.

$G\alpha_{q/11}^{-/-}$ MEFs formed more compact spheroid cores with greater central epithelial cell exclusion and a clearer external epithelial shell than WT controls, implying altered cell-cell interactions.

RNASeq revealed enhanced repair activity, including genes associated with ECM components, fibroblast-to-myofibroblast differentiation, cytoskeletal tension, and cell adhesion, when $G\alpha_{q/11}^{-/-}$ MEFs were transferred to WT-generated ECM. Conversely, WT MEFs exhibited dampened wound healing profiles on $G\alpha_{q/11}^{-/-}$-generated ECM, with increased expression of TGFβ-inhibitory genes, and downregulated immune-related cytokines.

Conclusion Fibroblast $G\alpha_{q/11}$ inhibition modifies ECM properties, influencing multiple components of the injury response, and affects fibroblast-epithelial cell interactions. Further evaluation of these pathways may identify methods of manipulating lung repair therapeutically.

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S155 HEMICENTIN-1 MEDIATES PROFIBROTIC SIGNALLING IN LUNG FIBROBLASTS IN IDIOPATHIC PULMONARY FIBROSIS

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Background and Aims Idiopathic pulmonary fibrosis (IPF) is a progressive, chronic lung disease with a poor prognosis. Whole genome sequencing identified a large ECM glycoprotein hemicentin-1 (HMCN1) as having a high burden of rare variants associated with IPF. Transcriptomic analysis indicates HMCN1 upregulation in IPF lung tissues although its role in IPF pathogenesis remains unclear. Our study aims to understand the function of HMCN1 in lung fibrosis.

Methods mRNA expression of human healthy (non-IPF) and IPF human lung fibroblasts (HLFs), airway basal cells, AT2 cells, pulmonary microvascular endothelial cells (HPMEC) and immortalised human bronchial epithelial cells (iHBECs) was assessed by RT-qPCR. Knockdown (KD) of HMCN1 in fibroblasts was performed via CRISPR-Cas 9 targeting exon 1 and validated using PCR and Immunofluorescence (IF). Downstream of the TGFβ signalling pathway genes were assessed. Proliferation of KD cells after TGFβ were measured using the BrdU ELISA approach. Cell migration was measured using wide-field microscopy, with ihBECS seeded on top of a CRISPR-mediated KD fibroblast-derived matrix.

Results Expression of *HMCN1* mRNA is high in fibroblasts while low or undetectable in epithelial and endothelial cells, with the IPF fibroblasts having the highest levels. TGFβ stimulation (2ng/ml) induced *HMCN1* expression in a time-dependent manner in non-IPF HLFs but not in IPF HLFs. TGFβ-induced *SERPINE1, and FN1* mRNA expression was dampened in HMCN1-KD HLFs compared to HLFs with intact HMCN1 expression, while CCN2 remained comparable. HMCN1 knockdown did not affect fibroblast proliferation under fibrotic conditions, and epithelial cell migration was reduced on fibrotic matrices compared to baseline conditions.

Conclusions In conclusion, our current data suggest that HMCN1 may be involved in profibrotic signalling in fibroblasts which may indicate a role in IPF pathogenesis.

S156 FACTOR-INHIBITING HIF (FIH) REGULATES FIBROBLAST PHENOTYPE AND EXTRACELLULAR MATRIX REMODELLING IN THE AGEING HUMAN LUNG

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Background With ageing, human lungs progressively lose compliance and exhibit extracellular matrix (ECM) remodelling, but upstream molecular drivers remain uncertain. ECM remodelling is also a hallmark of fibrogenesis, raising the possibility of shared molecular programmes. We previously identified that, in lung fibrosis, loss of Factor inhibiting HIF (FIH) promotes formation of 'bone-type' pyridinoline collagen cross-links and tissue stiffness. However, any role for FIH in lung ageing remains uncertain.

Aims To identify molecular determinants of human lung ageing and define any role of FIH in this process.

Methods Human lung RNA-seq data were analysed for age-associated changes. Gene silencing was performed in primary lung fibroblasts, with phenotypic and transcriptomic profiling. FIH protein abundance and pyridinoline collagen cross-links were quantified in human lung tissue across a range of ages.

Results FIH expression declined with age at both transcript and protein levels in human lung tissue. This was accompanied by activation of extracellular matrix remodelling pathways without induction of a canonical glycolytic HIF programme. FIH-silenced fibroblasts displayed reduced proliferation and features of senescence. Transcriptomic profiling identified overlap with the ageing human lung, including enrichment of ECM-remodelling pathways. Aged lung tissue exhibited increased pyridinoline collagen cross-links, consistent with enhanced matrix maturation following loss of FIH activity.

Conclusions We identify FIH as a regulator of fibroblast phenotype and ECM composition in the human lung. Age-related loss of FIH is associated with selective activation of matrix-remodelling programmes and increased 'bone-type' collagen cross-linking, supporting a role for FIH as an upstream regulator of age-related structural remodelling.

Advancing lung cancer diagnostics and treatment

P1 ROBOTIC BRONCHOSCOPY FOLLOWING PRIOR NON-DIAGNOSTIC BIOPSY: DIAGNOSTIC YIELD AND MALIGNANCY DETECTION IN A SALVAGE DIAGNOSTIC COHORT

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Background Robotic-assisted bronchoscopy (RAB) is increasingly utilised for diagnosis of peripheral pulmonary nodules. However, its performance following prior unsuccessful or non-diagnostic biopsy remains poorly defined. We evaluated the diagnostic performance of RAB in lesions with previous failed biopsies and its effectiveness in detecting malignancy.

Methods A retrospective analysis was performed of the first 271 RAB procedures over a three-month period at a tertiary interventional pulmonology centre, comprising 310 pulmonary nodules after exclusion of therapeutic procedures. Nodules were stratified into those with one or more prior failed or non-diagnostic biopsies (prior biopsy), and biopsy-naïve lesions. Primary outcomes were diagnostic yield per nodule determined using the American Thoracic Society DELPHI strict definition, and sensitivity for malignancy. Secondary outcomes included need for repeat biopsy following index RAB. Categorical variables were compared using chi-squared testing; continuous variables are presented as median (IQR).

Results Twenty-five nodules (8.3%) underwent RAB following a prior non-diagnostic CT-guided transthoracic needle biopsy (n=22) or navigational bronchoscopy (n=3). Compared with biopsy-naïve lesions, nodules in the prior biopsy cohort were more frequently located in the outer third of the lung (76.0% vs 44.0%, p=0.009) and demonstrated a lower prevalence of malignancy (60.0% vs 72.9%, p=0.17). Diagnostic yield was lower following prior biopsy (64.0% vs 80.9%, p=0.046); however, sensitivity for malignancy remained high and comparable between groups (93.3% vs 94.6%, p=0.842). Among lesions undergoing repeat RAB after a prior non-diagnostic RAB (n=8), diagnostic yield was 75% with 100% sensitivity for malignancy and no subsequent biopsies required.

Abstract P1 Table 1

Baseline Characteristics	Prior biopsy	Biopsy-naïve	Wilson 95% Confidence Interval (CI)	P-value	Prior non-diagnostic RAB
Number of Nodules	25	277			8
Median Lesion size (mm) (IQR)	16 (12-25)	14 (10-19)			
Centrality:				**0.009**	
Inner	3 (12.0%)	69 (24.9%)			2 (25%)
Middle	3 (12.0%)	86 (31.0%)			2 (25%)
Outer	19 (76.0%)	122 (44.0%)			4 (50%)
Nodule Type:				0.532	0 (0%)
GGO	2 (8.0%)	11 (4.0%)			1 (12.5%)
Part-solid	6 (24.0%)	48 (17.3%)			1 (12.5%)
Peri-cystic	0 (0%)	6 (2.2%)			6 (75%)
Solid	17 (68.0%)	212 (76.5%)			
Median number of CBCT spins (IQR)	2 (1-3)	2 (1-3)			
Prior biopsy modality:					-
CT-TTNB	22	-			
Nav Bronchoscopy	3	-			
Malignancy prevalence (%)	60.0%	72.9%	40.7-76.6%; 67.4-77.8%	0.17	62.5%
Diagnostic Outcomes	**Prior failed biopsy**	**No prior biopsy**		**P-value**	
Diagnostic yield per nodule (%)	64%	80.9%	44.5-79.8%; 75.8-85.1%	**0.046**	75%
Malignancy sensitivity (%)	93.3%	94.6%	70.2-98.8%; 90.5-96.9%	0.842	100%
Non-diagnostic rate (%)	36%	19.1%		**0.046**	25%
Repeat biopsy required following index RAB (%)	4.0%	8.3%		0.446	0
Lesion size (max diameter of AP/SI/				**0.011**	
LP planes):	0 (0.0%)	52 (18.8%)			1 (12.5%)
<10 mm	16 (64.0%)	156 (56.3%)			4 (50%)
10-19 mm	3 (12.0%)	46 (16.6%)			2 (25%)
20-29 mm	6 (24.0%)	23 (8.3%)			1 (12.5%)
≥30 mm					

Values are presented as median (IQR) or n (%). CBCT = cone beam CT; CT-TTNB = CT-guided transthoracic needle biopsy; EBUS = endobronchial ultrasound; RAB = robotic-assisted bronchoscopy.
*Chi-squared test for overall centrality distribution

Conclusion RAB maintains high sensitivity for malignancy following prior failed or non-diagnostic biopsy while minimising further invasive procedures. Although strict diagnostic yield was lower in the prior biopsy cohort, lesions were more peripheral and demonstrated lower malignancy prevalence, which may contribute to the reduced yield given the greater challenge of establishing definitive benign diagnoses. Similar findings were observed in the repeat-RAB subgroup. These findings suggest that RAB effectively identifies missed cancers in this difficult-to-diagnose population and support its use as an effective salvage diagnostic strategy for technically challenging and previously non-diagnostic pulmonary nodules.

P2 THE IMPACT OF EXPERIENCE ON RADIATION DOSE AND PROCEDURE DURATION IN SHAPE-SENSING ROBOTIC-ASSISTED BRONCHOSCOPY

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Introduction Shape-sensing robotic-assisted bronchoscopy (ssRAB) is increasingly used for the diagnosis of peripheral pulmonary lesions, but data describing operator learning beyond diagnostic yield remains limited. Procedural efficiency and radiation exposure are important markers of technical development and patient safety, yet their evolution during early operator experience is poorly characterised. We evaluated changes in procedure duration and radiation dose across the first 100 diagnostic cases of three staggered operators at a tertiary referral centre.

Methods We conducted a retrospective analysis of prospectively collected data. Three operators performing ssRAB were included, with staggered start dates. Operator 1 commenced first, followed sequentially by Operator 2 after 7 cases and operator 3 after 124 total cases. The first 100 consecutive diagnostic cases per operator were analysed. Procedure time per nodule and radiation dose per nodule were calculated for each case. Cases were divided into quartiles of 25 and median values with interquartile ranges (IQR) reported. Differences between groups were assessed using the Mann-Whitney U test. A p-value <0.05 was considered statistically significant.

Results Significant reductions in procedure time per nodule and radiation dose per nodule were demonstrated across the cohort. Results are presented in table 1.

Conclusion All three operators demonstrated reductions in radiation dose per nodule across their first 100 cases (Op 1: -63.2%, $p<0.001$; Op 2: -38.0%, $p=0.050$; Op 3: -44.3%, $p=0.003$). Operator 3, who commenced after Operators 1 and 2 were established, started with the lowest baseline procedure duration and radiation dose, suggesting later operators benefit from the accumulated experience of their predecessors. These findings support a structured, staggered operator introduction model during ssRAB programme development.

P3 REAL-WORLD OUTCOMES OF NAVIGATIONAL BRONCHOSCOPY FOR PERIPHERAL PULMONARY LESIONS: A SINGLE CENTRE ANALYSIS OF DEMOGRAPHICS AND PDL1 SPECIMEN ADEQUACY

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Background Electromagnetic navigational bronchoscopy (ENB) is a minimally invasive technique that improves diagnostic access to peripheral pulmonary lesions (PPLs) while maintaining a favourable safety profile. Recent evidence has demonstrated non-inferior diagnostic accuracy compared with transthoracic needle biopsy,[1] with significantly fewer complications. However, real-world data on diagnostic performance, safety, and suitability for molecular testing remain limited.

Aim To evaluate the diagnostic yield, safety profile, and molecular adequacy of ENB for PPLs in a tertiary referral centre.

Methods A retrospective review was performed of all ENB procedures undertaken using the Medtronic Illumisite™ navigation platform between 2023 and 2025. Data collected included patient demographics, lesion characteristics, diagnostic outcomes, complications, and adequacy for ancillary molecular studies.

Results Fifty-two ENB procedures were performed. Most patients (86.5%, n=45) underwent ENB for suspected primary lung cancer. Lesions were predominantly located in the upper lobes (58%, n=30), with a mean diameter of 2.7cm ± 1.4SD.

Overall diagnostic yield was 69.2% (36/52). Malignancy was diagnosed in 29 cases (55.8%), comprising 25 primary lung cancers and 4 other malignant lesions. Diagnostic yield was higher for lesions >20 mm than for lesions ≤20 mm (77.4% vs 60.9%).

The overall complication rate was 5.8% (n=3), including Pneumothorax (3.8%), with one requiring chest drain insertion (1.9%) and one episode of bronchospasm (1.9%). No major procedure-related mortality occurred.

80% (n=20) of the primary lung cancers were non-small cell lung carcinoma (NSCLC), with adequate tissue for PD-L1

Abstract P2 Table 1 Showing the change in median procedure time and radiation dose per nodule for operators 1 - 3

Metric	Operator	Q1 Median (IQR)	Q4 Median (IQR)	% Change	p-value
Radiation dose per nodule (Gy.cm^2)	Op 1	37.0 (27.0-83.0)	13.6 (8.8-30.1)	-63.2%	0.0003
	Op 2	28.0 (15.0-40.5)	17.4 (11.1-24.9)	-38.0%	0.050
	Op 3	14.3 (9.7-23.1)	8.0 (3.5-13.3)	-44.3%	0.003
Time per nodule (mins)	Op 1	49.0 (37.0-60.0)	47.0 (21.0-59.0)	-4.1%	0.352
	Op 2	44.0 (34.0-61.0)	34.0 (25.0-49.0)	-22.7%	0.045
	Op 3	35.0 (24.5-50.0)	26.0 (20.5-34.0)	-25.7%	0.067

testing obtained in 90% of NSCLC cases (18/20). Six initially negative cases were subsequently confirmed as malignant following image-guided biopsy or surgical resection, yielding a sensitivity of 79.3%.

Diagnostic performance improved over the study period, with diagnostic accuracy increasing from 66.7% in year 1 to 72.2% in year 3.

Conclusion ENB demonstrated favourable diagnostic yield, low complication rates, and high molecular adequacy for PD-L1 testing. These findings support ENB as a safe and effective alternative to TTNB for the diagnosis of PPLs and its role in facilitating biomarker-driven lung cancer care.

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P4 ADEQUACY OF TISSUE ACQUIRED BY SHAPE-SENSING ROBOTIC BRONCHOSCOPY FOR MOLECULAR PROFILING OF NON-SMALL CELL LUNG CANCER

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Introduction and Objectives Shape-sensing robotic bronchoscopy (ssRAB) is increasingly utilised to obtain tissue diagnosis of pulmonary nodules. Data on tissue adequacy for comprehensive molecular profiling via ssRAB are, however, limited.[1] We evaluated tissue acquired for diagnosis, PD-L1 testing and next generation sequencing (NGS) in patients with suspected lung cancer.

Methods Retrospective analysis of 206 patients undergoing ssRAB-guided biopsy with a combined total of 212 pulmonary nodules between 01/2024 and 01/2025. Patient demographics, nodule characteristics, diagnostic outcomes, and molecular adequacy were analysed. ssRAB was performed in conjunction with radial endobronchial ultrasound (rEBUS) and fluoroscopy alone (no cone-beam CT).

Results The cohort included 98 males and 108 females with 157 ever smokers and 49 never smokers. Median nodule size was 22.5mm (IQR 14.5-32mm). Overall diagnostic yield of all nodules was 77.8% with 121/212 nodules (57.1%) confirming lung cancer (figure 1). Adenocarcinoma was the most common histological diagnosis in 82 of the 121 nodules confirming lung cancer (67.8%). Reflexive testing for PD-L1 and NGS was performed in 110 and 89 cases respectively. PD-L1 was completed in 99/110 cases (90%), and complete DNA and RNA NGS in 70/89 cases (78.7%) with partial sequencing in 5/89 (5.6%). 58 gene mutations were detected, the most common being KRAS 31/58 (53%) and EGFR 14/58 (24.1%). Additional alterations were identified in MET, BRAF, HER2, ALK, RET and ROS1. Mean NGS processing time was 26.4days.

Complete NGS was more frequently achieved in nodules demonstrating concentric rEBUS views (82.8% vs 50%). Insufficient NGS samples were more frequent in part-solid versus solid nodules (27.3% vs 13.1%). Cryobiopsy was performed in 104 cases of confirmed lung cancer and yielded the diagnosis in 97 cases (93.3%).

Conclusions We demonstrated that fluoroscopy-guided ssRAB biopsy provides high diagnostic and molecular profiling adequacy for pulmonary nodules, particularly in solid nodules with concentric rEBUS view and when cryobiopsy is utilised.

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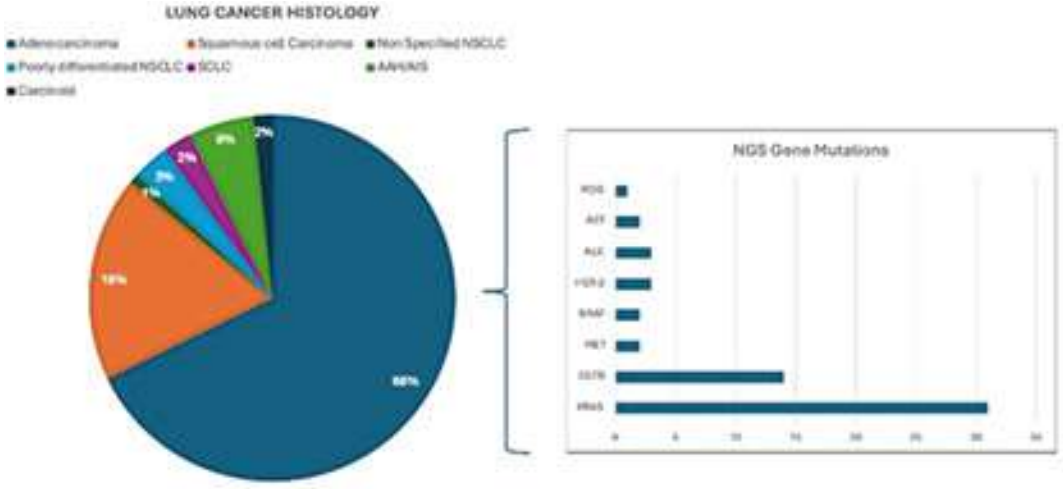
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P5 DIAGNOSTIC YIELD AND SAFETY OF CT-GUIDED LUNG BIOPSY PERFORMED FOLLOWING MULTI-DISCIPLINARY TRIAGE OF ROBOTIC NAVIGATIONAL BRONCHOSCOPY REFERRALS

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Introduction National lung cancer screening has increased the detection and work-up of small pulmonary nodules. As a tertiary referral centre with a robotic-assisted navigational bronchoscopy (RAB) biopsy service, referrals for robotic-assisted lung nodule biopsy have risen substantially from across the region. In December 2024, a multidisciplinary triage meeting involving



Abstract P5 Figure 1 Histology of lung cancers diagnosed by ssRAB-guided biopsy of pulmonary nodules, and reflexic NGS profile testing

Abstract P5 Table 1 Histopathological classification of biopsy outcomes

Positive	Malignancy
True negative	Necrotising or non-necrotising granulomatous inflammation. Organising pneumonia with MDT consensus that needle position reflects the nodule and CT appearances consistent with OP. Other – including amyloid, PAP, hamartoma. Non-malignant biopsy where subsequent follow up imaging demonstrated resolution or shrinkage of the nodule or stability over 12 months.
Indeterminate	Nodules undergoing surveillance after biopsy including those reported as fibrosis or inflammation.
False negative	Failed biopsy (no tissue or procedure abandoned for technical reasons). Other biopsy result – where subsequent repeat biopsy or surgery demonstrated malignancy.

bronchoscopists and radiologists was introduced to direct patients towards surveillance, CT-guided biopsy (CTB), or RAB. This study evaluated all CTB outcomes over a 12-month period and compared diagnostic yield and complication rates between patients initially referred from external institutions for RAB but re-directed to CTB (group 1) and those referred directly for CTB (group 2) from both internal and external referral sources.

Methods Biopsy outcomes were classified as positive, true negative, false negative, or indeterminate according to table 1, following review of the electronic patient record and PACS. Mediastinal, chest wall and pleural biopsies were excluded from the analysis. Outcomes and complication rates, including pneumothorax (PTX), chest drain insertion requiring admission, haemoptysis, and air embolism were compared between groups. A secondary analysis compared maximum nodule size (mm) between groups.

Results A total of 208 CT-guided lung biopsies were performed: 47/208 (22.6%) in group 1 and 161/208 (77.4%) in group 2. Positive or true negative results were achieved in 41/47 (87.2%) of group 1 and 138/161 (85.7%) of group 2 (p=0.79). PTX occurred in 8/47 (17.0%) of group 1 and 41/161 (25.5%) of group 2 (p=0.24). Chest drain insertion requiring admission was required in 4/47 (8.5%) and 17/161 (10.6%) respectively (p=0.68). One air embolism occurred in group 2. Median nodule diameter was 18 mm (range 7–50 mm) in group 1 and 21 mm (range 6–142 mm) in group 2.

Conclusions Patients redirected to CT-guided biopsy following initial navigational bronchoscopy referral and triage had comparable diagnostic yield and complication rates at biopsy compared to those referred directly for CTB. Multidisciplinary triage may therefore support efficient management of a high-volume tertiary referral pathway for lung nodule biopsy.

P6 OPTIMISING MOLECULAR CHARACTERISATION OF NSCLC THROUGH COMBINED FLEXIBLE BRONCHOSCOPY AND EBUS-TBNA

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10.1136/thorax-2026-BTSabstracts.168

Background It is estimated that at diagnosis a total of 50%-60% of patients with advanced Non Small Cell Lung Cancer (NSCLC) have an actionable driver alteration, with similar estimates reported for early disease.[1] Advances in molecular testing and precision oncology have transformed the management of NSCLC, enhancing diagnosis, treatment and outcomes.[1] Comprehensive molecular testing is recommended by current guidelines, with biomarker-guided treatment demonstrating superior survival outcomes to non-targeted therapy.[2]

Endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) is the standard first-line investigation for mediastinal staging and diagnosis of lung cancer. While EBUS provides high diagnostic yield, concerns remain regarding the adequacy of cytology specimens for comprehensive molecular profiling in NSCLC. The addition of flexible bronchoscopy with endobronchial biopsies and brushings during the same procedure may improve adequacy.

Aim To evaluate the impact of adding flexible bronchoscopy to EBUS-TBNA on molecular adequacy for predictive biomarker testing in NSCLC.

Method A retrospective review was conducted of all EBUS procedures performed at our institution over a 12-month period. Procedures were classified as EBUS alone or combined EBUS with flexible bronchoscopy. Rates of molecular adequacy for comprehensive predictive biomarker testing were analysed in patients with NSCLC.

Results A total of 387 EBUS procedures were performed, including 155 combined EBUS-bronchoscopy procedures. Malignancy was diagnosed in 156 cases, of which 92 (59%) were NSCLC (60 adenocarcinoma, 32 squamous cell carcinoma).

Overall molecular adequacy was achieved in 83/92 (90%) cases. EBUS cytology alone provided adequate molecular profiling in 50 cases (54%). The addition of bronchoscopic biopsies and brushings increased cumulative adequacy to 90% (83/92). Notably, 16 patients achieved adequate molecular profiling from bronchoscopic samples despite negative EBUS cytology, potentially avoiding a repeat diagnostic procedure.

Conclusion Adding flexible bronchoscopy to EBUS-TBNA significantly improves molecular adequacy in NSCLC. A combined approach enhances diagnostic efficiency, and may reduce the need for repeat invasive procedures.

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P7 A LONGITUDINAL ANALYSIS OF THE FREQUENCY AND INDICATION FOR REPEAT ENDOBRONCHIAL ULTRASOUND TRANSBRONCHIAL NEEDLE ASPIRATION (EBUS-TBNA) SAMPLING IN A NATIONAL ACADEMIC CANCER CENTRE

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Introduction In the era of immunotherapy and targeted therapy, obtaining adequate samples will maximise the options

available for therapy. The WABIP Expert Panel consensus statement designates the rate of inadequate EBUS-TBNA sampling a quality standard for.[1] The aim of this longitudinal study is to evaluate the frequency of and reason for repeat sampling in a large academic centre with a designated national cancer service.
Methods With ethical approval, a retrospective chart review was performed on cases in which an EBUS-TBNA was performed. The locations included an academic (with rapid onsite evaluation (ROSE)) and a satellite hospital (without ROSE).
Results Between January 2019 and December 2025, 2540 EBUS procedures were performed (2367 in academic centre (93%) and 173 in satellite centre (7%). A total of 106 (4.5%) repeat EBUS-TBNA were performed (94 patients had 2 and 6 patients had 3 procedures). The number of repeat EBUS-TBNA increased from 5 (1.4% of total EBUS procedures) in 2019 to 26 (6.1%) in 2025 (p=.0015).

The number of EBUS procedures requiring one or more repeat was higher in the satellite centre (no ROSE 18/173 10.4%) in comparison to the academic centre (82/2285 (3.5%)) p=.0001). The indications for repeat procedure were first sample inadequate for cytological analysis (n=39 (37%)) repeat after cancer therapy including for recurrence (n=29 (27%)), first sample was adequate for cytological analysis but not enough for further molecular and genetic tests (n=13(12%)), first sample was adequate for cytological analysis but further nodal sampling required after MDT (n=7(6.6%)), other indication (n=17 (16%)) and inability to complete test (n=1 (0.9%)).

In terms of final pathology, 48 (45%) had malignant cytology, 6 (6%) were non-diagnostic and 52 (49%) were benign.
Conclusion In an academic centre with a designated national cancer service, our rate of repeat EBUS sampling is statistically increasing. Our repeat sampling was well within international national quality indices. The use of ROSE was associated with reduced requirement for repeat procedures. The complexity of cancer therapy is hypothesised to be the main driver of this increase.

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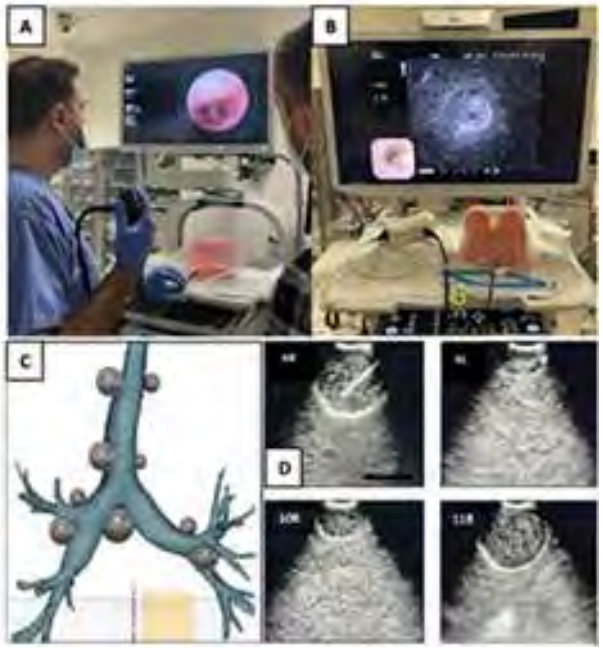
P8 TRANSFORMING TRAINING IN LUNG CANCER DIAGNOSTICS: EVALUATION OF NOVEL PATIENT-DERIVED LUNG PHANTOMS

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Introduction and Objectives Both endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) and navigational bronchoscopy are increasingly established diagnostic and staging procedures in patients with lung cancer. However, training in the UK remains largely apprenticeship-based with variable procedural exposure and experience. Simulation offers a safe environment for deliberate practice, with potential downstream implications on procedural and patient outcomes, but widespread adoption is limited by cost and accessibility. In this study, we evaluated the realism, fidelity, and training utility of novel patient-derived lung phantoms for EBUS and peripheral bronchoscopy.
Methods Lung phantoms (OmioLabs, UK) are 3D-printed models derived from anonymised patient computerised tomography (CT) scan data (figure 1). Bronchoscopists with varying levels of experience assessed EBUS and peripheral bronchoscopy phantoms in a simulated environment. Individual anonymised feedback on anatomical realism, procedural fidelity, and educational value in simulation training was collected using a mixed-methods approach; quantitative responses via structured Likert-scale questions were analysed descriptively, and qualitative free-text responses underwent thematic analysis.
Results Respiratory consultants and trainees evaluated the lung phantoms for EBUS (n=12) and peripheral bronchoscopy (n=11), respectively. Mean scores exceeded 8.5/10 across all assessed domains. Participants particularly valued lymph node visualisation and tissue sampling realism in the EBUS phantom, and tactile feedback and anatomical realism in the peripheral bronchoscopy phantom. Overall educational utility was rated highly (EBUS: mean score 9.8/10, SD 0.4; peripheral bronchoscopy: mean score 9.8/10, SD 0.6).

Qualitative feedback highlighted opportunities to enhance lymph node appearances to better represent benign and malignant pathology, and to incorporate an upper airway and additional endobronchial pathology to broaden procedural training applications.
Conclusions Patient-derived 3D-printed lung phantoms were highly rated for realism and educational utility across EBUS-TBNA and peripheral bronchoscopy simulation. These findings support their potential role in accessible, standardised bronchoscopy training. Future studies will evaluate their role in competency-based bronchoscopy training pathways to address gaps in procedural training.



Abstract P8 Figure 1 Lung phantom in action: (A) demonstration of flexible bronchoscope in the lung phantom showing endoscopic view, (B) ultrasound image with concentric view obtained using radial EBUS of peripheral nodule within the lung phantom, (C) key mediastinal lymph node stations visualised using the computer-aided design tool incorporated into the EBUS phantom, and (D) ultrasound images of multiple mediastinal lymph nodes with transbronchial needle entry seen in lymph node station 4R (top left)

P9 AN EVALUATION OF EBUS-TBNA TRAINING AMONG UK RESPIRATORY TRAINEES

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Introduction Endobronchial ultrasound with transbronchial needle aspiration (EBUS-TBNA) is a key diagnostic and staging tool particularly important in thoracic malignancy. However, competency in EBUS is not mandatory within the UK respiratory training curriculum.[1] We aimed to evaluate current training exposure, competence, and confidence in EBUS among UK respiratory trainees.

Methods We conducted a national online survey of UK respiratory trainees over a three-month period. The survey, distributed via training programme directors and trainee networks, included 29 questions covering training exposure, procedural experience, competence, and confidence. Responses with >80% completion were analysed (n=164).

Results Most trainees (86.6%, n=142) reported that their EBUS-TBNA training had been primarily through hands-on exposure during clinical placements. However, only 7.9% (n=13) had received formal theoretical teaching, 15.9% (n=26) had access to simulation-based training, and 14.6% (n=24) had attended EBUS-related courses or workshops. Ten trainees (6.1%), including two final-year trainees (ST7), reported no EBUS training at all.

Access to EBUS lists during current placements at the time of the survey was inconsistent, 43.9% (n=72) attended lists once a month or less, while 21.3% (n=35) had no access to lists at the time of the survey.

Competence was limited, only 15.9% (n=26) of all trainees surveyed felt able to independently perform EBUS-TBNA. Among final-year (ST7) trainees, 70.3% (n=19/27) reported being unable to perform EBUS-TBNA independently.

Self-reported confidence scores across four key EBUS procedural domains were assessed against training exposure (table 1). Greater procedural experience was strongly associated with higher procedural confidence. Trainees who had performed ≥50 procedures or completed dedicated fellowships demonstrated significantly higher mean confidence scores across all four domains.

Conclusion EBUS training varies across the UK, and many trainees do not feel competent to perform independent procedures by completion of training. Limited access to procedures and a lack of structured training opportunities are key difficulties. Increasing procedural exposure, access to formal teaching and simulation, and developing a more standardised curriculum may improve trainee competence and confidence.

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P10 ASSESSMENT OF ENDOBRONCHIAL ULTRASOUND (EBUS) TRAINING EXPOSURE AND CONFIDENCE AMONG RESPIRATORY TRAINEES IN YORKSHIRE & HUMBER

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Introduction Endobronchial ultrasound (EBUS) is a key diagnostic tool in respiratory medicine, yet achieving procedural competency during training remains challenging due to variability in exposure and access to structured teaching. This study aimed to evaluate current access to EBUS training, procedural exposure, trainee confidence, and perceived barriers to competency among higher specialty respiratory trainees in the Yorkshire & Humber region.

Methodology A web-based cross-sectional survey was conducted over a four-month period (November 2025 to February 2026) and distributed to respiratory trainees across the region. Data were collected on training grade, institutional factors,

Abstract P9 Table 1 Trainee self-reported confidence scores in key EBUS domains, significance between groups defined as p< 0.05, calculated using an independent two-sided T-test

	MEAN OF TRAINEE REPORTED CONFIDENCE SCORES (0-10)			
	Handling the EBUS scope and intubating	Identifying individual lymph nodes of interest correctly	Sampling a Lymph node of interest	Managing complications
Performs EBUS once a month, less than once a month or never	6.2	4.3	4.7	3.6
Performs EBUS fortnightly or more frequently	8	6.4	6.6	5.4
p-value	<.001	<0.001	<0.001	<0.001
Fellowship involving EBUS practical training	8.67	7.9	7.8	7.2
No fellowship	6.59	4.7	5.12	3.9
p-value	<0.001	<0.001	<0.001	<0.001
Has performed 50 or more EBUS procedures	9	8	8.1	6.9
Has performed <50 EBUS procedures	6.1	4.1	4.5	3.4
p-value	<0.001	<0.001	<0.001	<0.001

EBUS exposure, confidence levels, and perceptions of training adequacy.

Results A total of 57 responses were analysed. Most respondents were ST4–ST7 trainees, with 56% working in tertiary centres. EBUS service provision varied, with 42% reporting three lists per week. Despite this, individual exposure was limited, with 51% attending only one list per month. While 63% had received hands-on training, only 14% reported formal theoretical teaching. Confidence was highest for intubation but significantly lower for lymph node visualisation and sampling. Although 96% of trainees expressed interest in achieving competency, only 42% believed they would achieve this during training. Key barriers included limited list availability, competition for cases, and lack of structured training.

Conclusion There is a clear mismatch between trainee demand and the current provision of EBUS training across the whole region, with many trainees' lacking sufficient exposure and structured educational support to achieve competency. Despite strong motivation to acquire EBUS skills, confidence remains variable, particularly in more complex aspects such as lymph node identification and sampling, highlighting gaps that extend beyond basic procedural technique. Increasing the number of EBUS lists alone is unlikely to address these issues without a more coordinated training approach.

P11 FACTORS INFLUENCING PATIENT TOLERANCE AND SAMPLE ADEQUACY OF EBUS PROCEDURES: A SINGLE-CENTRE RETROSPECTIVE STUDY

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Introduction and Objectives Endobronchial ultrasound (EBUS) is a key investigation for mediastinal staging and diagnosis of lung cancer. Procedural tolerance is essential to achieve adequate sampling, minimising the need for repeat or alternative invasive procedures. Poor tolerance may increase the need for further diagnostic procedures. Factors influencing tolerance and subsequent procedural burden remain incompletely understood. The aim of this study was to assess EBUS tolerance and examine associations with sedation, operator number and subsequent additional diagnostic procedures.

Methods A retrospective review of EBUS procedures performed at a tertiary respiratory centre from January to September 2025 was undertaken. Data collected included procedure tolerance, sedation dose, operator number, sample adequacy and further diagnostic procedures. Procedures were categorised as well tolerated or poorly tolerated and comparisons were made between groups. Statistical analysis was performed to assess associations between procedural tolerance, sedation requirements, operator number and the need for additional diagnostic investigations.

Results 92 EBUS procedures were analysed. 54 procedures (59%) were well tolerated, while 38 (41%) were poorly tolerated. Median doses of midazolam (4.0 vs 2.75 mg) and fentanyl (100 vs 75 mcg) were higher in poorly tolerated procedures. Neither midazolam dose (p=0.36), fentanyl dose (p=0.49), nor age (p=0.54) were independently associated with poor tolerance, suggesting sedation was reactive rather than causative.

Poor tolerance was linked to increased repeat EBUS (p=0.0004). Following EBUS, 48 patients required at least 1 further procedure, including repeat EBUS (n=20), EBUS under general anaesthesia (n=14), CT-guided biopsy (n=16), and bronchoscopy performed at the same sitting (n=12). Operator number did not influence tolerance (p=0.93), number of nodes sampled (p=0.72), or adequacy of pathology samples (p=0.66).

Conclusions Poor tolerance of EBUS was common and was associated with increased need for repeat EBUS and additional diagnostic procedures. Higher sedation doses were likely caused by, rather than the cause of, poor tolerance. Operator number did not influence tolerance, number of nodes sampled or pathology adequacy. Identifying patients at risk of poor tolerance may reduce repeat procedural burden, minimise the need for additional invasive investigations and improve efficiency within the diagnostic pathway.

P12 EVALUATION OF EBUS PROCEDURES PERFORMED IN A SINGLE SITE OVER FOUR YEARS AGAINST NHS EBUS STANDARD SPECIFICATIONS

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Introduction Endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) is an established minimally invasive technique for diagnosing and staging thoracic malignancy. The NHS EBUS Service Specification (2019)[1] defines quality standards relating to diagnostic performance, molecular adequacy, complication rates and service quality. This study evaluated the performance of a single-site EBUS service over four years against these national standards.

Methods Aretrospective evaluation of all EBUS procedures performed at a single centre between 2022–2025 was undertaken. Outcomes assessed included total procedures performed, pathological confirmation rate, molecular adequacy for biomarkers used in treatment planning (EGFR, ALK, ROS1 and PD-L1), rates of NSCLC-NOS diagnoses, repeat biopsy requirements and major complications. Annual performance was compared with NHS EBUS service standards.

Results A total of 197 EBUS procedures were performed during the study period (see table 1), ranging from a number of 39 to 63 EBUS cases annually. Pathological confirmation rates ranged from 85.19–97.67%, meeting the NHS target (>90%) in 2024 only.

Molecular adequacy generally demonstrated strong performance. EGFR and PD-L1 adequacy consistently exceeded the national standard (>90%) throughout (92.30–94.44% and 93.00-96.40% respectively). ALK and ROS1 adequacy improved over time; both fell below target in 2022 (85.70%) but subsequently both achieved rates ≥92.3%.

NSCLC-NOS rates remained below the recommended threshold (<10%) in 2022 and 2023 but increased thereafter, reaching 12.90% in 2024 and remaining above target in 2025. Repeat biopsy rates improved substantially during the evaluation period, reducing from 14.30% in 2022 to rates below the national threshold (<10%) in later years. No major complications occurred at all throughout the study period.

Conclusion This single-site EBUS service demonstrated strong overall performance with consistently high molecular adequacy, low complication rates and improving repeat biopsy requirements. Although pathological confirmation rates and certain quality indicators did not consistently achieve NHS standards throughout the study period, improvements were observed over time. Future work should focus on improving diagnostic

Abstract P12 Table 1 EBUS procedures performed over 4 years in a single site against NHS EBUS service standards 2019

Year	Procedures	Pathological Confirmation Rate	EGFR	ALK	ROS-1	PDL1	NSCLC-NOS	Cases Requiring A Repeat Biopsy	Major Complications
2022	48	85.70%	93.33%	85.70%	85.70%	93%	3.20%	14.30%	0%
2023	63	85.19%	94.44%	94.74%	94.44%	96.30%	8.11%	10.00%	0%
2024	47	97.67%	92.80%	93%	93%	96.40%	12.90%	2.30%	0%
2025	39	89.66%	92.30%	92.30%	92.30%	95.50%	12.50%	5.10%	0%

rates and reducing NSCLC-NOS cases whilst maintaining the standards currently met and patient safety.

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P13 MANAGEMENT OF MODERATE/LARGE PERICARDIAL EFFUSION IN PATIENTS WITH LUNG CANCER IN A DISTRICT GENERAL HOSPITAL

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10.1136/thorax-2026-BTSabstracts.175

Introduction and Objectives Pericardial effusion (PcE) is a rare complication of advanced-stage lung cancer with incidence <3 months associated with a poor prognosis. PcE presents with progression of lung cancer and requirement for intervention with survival <6 months.[1] Our aim is to seek adverse prognostic signs in lung cancer patients with moderate/large pericardial effusion at diagnosis and during systemic active treatment (SACT) in an observational study.

Methods Retrospective review of 19 lung cancer patients with moderate/large PcE from May 2015 to 2026. Lung cancer diagnosis, staging and treatment obtained from clinic correspondence, discharge summaries, lung MDT outcomes, CT imaging, echocardiogram and histology/cytology reports on Oracle Health. Data collected on mean time from presentation of pericardial effusion to death.

Results Mean age was 64.1 (47–85), M:F 11/8. 18 (94.7%) with stage 4 disease. Incidence of 1%. 17 patients with adenocarcinoma (included 7 from pericardial fluid: 5 at diagnosis and 2 on SACT) and 2 small cell carcinoma all had histological diagnosis at other sites. Survival for 15 adenocarcinoma vs 2 small cell carcinoma were 132 vs 283 days. Adenocarcinoma at diagnosis with positive pericardial cytology survived 98 days vs 156 days for those without. Longer survival in 3 never smokers (>226 days); 9 ex-smokers died after 119 days. 13 patients with PcE at diagnosis vs 4 PcE developed on SACT survived 110 vs 140 days. Pericardiocentesis, pericardial drain ± window for 9 patients vs 8 patients with monitoring survived 128 days vs 145 days. Adenocarcinoma with positive PDL1, EGFR and negative mutations had survival times of 108, 144 and 192 days. 2 patients died before treatment vs 6 best supportive care vs 9 SACT with survival times of 40, 79 and 222 days.

Conclusions Adverse prognostic signs for lung cancer patients with moderate/large pericardial effusion are positive pericardial cytology, smoking history, adenocarcinoma, positive PDL1 mutation and non-treatment with SACT. In view of its rare incidence, we propose a national registry of lung cancer patients with pericardial effusion to improve patient outcomes.

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P14 THE CHANGE IN THE PREVALENCE OF CENTRAL AIRWAYS OBSTRUCTION IN LUNG CANCER OVER 10 YEARS

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10.1136/thorax-2026-BTSabstracts.176

Introduction The prevalence of lung cancer presenting with central airways obstruction (CAO) has been dropping. We sought to update the prevalence of this phenotype of disease and understand trends.

Methods We combined previous audit datasets from 2014, 2019 and 2020, with 2023 and 2025 – using the same methodology. CT scans were retrospectively reviewed for all new lung cancer patients referred to the cancer pathway in each respective year. The central airways were reviewed to determine disease involvement in the trachea, main bronchi, bronchus intermedius and at LC2.

Results Between 2014 and 2020 – 10-14% of patients presented with CAO disease. In 2023 and 2025 this dropped to 7.5-7.7%. After adjusting for screening (which commenced August 2022) the prevalence was 8.4% and 9.8% respectively. In those participants who had undergone at least one screening scan, the prevalence of CAO was 1.7-2.3%.

An increase in the median age of patients was observed, with the median[IQR] age in 2023 and 2025 being 76[68-82] and 73 [67-78] years respectively, while between 2014 and 2020, where the median age was 71 years. Men accounted for 89% of patients in 2025, despite consistently having represented 45-55% of cases.

Stage IV disease at presentation accounted for 80% of cases, with the exception of 2020 where 46% of cases were Stage 3. Small cell lung cancer accounted for 12-14.5% and did not significantly over time. Half of patients each year consistently had a performance status of ≤2.

Using datasets from 2014 and 2019, the expected Stage estimates of CAO prevalence, showed the expected number of cases dropped by approximately 10 cases per year.

Conclusion Despite remaining a significant burden, the prevalence of CAO shows signs of decreasing. Some of this effect is due to the rise in the denominator of cases driven by Lung Cancer Screening, however the prevalence in non-screening pathways also shows signs of reducing. Further work to determine the drivers for this will be important, including the chagning epidemiology of lung cancer, the impact of the NOLCP, increased referrals through traditional pathways and lung cancer screening.

P15 PATIENTS WITH IMPAIRED VENTILATORY FUNCTION UNDERGOING POTENTIAL LUNG CANCER SURGERY. A PROPENSITY-MATCHED STUDY

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10.1136/thorax-2026-BTSabstracts.177

Introduction Despite advancements in minimally invasive resections, lung cancer patients, often elderly patients and with significant pulmonary comorbidities, are deemed unfit for surgery because of their poor ventilatory status. Although some studies have evaluated operative outcomes in high-risk lung resections, evidence remains limited regarding outcomes comparing these with their lower-risk counterparts. This study aimed to evaluate the early and long-term postoperative outcomes of patients undergoing lung resections for suspected lung cancer, comparing high-risk and non-high-risk individuals.
Methods We performed a retrospective analysis of a prospectively maintained database including patients who underwent uniportal VATS anatomical lung resection for suspected lung cancer between January 2017 and December 2024. High-risk status was defined as a ppoFEV1 or ppoTLCO ≤40%. Patients were compared with a non-high-risk cohort, and propensity score matching was used to minimise baseline differences.
Results Over an 8-year period, 2400 lung resections were carried out at our institution, out of which 161 consecutive cases comprised our sample. This was represented by 80 high-risk and 81 non–high-risk patients. Following PSM, 63 patients were included in each group. Median age was 68 years (62–73), with a slight female predominance (55%, n = 69). Analysis of early postoperative outcomes revealed that high-risk patients experienced a greater incidence of prolonged air leak (46% vs. 27%; P = 0.026), as well as extended durations of chest drain placement (4 [2–8] vs. 3 [1–7] days; P = 0.008) and hospital length of stay (6 [4–9] vs. 4 [2–6] days; P = <0.001). After matching, no major postoperative complications were observed. Thirty-day mortality did not differ significantly between groups (0.8% vs. 0%; P > 0.315). At a median follow-up of 31 months, overall survival was 81% (n = 102). When comparing both of our study groups, we observed a higher survival rate in the non-high-risk cohort (86% vs. 76%, log-rank P = 0.041) (figure 1).
Conclusion High-risk patients undergoing uniportal VATS anatomical lung resection experienced acceptable perioperative outcomes and low postoperative mortality. Although long-term survival was lower than in non-high-risk patients, careful multidisciplinary assessment and patient selection can allow surgery to be performed safely in appropriately selected individuals.

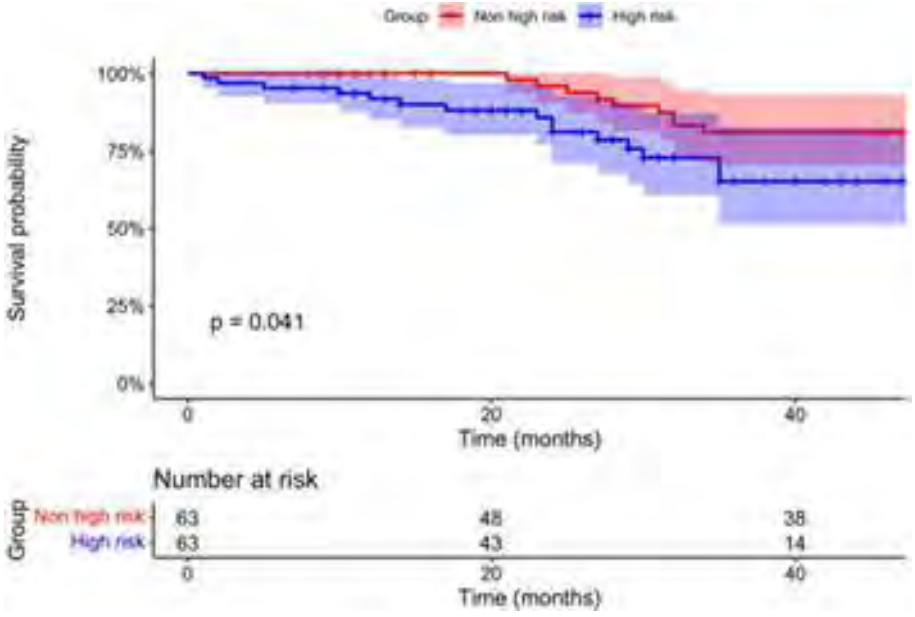
Cough: clinical profiles and novel interventions

P16 REFRACTORY CHRONIC COUGH PATIENTS WITH LOW OBJECTIVE COUGH FREQUENCY HAVE A SEVERE COUGH AND BURDEN

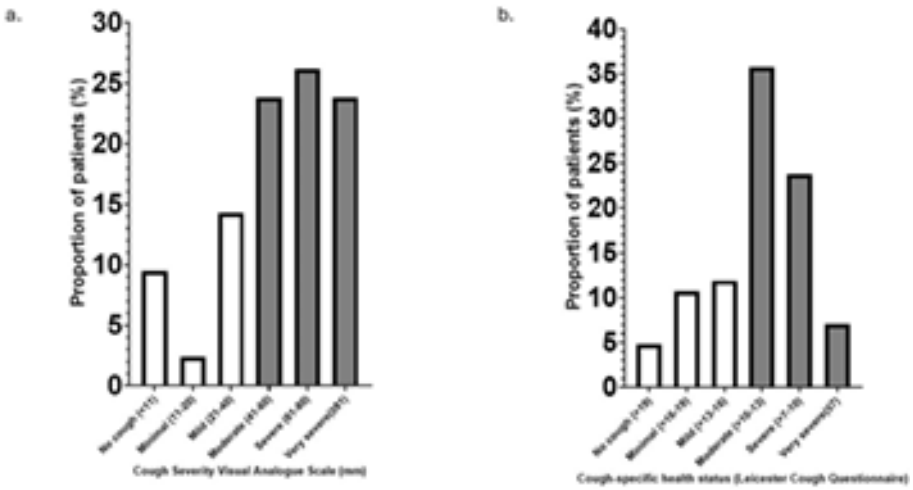
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Introduction Objective hourly cough frequency (CF) is often used as an inclusion criterion in anti-tussive clinical trials, and a threshold of >/=20 coughs hr^{-1} is commonly used. Such a threshold may exclude access for patients with a lower CF that may be burdened by cough. We assessed the prevalence, severity and impact of chronic cough in refractory chronic cough (RCC) patients with a CF <20 coughs hr^{-1}.
Methods Consecutive patients with RCC from a tertiary cough clinic underwent 24-hour objective CF monitoring with the



Abstract P15 Figure 1 Cough: clinical profiles and novel interventions



Abstract P16 Figure 1 Cough severity and health status in chronic cough patients with objective hourly cough frequency <20 cough.hr^{-1} (n=42) a. cough severity VAS category b. LCQ category

Leicester Cough Monitor. Participants also completed the cough severity visual analogue scale (VAS; 0-100 mm) and cough-specific health status Leicester Cough Questionnaire (LCQ; 3-21).
Results A total of 75 patients with RCC completed assessments; mean (SD) age 54 (13) years, 49 (65%) female, and median (IQR) duration of cough 36 (21-108) months. Adequate data capture was verified for all patients. Patients had geometric mean (SD) CF 14.3 (2.6) coughs hr^{-1}, and reported severe cough severity and moderate health status impairment; median (IQR) VAS 66 (45-82) mm and LCQ score 12.4 (8.5-14.0). Over half of the patients (n=42; 56%) had CF <20 coughs hr^{-1}, of which over two-thirds had moderate or higher cough severity (n=31/42; 74%) and health status impairment (n=28/42; 67%); median (IQR) VAS 51 (27-76) mm and LCQ score 12.5 (8.5–15.9), respectively (figure 1). In patients with moderate or more severe cough severity (n=59/75; 79%), over half of them (n=31; 53%) had CF <20 coughs hr^{-1}.
Discussion A large proportion, more than half, of patients with RCC had objective cough frequency <20 coughs hr^{-1}; however, most of these patients reported moderate or higher cough severity and health status impairment. An inclusion threshold of CF threshold at >/=20 coughs hr^{-1} may exclude the inclusion of patients in clinical trials who need and may respond well to novel therapies. There is a need to investigate other objective cough parameters such bouts, intensity and cough free time that may better align with patient burden.

P17 CONTROL OF DISEASE IN CHRONIC COUGH: DEVELOPMENT OF A CONCEPTUAL FRAMEWORK

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Introduction Control of disease is a commonly applied concept in respiratory diseases, and often guides clinical management, for example in asthma. Little is known about the paradigm of control of disease in chronic cough (CC). This mixed methods study aimed to develop a conceptual framework of disease control in CC through a patient survey and semi-structured interviews.
Methods Consecutive patients with CC were recruited from a specialist tertiary cough clinic. A total of 28 themes on control of disease in CC were previously identified from patient focus groups (n=14) and multi-disciplinary expert panels (n=18). Patients completed a survey by reporting the occurrence (0= no, 1= yes) and burden (1-5; higher score indicates worse burden) of each theme in the past 12 months. The product of the two ratings yielded a clinical impact score (0-5; higher scores indicate greater impact), and a score >3 was considered significant. Patients also underwent a semi-structured interview to identify any duplicate or redundant themes.
Results A total of 47 patients completed the survey and semi-structured interviews; median (IQR) age 64 (57-70) years, 37 (79%) female and 41 (87%) with refractory chronic cough. 22 (79%) of the 28 themes occurred in >50% of participants, and 12 (43%) out of 28 themes were identified as duplicate or redundant. A total of 6 out of the remaining 16 themes had significant clinical impact scores (>3; range 3.2-4.3) and no redundancy, which included coughing bouts, coughing frequently, inability to suppress cough, severe cough, frequent urge to cough and interference with activities.
Conclusion Our study identified key patient-focused themes to aid the construction of a conceptual framework in CC. In turn, the conceptual framework may facilitate the development of the first control of disease patient-reported outcome measure in CC.

P18 MICROBIOLOGICAL, PHYSIOLOGICAL AND INFLAMMATORY PROFILES OF EXCESSIVE DYNAMIC AIRWAY COLLAPSE

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Background Excessive dynamic airway collapse (EDAC) causes persistent cough, impaired airway clearance and recurrent lower respiratory infections. Its impact on microbiological, physiological and inflammatory changes is unknown, limiting treatment strategies.

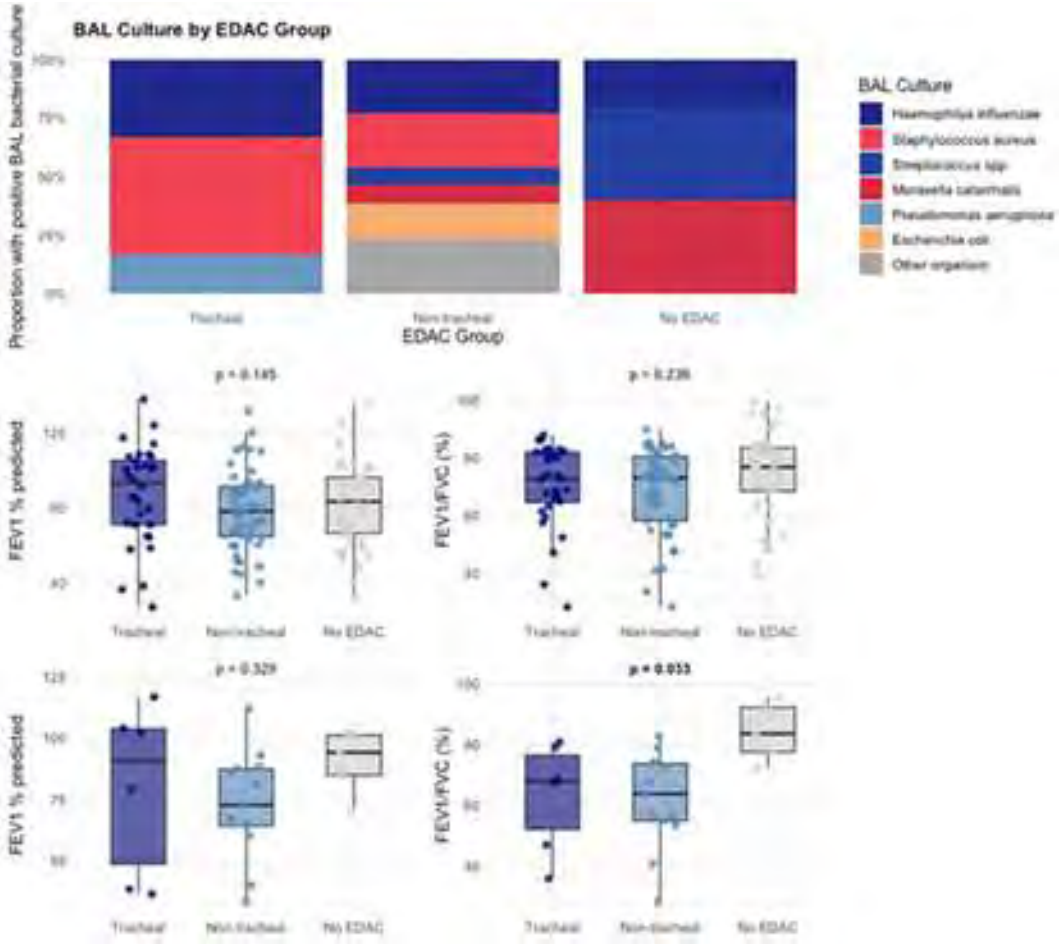
Aim To characterise the clinical, microbiological, physiological and inflammatory features of EDAC and identify predictors of disease.

Methods We retrospectively analysed patients with asthma or suspected asthma and poor treatment response who underwent flexible bronchoscopy at a tertiary UK respiratory centre between 2020 and 2026. Tracheal and non-tracheal EDAC were defined as ≥75% luminal collapse on forced expiration at the trachea or at either/both main bronchi, respectively. Bronchoalveolar lavage (BAL) provided cell differentials and microbiological cultures. Demographics, clinical characteristics, radiology, lung function and longitudinal inflammatory biomarkers were collected. Predictors of EDAC were identified by univariable logistic regression, with optimal cut-offs from the Youden index.

Results Of 135 patients, 85 (63%) had EDAC and 50 (37%) did not. Median luminal collapse was 90% (IQR 80–100%) in those with EDAC. The EDAC group was older (58 vs 49 years, p=0.009) but similar in sex (female 77% vs 80%, p=0.84), BMI (33 vs 29, p=0.18), concurrent asthma diagnosis (58% vs 54%, p=0.86), daily oral corticosteroid use (24% vs 18%, p=0.61), biologic use (40% vs 41%, p>0.99) and prophylactic azithromycin (15% vs 14%, p>0.99).

Positive BAL cultures occurred in 23 patients (18%), with distinct microbiological profiles across tracheal, non-tracheal and no-EDAC groups. Among culture-positive patients, EDAC was associated with greater airflow obstruction than no EDAC (figure 1). CT findings (bronchiectasis, emphysema, bronchial wall thickening, mucus plugging) and blood/airway biomarkers (FeNO, CRP, BAL granulocyte percentages) did not differ. However, lower blood eosinophils (OR 0.21, 95% CI 0.07–0.61) and older age (OR 1.06, 95% CI 1.02–1.10) were associated with tracheal EDAC, with optimal cut-offs of $<0.08 \times 10^9$/L and >55 years, respectively.

Conclusion Culture-positive tracheal EDAC may represent an important group with poorer outcomes, distinguishable by older age, low blood eosinophils and differing microbiological profiles. Prospective studies of whether identifying and treating EDAC improves infection burden and lung function are needed.



Abstract P18 Figure 1 BAL bacterial culture and lung function by EDAC group. Upper panel: proportion of positive BAL bacterial cultures by organism across three EDAC groups (Tracheal + Non-tracheal, No EDAC). **Middle panels**: FEV1 % predicted and FEV1/FVC (%) at bronchoscopy across groups in all patients. **Lower panels**: FEV1 % predicted and FEV1/FVC (%) in BAL culture-positive patients only. P- values calculated using Kruskal-Wallis test

P19 EVALUATING CLINICAL DELIVERY COSTS OF A VIRTUAL GROUP PHYSIOTHERAPY INTERVENTION (VIP-COUGH) VERSUS TRADITIONAL ONE-TO-ONE COUGH SUPPRESSION THERAPY

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Introduction Increasing demand for respiratory physiotherapy outpatient services, combined with financial pressures within the National Health Service (NHS) in the UK (UK) highlights the need for cost-effective delivery models. To address this, we developed a Virtual Physiotherapy Group Intervention for Chronic Cough (ViP-Cough), which has previously demonstrated improvements in cough severity and self-management confidence.[1] This evaluation compares the clinical delivery cost of traditional one-to-one cough control physiotherapy with ViP-Cough.

Methods Traditional face-to-face cough control physiotherapy comprised of a 1-hour initial assessment and four 30-minute follow-up sessions (total 3 hours). ViP-Cough consisted of a 30-minute initial assessment, a 1.5-hour virtual group session for 10 patients, and a 30-minute discharge assessment. Cost estimates were derived using the NHS Agenda for Change pay scale for a midpoint Band 7 physiotherapist (£25.71 per hour). Employer oncosts were applied, including National Insurance at 14% and Superannuation at 15%, resulting in a total employment cost of £33.17 per hour. A further 25% uplift for organisational overheads was added, giving a full NHS cost estimate of £41.46 per hour.

Results The estimated full NHS cost per patient for traditional Physiotherapy was £124.38. For ViP-Cough, the cost per patient was £47.68, representing a £76.71 reduction (62% lower). Savings were primarily achieved through shared clinician time during group sessions.

Conclusion ViP-Cough offers substantial potential cost efficiencies compared with traditional one-to-one delivery, without requiring additional resources. Further research is needed to establish both the clinical and cost-effectiveness of ViP-Cough.

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P20 PREVALENCE AND CHARACTERISTICS OF BREATHING PATTERN DISORDERS IN ADULTS WITH REFRACTORY CHRONIC COUGH OR INDUCIBLE LARYNGEAL OBSTRUCTION: A SYSTEMATIC REVIEW

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Introduction and Objectives Breathing Pattern Disorders (BPD) are commonly associated with upper airway disorders, including Refractory Chronic Cough (RCC) and Inducible Laryngeal Obstruction (ILO). However, the prevalence and clinical characteristics of BPD within these populations is unclear, making it difficult to determine how commonly BPD occurs and which features are most relevant to assess, impacting accurate clinical diagnosis and management.

Methods A systematic search of the literature following PRISMA methodology was completed to answer the question 'What is the reported prevalence and characteristics of breathing pattern disorders in adults with RCC or ILO?'. Studies were included if they reported data on the prevalence and/or clinical characteristics of BPD in adults (^{3}18yrs) within the target populations. Following de-duplication, 151 publications were identified, with four meeting the eligibility criteria. Methodological quality was assessed using the Joanna Briggs Institute Critical Appraisal Checklist for Studies Reporting Prevalence Data.[1]

Results BPD was reported to be a common and clinically relevant comorbidity in RCC and ILO cohorts, with 73% of RCC patients and 69–89% of ILO patients demonstrating at least one breathing abnormality. Across studies, abnormalities in breathing region (thoracic vs abdominal) and route (oral vs nasal) were more consistently reported than abnormalities in breathing rate. Similar breathing pattern characteristics were observed across RCC and ILO cohorts, suggesting overlapping physiological features. In contrast, findings in exercise-induced laryngeal obstruction (EILO) were less distinct, with similar breathing patterns also observed in non-EILO groups. However, interpretation is limited by the small number of included studies and heterogeneity in definitions and assessment of BPD.

Conclusions BPD appears to be a common comorbidity in RCC and ILO; however, interpretation is limited by the small evidence base and lack of standardised assessment approaches. Despite this, the included studies identified clinically relevant BPD characteristics and suggest a bidirectional relationship between abnormal breathing patterns and laryngeal dysfunction influenced by precipitating factors. Diagnostic overlap between BPD, RCC, and ILO may complicate assessment and management, particularly in centres without access to laryngoscopy. Clinicians should consider comprehensive breathing assessment within routine upper airway assessment.



Abstract P20 Figure 1

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P21 EXPERIENCE OF PRESCRIBING GEFAPIXANT IN A UK COUGH CLINIC

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Introduction Gefapixant, a P2X3 antagonist, is licensed for the treatment of chronic cough (CC) and is currently marketed in Japan and Europe. Randomised controlled trials have demonstrated its efficacy, but real-world data remain limited.

Objective To evaluate the real-world effectiveness, tolerability, and patient-reported outcomes of gefapixant in UK patients with CC.

Methods Patients were prescribed gefapixant 45 mg twice daily in routine clinical practice. Follow-up assessments were conducted one week after treatment initiation, and subsequently at monthly intervals. Subjective cough outcomes were recorded at baseline and follow-up using validated patient-reported outcome measures.

Results Sixty patients were included (47 females, 78.3%), with a median (interquartile range) age of 68.0 (61.2 to 73.8) years. Thirty-five patients (58.3%) perceived treatment benefit, with most responders experiencing improvements within one week that was sustained thereafter. Significant reductions were observed in HARQ scores (mean [standard deviation]: 45.8 [13.0] vs. 22.6 [16.5]; $p = 0.005$), alongside significant improvements in LCQ scores (8.7 [3.2] vs. 17.3 [3.3]; $p = 0.002$) in a week. Urge to cough (7.7 [2.4] vs. 2.7[2.2]; $p < 0.001$) and cough severity (7.7 [1.8] vs. 2.5 [2.2]; $p < 0.001$), measured using the modified Borg scale, also improved. Improvements were also observed in intensity ($p = 0.031$) and breathless ($p = 0.02$) domains of the Symptom Assessment Score (figure 1). Taste disturbance was reported in 37 patients (61.7%), including 26 responders (70.3%).

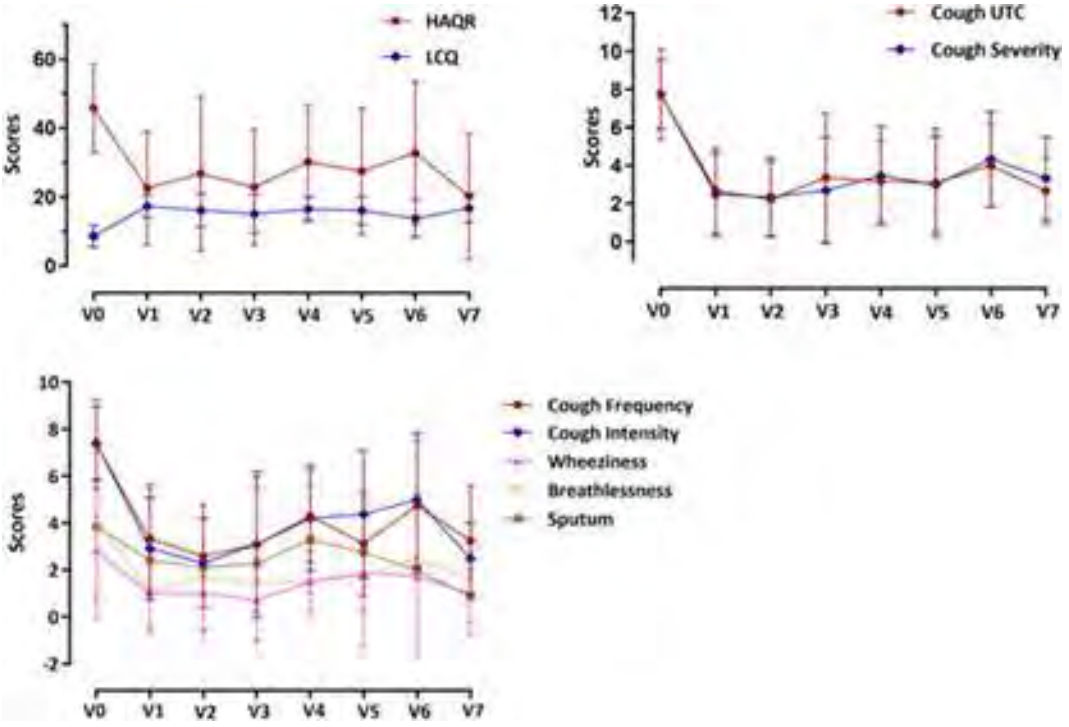
Conclusions In UK real-world practice, more than half of patients with CC reported a rapid and sustained response to gefapixant. Treatment effects and adverse events were consistent with findings from randomised trials, supporting the clinical effectiveness of P2X3 inhibition in CC.

P22 DAY-TO-DAY VARIABILITY OF OBJECTIVE COUGH MONITORING IN FIBROTIC INTERSTITIAL LUNG DISEASE (FILD)

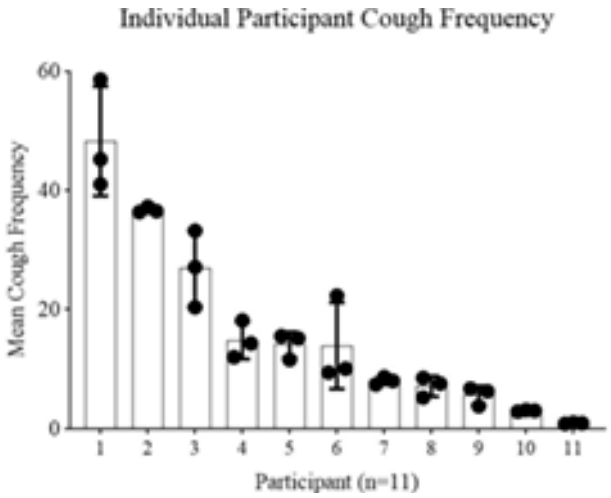
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Introduction Cough is common in fILD and significantly impacts patient quality of life. Ambulatory cough monitoring (ACM) objectively assesses cough frequency (CF) and temporal cough patterns in clinical and research settings and is increasingly used as a research outcome measure. Advances in artificial intelligence enable extended-duration CF monitoring but raise questions regarding acceptability and cost-effectiveness. Cough variability is described in chronic refractory cough but is not characterised in fILD and the duration of ACM required for reliable, representative estimates of individual cough burden is unknown. The aim of this study was to investigate day-to-day variability of objective CF in patients with fILD.



Abstract P21 Figure 1 Changes in HARQ, LCQ, urge to cough, cough severity, and symptom assessment score in responders



Abstract P22 Figure 1 Individual participant mean cough frequency (coughs/hour). Error bars represent standard deviation. Dots represent single day measurement

Methods Participants with fILD were prospectively recruited from two tertiary ILD centres. They underwent three consecutive 24-hour periods of objective CF monitoring with the Leicester Cough Monitor (LCM). Variability was characterised using average absolute deviation (AAD; mean absolute deviation of each day's values from the individual mean). Reliability was assessed using intraclass correlation coefficient (ICC) derived from a mixed-effects repeated-measures model. Agreement of Day 1 (24-hour) CF with 2-day and 3-day mean CF was evaluated using Bland-Altman analysis.

Results A total of 11 participants completed three full consecutive 24-hour recording periods. The three-day geometric mean CF was 9.99 (95% CI 4.61-21.63) coughs/hour (median 13.93, IQR 5.60-26.90) with day-to-day variability shown in figure 1. There was no significant difference in CF across recording days ($p=0.27$). Mean AAD was 2.19 ±2.36 coughs/hour (median 1.27, IQR 0.38-4.33). The average relative AAD (AAD/mean) was 13.89 ±10.86% (median 14.27%, IQR 5.53-17.84%) suggesting a single 24-hour recording would deviate from overall cough burden by approximately 14%. ICC was 0.96 (95% CI 0.90-0.99). Bland-Altman analysis demonstrated minimal bias between Day 1 CF and the 3-day mean CF (bias 0.66 coughs/hour, limits of agreement -6.45 to 7.78 coughs/hour), and between 2-day and 3-day mean CF (bias 0.70 coughs/hour) with narrower limits of agreement (-1.99 to 3.38 coughs/hour).

Conclusion There is some day-to-day objective CF variability (2 coughs/hr or 14% change) in fILD. Further studies are needed to determine if this is clinically significant and to establish the optimal duration of cough monitoring.

P23 ROLE OF PHYSIOTHERAPY WITHIN A CHRONIC COUGH SERVICE – REVIEW OF REFERRALS

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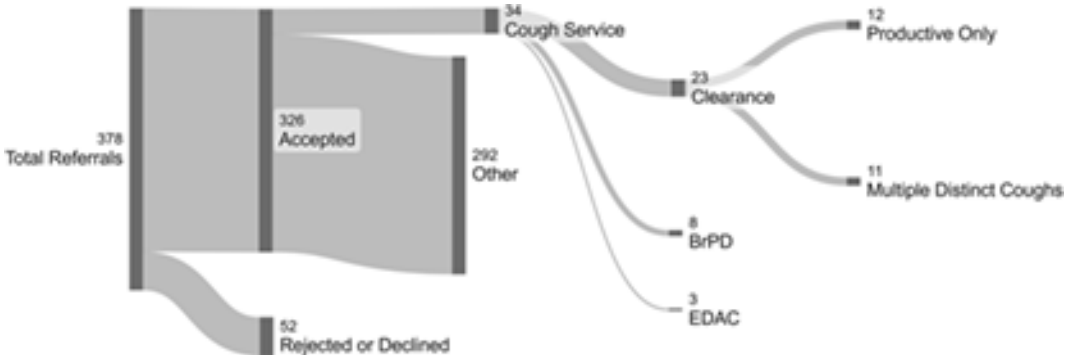
Introduction Within our regional severe asthma and airways service, specialist physiotherapists are core members of the MDT. The regional chronic cough service sits within this structure. Guidelines on chronic cough highlight treatable traits, many of which are managed by physiotherapy. Despite this, there is no recommendation for physiotherapy within this area, beyond access to services (Parker, 2023). In our services, physiotherapy within chronic cough is not funded. We had seen a rise in referrals from the chronic cough team to physiotherapy and aimed to review reasons for referral in order to provide evidence for the role of physiotherapy as a core part of a chronic cough service.

Methods We conducted a retrospective review of referrals from January 2024 to December 2025, excluding rejected cases and those who declined input. Cough service referrals were identified and categorised by primary indication: Excessive Dynamic Airways Collapse (EDAC), chest clearance, or suspected breathing pattern disorder (BrPD). Clearance referrals were subdivided into productive cough only, or multiple distinct coughs; a productive chest cough plus a separate dry upper airway cough.

Results Of 378 referrals made to the airways physiotherapy service, 34 (10.4%) originated from the chronic cough service.

Of these 34, 3 (8.8%) referrals were classified as EDAC, 8 (23.5%) were for suspected BrPD, and 23 (67.6%) were for clearance. The clearance category was further analysed; 12 (52.2%) had productive cough only, while the remaining 11 (47.8%) had multiple distinct coughs (figure 1).

Outcomes From this data, there is evidence to suggest that within a chronic cough service skilled physiotherapy support is needed for the management of sputum clearance, BrPD, and



Abstract P23 Figure 1

EDAC. Further analysis and review is needed to explore number of physiotherapy contacts, treatment provided, and outcomes to fully evaluate the role of physiotherapy within a chronic cough service and inform future planning for service provision.

REFERENCE

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P24 CLINICAL DIAGNOSTIC CONFIDENCE AND TREATMENT PATTERNS IN COUGH MANAGEMENT: A CROSS-SECTIONAL SURVEY AMONG PHYSICIANS

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Introduction and Objective As a common disease, cough brings diagnostic and therapeutic challenges in clinical practice. Here, we reported the results of a cross-sectional survey for diagnostic confidence, treatment patterns, and effectiveness from physicians across the tiered system in China.

Methods The survey was conducted from Aug 2025 to Apr 2026. Responses of 3,279 physicians represent 258 primary, 254 secondary, and 462 tertiary hospitals in China. Group comparisons used χ^2/Fisher's exact (categorical) and ANOVA/Kruskal-Wallis (continuous) tests.

Results Among 3,279 physicians, 54.7% were from tertiary hospitals. The survey data showed that PIC was the most common cough etiology, with a frequency rating of 4.1±1.0 (from 1 = most rare to 5 = most common), followed by CVA (3.7±0.9) and AC (3.6±0.9), with EB and GERC rated as less common (both 3.3). Tertiary hospital physicians had the highest diagnostic confidence score for all etiologies (PIC: 4.1±0.9, CVA: 3.9±0.8, EB: 3.7±0.9, AC: 3.8±0.9, GERC: 3.7±0.9), compared with secondary (4.0/3.9/3.6/3.7/3.5) and primary hospitals (3.9/3.7/3.5/3.6/3.6) (PIC/EB: P = 0.002; CVA/AC: P <0.001; GERC: P = 0.008). They also reported high scores for diagnostic method accessibility, including FeNO test (3.9±1.1 vs. 3.5/3.2 [secondary/primary]), sputum eosinophil count (3.5±1.2 vs. 3.4/3.3), 24-h esophageal pH monitoring (3.2±1.2 vs. 3.0/2.9), and bronchial provocation test (3.7±1.2 vs. 3.5/3.1) (all P <0.001). Firstchoice regimens for each etiology were similar across hospital levels, but 64.7% of tertiary hospital physicians added traditional Chinese medicine more often to reduce Western medicine side effects, significantly higher than primary (50.9%) or secondary hospitals (60.8%, P <0.001). Regarding treatment effectiveness, tertiary hospital physicians reported a higher rate of >90% effectiveness for CVA (11.7%) than secondary (8.6%) and primary (7.4%) hospitals (P <0.001). During follow-up visits, they generally tend to adjust dosages (e.g., dose reduction) rather than modify the overall treatment regimen (all P <0.001).

Conclusion Cough management varies by hospital level, with tertiary hospitals showing better diagnostic confidence and treatment effectiveness, underscoring the value of standardized hierarchical care.

P25 ASSOCIATION BETWEEN COMMON COMORBIDITIES AND DISTRIBUTION OF CHRONIC COUGH ETIOLOGIES IN CHINA: AN INTERIM ANALYSIS OF A CROSS-SECTIONAL CLINICIAN SURVEY

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Purpose Common chronic cough (CC) etiologies include post-infectious cough (PIC), cough-variant asthma (CVA), eosinophilic bronchitis (EB), allergic cough (AC), and gastroesophageal reflux cough (GERC). Comorbidity is associated with

Abstract P25 Table 1 Association between comorbidities and etiology commonness scores

Exposure	Outcome	Linear regression β (95% CI)	P value	Ordinal logistic aOR (95% CI)	P value
COPD	PIC	0.019 (-0.051, 0.089)	0.6	1.049 (0.907, 1.214)	0.52
Asthma	CVA	0.141 (0.078, 0.204)	<0.001	1.401 (1.209, 1.624)	<0.001
Asthma	EB	0.138 (0.069, 0.208)	<0.001	1.364 (1.178, 1.578)	<0.001
Chronic sinusitis	AC	0.082 (0.021, 0.144)	0.009	1.257 (1.094, 1.444)	0.001
GERD	GERC	0.364 (0.298, 0.431)	<0.001	2.103 (1.835, 2.411)	<0.001
Comorbidity burden index	PIC	0.012 (-0.015, 0.038)	0.386	1.033 (0.976, 1.093)	0.261
	CVA	0.011 (-0.013, 0.035)	0.362	1.038 (0.981, 1.097)	0.196
	EB	0.048 (0.022, 0.074)	<0.001	1.107 (1.047, 1.170)	<0.001
	AC	0.011 (-0.013, 0.036)	0.377	1.027 (0.972, 1.085)	0.343
	GERC	0.065 (0.037, 0.092)	<0.001	1.139 (1.078, 1.203)	<0.001

Note Values are fully adjusted associations between comorbidities or comorbidity burden index and clinician-rated chronic cough etiology scores. Linear regression β indicates mean score difference; ordinal logistic aOR indicates odds of a higher etiology commonness score. COPD, chronic obstructive pulmonary disease; GERD, gastroesophageal reflux disease; PIC, post-infectious cough; CVA, cough-variant asthma; EB, eosinophilic bronchitis; AC, allergic cough; GERC, gastroesophageal reflux-related cough; CI, confidence interval; aOR, adjusted odds ratio.

CC, but quantitative links to specific etiologies are lacking. This study investigates this association from clinicians' perspective.
Methods This multicenter, cross-sectional survey was conducted among clinicians across 16 provinces from August 12, 2025. Interim data collection ended on April 29, 2026. Clinicians reported common comorbidities in CC and rated five CC etiologies on a 1–5 commonness scale. A comorbidity burden index was calculated. Analyses included t-test, Spearman correlation, multivariable linear regression, and ordered logistic regression as sensitivity analysis.
Results Overall, 3142 clinicians were included. Group comparisons showed higher CVA and EB scores among clinicians reporting asthma, higher AC scores among those reporting chronic sinusitis, and higher GERC scores among those reporting GERD. In adjusted regression models, the strongest association was observed between GERD and GERC (β=0.364, 95% CI 0.298 to 0.431, P<0.001; aOR=2.103, 95% CI 1.835 to 2.411, P<0.001). Asthma was associated with higher CVA (β=0.141, 95% CI 0.078 to 0.204; aOR=1.401, 95% CI 1.209 to 1.624) and EB scores (β=0.138, 95% CI 0.069 to 0.208; aOR=1.364, 95% CI 1.178 to 1.578; all P<0.001). Chronic sinusitis was associated with higher AC scores (β=0.082, 95% CI 0.021 to 0.144, P=0.009; aOR=1.257, 95% CI 1.094 to 1.444, P=0.001). Comorbidity burden index showed significant associations with EB (β=0.048, 95% CI 0.022 to 0.074, P<0.001; aOR=1.107, 95% CI 1.047 to 1.170, P<0.001) and GERC (β=0.065, 95% CI 0.037 to 0.092, P<0.001; aOR=1.139, 95% CI 1.078 to 1.203, P<0.001), but not with PIC, CVA or AC.
Conclusion Clinically logical associations exist between common comorbidities and CC etiology scores, particularly for GERD with GERC, asthma with CVA and EB, and chronic sinusitis with AC. Higher comorbidity burden was mainly associated with higher EB and GERC scores. While identifying comorbidities may aid diagnosis, multimorbidity increases complexity, warranting further research on diagnostic pathways.

P26 FROM COMMUNITY CLINICS TO TERTIARY CENTERS: HOW HOSPITAL HIERARCHIES SHAPE THE CHARACTERIZATION OF COUGH MANAGEMENT

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Introduction Cough is a common reason patients seek medical care, carrying a significant public health burden in China. The tiered medical services system is established for better resources allocation and patient outcomes. We reported results of a survey characterizing knowledge, attitude, and practice among physicians across the tiered system.
Methods The survey was conducted in August 2025 - April 2026. Responses of 2449 physicians represent 276 community hospitals, 184 secondary and 288 tertiary hospitals in East China. ANOVA and chi-square tests are used.
Results Tier-3 hospitals have more young (aged 25-35) and senior physicians (aged 55+), consisting of 43% respiratory and 28.2% ENT doctors, while 84.5% physicians in community hospitals are general practitioners. PIC (63.1%), CVA (14.3%) and UACS (13.1%) are three most frequent cough types. Physicians in tertiary centers saw more patients with subacute cough (33.1% vs 27.8%), less acute cough (60.5% vs 67.5%) and similar chronic (6.4% vs 4.7%) cough. Their patients have more asthma, chronic sinusitis, but fewer COPD, GERD and CVD. About 66% of them consider age 16-59 the most prevalent subacute and chronic cough population, while 59% of community physicians choose 60+ population. All physicians pay attention to the duration and severity of comorbidities, but community physicians are less concerned about etiology, prior treatment and control status. Physicians have consensus weighing environment (75.5%), medication (85.2%) and emotion (56.6%) as keys to cough relief and urged more research for population with recurrent chronic cough (85.5%). More physicians in tertiary (69.4%) and secondary (70%) hospitals value highly on RWE studies, compared to 55.6% otherwise.
Conclusion Cough patients are different across tiered hospitals in China. Physicians demonstrate similar understanding in key knowledge, with gaps in other aspects.

P27 AGE-RELATED DIFFERENCES IN ETIOLOGY OF CHRONIC COUGH: AN INTERIM ANALYSIS OF A MULTICENTER CROSS-SECTIONAL CLINICIAN SURVEY IN CHINA

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Introduction The etiological spectrum of chronic cough may vary by age, yet clinicians' perceptions differ. This study investigated the perceived major etiologies across patients' age from clinicians' perspectives.
Methods This multicenter, cross-sectional survey was conducted among clinicians across 16 provinces in China from August 12, 2025. Interim data collection ended on April 29, 2026. Five etiologies were scored for commonness on an ascending 1–5 scale: post-infectious cough (PIC), cough-variant asthma (CVA), eosinophilic bronchitis (EB), allergic cough (AC), and gastroesophageal reflux-related cough (GERC). The patients were grouped by age. Analyses included ANOVA, Kruskal-Wallis tests, linear mixed models, and multivariable regression.
Results Of 3026 valid responses, PIC scored highest across groups (mean±SD: children 4.04±0.98; working-age adults 4.02±0.98; older adults 4.15±0.91). ANOVA showed significant age-group differences for AC, GERC, PIC, and EB, but not CVA. The overall age-by-etiology interaction was significant ($F = 8.04$, $P < 0.001$). Using children as a reference, multivariable regression showed working-age and older adults had lower AC scores (β=-0.138, P=0.040; β=-0.238, P<0.001) and lower GERC scores (β=-0.225, P=0.003; β=-0.327, P<0.001). EB scores slightly decreased across groups (3.44, 3.34, and 3.28); CVA showed no significant differences. Stratified analyses suggested some heterogeneity across clinician backgrounds but generally consisted with the main analysis.

Abstract P27 Table 1 Association of commonness scores for chronic cough etiologies across age groups

Aetiology	Age group	Mean ± SD	Adjusted ß (95% CI)	P value	ANOVA P	Kruskal-Wallis P
AC	Children	3.73 ± 0.93	Reference	—	<0.001	<0.001
	Working-age adults	3.61 ± 0.89	-0.138 (-0.269, -0.006)	0.04	—	—
	Older adults	3.50 ± 0.85	-0.238 (-0.371, -0.104)	<0.001	—	—
CVA	Children	3.83 ± 0.91	Reference	—	0.194	0.119
	Working-age adults	3.73 ± 0.88	-0.108 (-0.236, 0.021)	0.101	—	—
	Older adults	3.71 ± 0.83	-0.117 (-0.248, 0.014)	0.079	—	—
EB	Children	3.44 ± 0.99	Reference	—	0.043	0.02
	Working-age adults	3.34 ± 0.95	-0.046 (-0.180, 0.087)	0.497	—	—
	Older adults	3.28 ± 0.93	-0.087 (-0.222, 0.049)	0.209	—	—
GERC	Children	3.52 ± 1.04	Reference	—	<0.001	<0.001
	Working-age adults	3.32 ± 0.98	-0.225 (-0.372, -0.078)	0.003	—	—
	Older adults	3.20 ± 0.97	-0.327 (-0.476, -0.178)	<0.001	—	—
PIC	Children	4.04 ± 0.98	Reference	—	0.001	0.003
	Working-age adults	4.02 ± 0.98	0.007 (-0.135, 0.149)	0.926	—	—
	Older adults	4.15 ± 0.91	0.123 (-0.022, 0.267)	0.096	—	—

Note Values are clinician-rated commonness scores on a 1–5 scale. Children were the reference group in adjusted regression models. Adjusted β values indicate mean differences versus children, adjusted for perceived sex differences in etiology, predominant cough type, dry/wet cough proportion, and regional environmental factors. ANOVA and Kruskal-Wallis tests assessed overall age-group differences. '—' indicates not applicable. AC, allergic cough; CVA, cough-variant asthma; EB, eosinophilic bronchitis; GERC, gastroesophageal reflux-related cough; PIC, post-infectious cough.

Conclusion Clinician-perceived etiology scores for chronic cough differ significantly across patient age, with AC and GERC scores declining as age increases, while PIC remains highly scored in all groups.

P28 INCIDENCE AND PREDICTORS OF FUTURE ASTHMA DIAGNOSIS IN SUBJECTS WITH COUGH

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Background Asthma is one of the most common background disorders for chronic cough but yet it is unknown which proportion of subjects with current cough will receive new asthma diagnosis in the future.
Research Question How common is incident asthma diagnosis in people with current cough? Which factors and cough characteristics predict a future asthma diagnosis in this population?
Study Design and Methods A prospective cohort of 2992 people (mean age 63.1 years, 68.3% women) with baseline questionnaires in 2017 or 2021. Altogether 209 participants (7.0%) had cough duration less than three weeks, 613 (20.5%) cough more than three weeks, and 519 (17.3%) cough more than 8 weeks. Characteristics predicting asthma diagnosis in 2025 were investigated with univariable and multivariable Cox regression analyses.
Results 88 (2.9%) received asthma diagnosis during follow-up. Incidence was higher in those with any current cough (incidence 6.3%), prolonged cough (6.7%), or chronic cough (7.3%), compared to those without cough (1.7%). Median time to diagnosis was 23.4 months in participants with current cough and 26.0 months in those without, p=0.72. Wheezing (adjusted hazard ratio (aHR) 3.31, 95% confidence interval (CI) 2.14-5.12), current cough (aHR 2.51, 95%CI 1.60-3.93), allergy (aHR 2.02, 95%CI 1.24-3.31) and chronic rhinitis (aHR 1.65, 95%CI 1.02-2.66) predicted future asthma diagnosis. Among participants with current cough, cough characteristics predicting asthma diagnosis were the number of cough triggers and impaired cough-related quality of life based.
Interpretation Any current cough seems to be a significant risk factor for future asthma diagnosis. Currently this doesn't seem to be well recognized as the time to diagnosis among subjects with current cough was not shorter than that among subjects without cough. Cough triggers and cough-related quality of life should be considered when evaluating a cough patient and the risk of future asthma diagnosis.

P29 PHYSICIANS' PERCEPTIONS OF FACTORS INFLUENCING COUGH SYMPTOM RELIEF: A CROSS-SECTIONAL SURVEY ACROSS CHINA'S TIERED HEALTHCARE SYSTEM

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Introduction and Objectives Cough is a common respiratory symptom whose complex etiology challenges differential

diagnosis and treatment selection. This study investigates the key factors influencing cough relief from physicians' perspective across the tiered system in China.

Methods This cross-sectional survey was conducted from August 2025 to April 2026, collecting data from 3279 physicians across 258 primary hospitals, 254 secondary hospitals, and 462 tertiary hospitals.

Results Of all physicians, 54.7% practiced in tertiary hospitals. Physicians across all hospital tiers consistently identified medication types, underlying diseases and environmental factors as the three key factors affecting cough relief, with medication types ranking first. Notably, physicians in primary hospitals paid less attention to medication adherence (49.1%, vs. 66.4% and 60.2% in secondary and tertiary hospitals) and individual allergic diathesis (72.1%, vs. and 77.9% and 75.5% in secondary and tertiary hospitals). In the patient assessment process, over 79% of physicians focused on the duration and severity of underlying diseases, and more than 80% also emphasized definitive etiological diagnosis, though with a lower rate in primary hospitals (80.2% vs. 86.6% and 85.3% in secondary and tertiary hospitals). Separately, over 83% of physicians believed that cough duration would influence treatment decisions. Regarding allergy management, 88.2% of physicians in tertiary hospitals routinely inquired about patients' allergy history, compared with 79.1% in primary hospitals. Pollen and dust mite allergies were considered the primary allergic triggers leading to prolonged cough. In terms of distribution of cough types, physicians in tertiary hospitals saw that allergic cough accounted for 8.8% of cases, which was higher than the 4.6% in secondary hospitals and the 2.2% in primary hospitals. Respiratory tract infections, cold air, and dust/fume were the three most common cough triggers observed clinically, which led to difficulty in cough relief, making complex etiology and frequent relapse two major challenges in clinical practice.

Conclusion Physicians across all hospital levels reached a consensus on the core factors influencing cough relief, with medication types identified as the primary determinant. Tertiary hospital physicians also paid closer attention to medication adherence and allergic diathesis.

Pulmonary vascular disease – from novel therapies to clinical service improvements

P30 THE PULSE-PH REGISTRY: REAL-WORLD UK DATA IN PATIENTS WITH INTERSTITIAL LUNG DISEASE AND SARCOIDOSIS-ASSOCIATED PULMONARY HYPERTENSION

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Background Pulmonary hypertension (PH) can complicate ILD (ILD-PH) and sarcoidosis (SAPH), worsening prognosis and quality of life. We present the first UK national ILD-PH and SAPH registry and describe outcomes and predictors of mortality.

Methods Patients with right heart catheter (RHC)-confirmed PH were identified from UK PH centres (2003-25). Survival analysis was performed using Kaplan-Meier and Cox regression (IRAS 350356).

Results 1063 patients were included: 808 (76.0%) ILD-PH, 255 (24.0%) SAPH. Mean age 63.3±11.9 years, 46.6% male. Most (90.9%) were in WHO FC III-IV, 6MWD 240±122.9 m, emPHasis-10 32.9±11.4. Main ILD subtypes included CTD-ILD (36.5%; 47.5% scleroderma) and IPF (15.8%). ILD-PH diagnoses increased over time (~11%/year; $p<0.001$).

At PH diagnosis, mPAP was 42.6±18.6mmHg, PCWP 9.9±4.4mmHg, PVR 7.6±4.4WU, FVC 63.4±20.9%, TLCO 25.2±14.5%, NT-proBNP 406 (115-2317) ng/L. On echocardiography, RVSP was 65.6±21.6mmHg; RA dilatation was present in 59.3%, RV dilatation in 66.9%, RV dysfunction in 64.5%. PDE5 inhibitor use increased from ~20% in 2008 to approximately 65-75% from 2014 onwards.

Median survival was longer in SAPH vs ILD-PH (3.5 vs 1.8 years; log-rank $p<0.001$). Mortality was associated with RA and RV dilatation (HR 2.26, 2.62), RV dysfunction (HR 1.63) and NT-proBNP (HR 1.32 per log unit, all $p<0.001$), but not lung function or haemodynamics.

Conclusion In this first UK ILD-PH and SAPH registry, survival differs by aetiology and is associated with right-heart echo changes, rather than lung disease severity.

P31 NATIONAL COHORT STUDY: CAUSES OF MORTALITY IN PATIENTS WITH CHRONIC THROMBOEMBOLIC PULMONARY DISEASE WITHOUT PULMONARY HYPERTENSION

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The natural course of chronic thromboembolic pulmonary disease (CTEPD) without pulmonary hypertension (PH) is less well defined than chronic thromboembolic pulmonary hypertension (CTEPH). A study of 113 patients revealed no disease progression over a median follow-up period of 37 months and mortality was mainly attributable to malignancy (Reddy, SA. et al. J Heart Lung Transplant 2023; 42(9):1275-85). As there is limited contemporary work, we set out to define long-term mortality in patients with CTEPD with no or borderline PH.

All patients referred to the National CTEPH Multidisciplinary Team with CTEPD and no or borderline PH between January 2015 and December 2022 were included in a retrospective study (351 patients). Age at diagnosis was 58 years ± 17. Deceased patients were older (69 years ± 15) and more likely to be in WHO class III. 51 (15%) died during the study period with a median follow-up duration of 7 years. The commonest cause of death was sepsis (n = 16, 31%) followed by malignancy (n = 14, 27%), then ischemic heart disease (n = 9, 18%). Commonest malignancies were hematological, renal and gas-

trointestinal. Although our study did not assess CTEPD disease progression, <5 of the 351 patients died of causes related to CTEPD (acute pulmonary embolism), with none resulting from its direct consequences, including CTEPH and right heart failure. Of deceased patients, 29 underwent right heart catheterization, with a mean diagnostic mPAP of 19 mmHg ± 4. 72% (n = 21) had a mPAP ≤20mmHg.

Sepsis, ischemic heart disease and malignancy represent the dominant causes of death in CTEPD without PH. Healthcare professionals should not lose focus on preventative care and treatment for common co-morbidities, as they are more likely to be life-limiting than CTEPD.

P32 HIDDEN IN PLAIN-SIGHT: ICD-10/SNOMED REASONS WHY THOUSANDS OF NHS PATIENTS WITH PULMONARY ARTERIOVENOUS MALFORMATIONS ARE MISSED BY MAJOR RARE DISEASE INITIATIVES

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Background Resource allocations and research priorities rely increasingly on digital capture of disease prevalence. The importance of pulmonary arteriovenous malformations (PAVMs) was evident in the 2005-2014 US Nationwide Inpatient Sample database where PAVMs caused 0.02% of 4,271,910 acute ischemic stroke admissions,[1] yet in the UK, PAVMs were not among 331 rare diseases captured to <10 cases across 58,162,316 individuals.[2]

Methods To test if NHS-based coding systems were preventing recognition, we reviewed methods of[2]; searched Orphanet, ICD-10, SnoMed and ICD-11 for 'pulmonary arteriovenous malformation' or 'pulmonary arteriovenous'; and queried an institutional pseudonymised Rare Disease database for face-to-face reviews.

Results Inclusion in[2] required an Orphanet code that mapped to clinically-validated ICD-10 or SnoMed terms, and a diagnosed, alive patient on 23 January 2020.

'PULMONARY ARTERIOVENOUS MALFORMATION' retrieved ORPHA:2038 with an appropriate descriptor, but 'No results found!'[in red] from ICD-10, and 303070000 from SnoMed, where cases required prior surgical cavopulmonary anastemoses (table 1).

'PULMONARY ARTEROVENOUS' retrieved ORPHA:2038 and two ICD-10 codes: One was appropriate (I28.0), but carried obsolete descriptors and anatomical inaccuracies ('arteriovenous fistula of pulmonary artery vessels'). The second, Q25.7 ('Other congenital malformations of pulmonary artery') linked to SnoMed 303070000 which referred to the post-surgical complication (table 1).

None of the 1,149 radiologically-confirmed PAVM cases in the NHS institutional database had undergone cavopulmonary anastemoses, so none would meet the criteria for pulmonary arteriovenous malformation coding in SnoMed. Of the 945 cases identified by 31.12.2020, 130 had a post 2020-2026 face-to-face review confirming alive status at the censor date.

Since alive patients were missed, we tested if ICD-11 would have improved recognition. This separates the post-procedural disorder (BE14.B) from natural pathologies, but 2 separate diseases map to LA90.5 ('pulmonary arteriovenous malformation' and 'pulmonary arteriovenous aneurysm') and >5 diseases/normal physiological states to BB02.0 (table 1).

Conclusion NHS patients with radiologically-confirmed PAVMs are missed in NHS-wide datasets based on ICD-10/SnoMed, despite a valid, unique Orphanet Code, because of aetiology-limiting, non-unique and/or inaccurate ICD-10/SnoMed codes that will not survive quality assurance and validation step filters. ICD-11 would not have improved. Urgent updates are required to reduce current inequities in service provision, training, funding and research.

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Abstract P32 Table 1 Codes retrieved for 'Pulmonary arteriovenous malformation' or pulmonary arteriovenous with mapping links

Source	Code	Name	Text Comment	ICD-10	ICD-11
Orphanet	2038	PAV malformation	Appropriate text indicating multiple causes including hereditary haemorhagic telangiectasia	Q25.7	LA90.5
SnomedCT	303070000	PAV malformation	Arteriovenous malformation of pulmonary vessels following cavopulmonary anastomosis	Q25.7	
	111289009	PAV fistula		I.28.0	
	270512000	PAV aneurysm		I.28.0	
ICD-10	I.28.0	Arteriovenous fistula of pulmonary vessels	Arteriovenous fistula of pulmonary artery vessels	I.28.0	
	Q25.7	PAV aneurysm	Other congenital malformations of pulmonary artery: Aberrant pulmonary artery, Agenesis, Aneurysm, congenital, Anomaly, Hypoplasia of pulmonary artery, Pulmonary arteriovenous aneurysm	Q25.7	
ICD-11	LA90.5	PAV fistula	2 separate diseases: pulmonary arteriovenous malformation and pulmonary arteriovenous aneurysm		LA90.5
	BB02.0	AV fistula of pulmonary vessels	4 separate diseases/physological states ([1] Arteriovenous fistula of pulmonary vessels [2] acquired arteriovenous pulmonary aneurysm [3] arteriovenous pulmonary shunt, [4] aortopulmonary fistula) stated.		BB02.0
	BE14.B	Postprocedural disorder following conduit or shunt	Formation of *pulmonary arteriovenous* fistulas or malformations due to superior *cavopulmonary* anastomosis		BE14.B

Sources last accessed 07.06.2026. PAV, PAV= pulmonary arteriovenous

P33 INTERMEDIATE-HIGH RISK PULMONARY EMBOLISM: OUTCOMES WITH SYSTEMIC THROMBOLYSIS OR INTRAVENOUS HEPARIN IN REAL-WORLD PRACTICE

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Introduction Management of intermediate-high risk pulmonary embolism (PE) remains controversial. The recent HI-PEITHO[1] study demonstrated benefit with catheter directed thrombolysis (CDT) although CDT remains unavailable in many UK centres. We evaluated outcomes of intermediate-high risk PE managed on a Respiratory HDU (RHDU), using systemic alteplase or intravenous heparin infusion.

Methods We performed a retrospective single-centre review of patients with intermediate-high risk PE between January 2017–December 2025. Patients received systemic alteplase or intravenous heparin infusion at clinician discretion. A pre-specified subgroup applied PEITHO 2026 eligibility criteria: moderate-to-large clot burden; RV dysfunction with troponin elevation; absence of shock; and ≥2 of: HR >110 bpm, SBP <110 mmHg, or respiratory compromise.

Results 282 intermediate-high risk patients were included (alteplase n=139, heparin infusion n=143). Within the alteplase group, full dose (40%) was preferentially administered to younger patients with lower comorbidity (age 59 vs 70, p<0.001; CCI (Charlson Co-morbidity Index)2 vs 3, p=0.014), compared to weight adjusted/half dose (60%). Malignancy was more prevalent in the heparin infusion group (18.3% vs 7.2%, p=0.007). Alteplase was not associated with a mortality benefit compared with heparin infusion: 28-day mortality 2.2% v 4.2% (p=0.501), 6-month mortality 5.0% v 10.6% (p=0.119). Major extracranial bleeding was higher with alteplase (6.5% vs 1.4%, p=0.033). Of 28-day deaths, 2/3 after alteplase were due to bleeding complications and 4/6 after heparin felt to be related to PE. All 6-month deaths (n=13) were attributable to pre-existing co-morbidities.

98 patients met PEITHO 2026 eligibility criteria (alteplase n=61, heparin infusion n=37), well-matched for age (64 vs 68 years, p=0.355), CCI (median 2 vs 3, p=0.108), and malignancy (10.0% vs 23.3%, p=0.115). Weight-adjusted/half dose alteplase was administered to 41 patients (67.2%) at clinician discretion, preferentially in older patients (age 67 vs 58, p=0.017). No intracranial haemorrhages or 28-day mortality occurred in either arm. Major extracranial bleeding occurred in 2/61 thrombolysed, both non-fatal.

Conclusion Intermediate-high risk PE meeting PEITHO criteria can be managed safely on a RHDU with systemic alteplase or heparin infusion. This data provides important context where CDT is unavailable and suggest good short-term outcomes with structured RHDU care.

REFERENCE

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P34 PRIOR PROSTACYCLIN PATHWAY EXPOSURE AND TOLERANCE OF INTRAVENOUS EPOPROSTENOL INITIATION IN PULMONARY ARTERIAL HYPERTENSION

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Background Intravenous epoprostenol remains an important treatment option for patients with advanced pulmonary arterial hypertension (PAH), although initiation is frequently complicated by prostacyclin-related adverse effects. Selexipag is an oral selective prostacyclin receptor agonist that acts on the same signalling pathway. We evaluated whether prior selexipag exposure was associated with improved tolerance of subsequent epoprostenol initiation.

Methods A retrospective service evaluation was performed in adults with PAH admitted for intravenous epoprostenol initiation between October 2023 and January 2026. Patients were stratified according to prior selexipag exposure and categorised into the Selexipag Group and the Non-Selexipag Group. Demographic, haemodynamic, echocardiographic and clinical outcome data were collected from electronic patient records.

Results Thirty-one patients underwent epoprostenol initiation, of whom eight were included in the Selexipag Group and twenty-three in the Non-Selexipag Group. Baseline age, sex, WHO functional class, ESC/ERS risk status and invasive haemodynamics were similar between groups. Severe right ventricular dysfunction was more common in the Non-Selexipag Group (52% vs 12.5%, p=0.04).

Headache occurred less frequently in the Selexipag Group (25% vs 70%, p=0.043), while nausea was observed only in the Non-Selexipag Group (0% vs 61%, p=0.004). No patient in the Selexipag Group required modification of epoprostenol titration due to adverse effects, compared with 61% of patients in the Non-Selexipag Group (p=0.004). Median epoprostenol dose at discharge was similar between groups (10 vs 9 ng/kg/min, p=0.618). Admission duration was shorter in the Selexipag Group (4 vs 6 days, p=0.008). Rates of hypotension, pulmonary oedema and vasopressor use were numerically lower in the Selexipag Group.

Conclusions Patients in the Selexipag Group experienced fewer treatment-related adverse effects during intravenous epoprostenol initiation, resulting in less frequent titration modification and shorter admissions. These findings raise the possibility that prior selexipag exposure may improve tolerance of subsequent epoprostenol initiation. Larger prospective studies are required, particularly given differences in baseline right ventricular dysfunction between groups.

P35 THROMBOLYSIS FOR ACUTE PE: TIME FOR RAPID MULTIDISCIPLINARY INTERVENTIONAL TEAM?

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Introduction Acute pulmonary embolism (PE) remains a significant cause of mortality; despite thrombolysis, 4-week mortality can reach 21.6%.[1] Identifying intermediate-high-risk patients at risk of deterioration remains a critical challenge. This study aimed to evaluate the characteristics and outcomes of patients requiring thrombolysis for acute PE.

Methods A retrospective review was conducted on patients receiving alteplase for suspected or confirmed PE between January 2023 and December 2025 at a tertiary respiratory hospital. Data included demographics, biomarkers, PE Severity Index (PESI) score, thrombolysis timing, and clinical outcomes. Non-parametric statistical methods were utilised.

Results Of 2,427 patients diagnosed with PE, 28 patients underwent thrombolysis (mean age 55.5±18.7; 12 males); 4 who presented in cardiac arrest prior to imaging were excluded. Of the 24 analysed, risk stratification was high in 6, intermediate-high in 17, and intermediate-low in 1. Imaging showed right ventricle strain in 23/24 (95.8%) cases. Troponin was elevated in all patients (Median 274 [IQR 110-566]).

There were no significant differences between the high and intermediate-risk groups regarding age, troponin and admission PESI score. Patients in the intermediate groups had higher in-hospital mortality (33% vs 0%, p=0.277) compared to the high-risk group, although not statistically significant.

18/24 (75%) patients received early thrombolysis (≤24 hours of admission; median time 343 mins). This group was associated with higher D-dimer (median 15.2 v. 3.9), Troponin, and night thrombolysis (72% vs 17%), but lower risk of in-hospital mortality (11% vs 67%, p=0.018) compared to the delayed thrombolysis group (table 1). There was no difference in PESI score between the two groups.

Overall in-hospital mortality was 6/24 (25%). Mild non-cranial haematoma was reported in 3/24 (12.5%) patients who underwent thrombolysis.

Conclusions Intermediate-high-risk patients represent the majority of this cohort and face substantial in-hospital mortality. Those receiving rapid thrombolysis (within 24 hours) were associated with better survival. These findings underscore the critical need for close monitoring, early recognition of deterioration, and timely multidisciplinary discussion for early catheter-directed interventions in intermediate-high-risk patients.

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Abstract P35 Table 1 Timing of thrombolysis. Characteristics of patients receiving thrombolysis for suspected or confirmed PE (N = 24) split by early (<24 hours after admission) and late (>24 hours) thrombolysis. M – male, F – female. Continuous variables were analysed using the Wilcoxon rank-sum test (Mann-Whitney U test) and are reported as medians with interquartile ranges (Median [IQR]). Categorical variables were assessed using Fisher's Exact Test and reported as counts per category

Variable	<= 24 hours (n=18)	> 24 hours (n=6)	P-value
Age	63.5 [44.8 - 70.8]	64.5 [58.0 - 68.0]	0.920
Sex	{'F': 8, 'M': 10}	{'F': 5, 'M': 1}	0.166
Risk on admission	{'High': 5, 'Intermediate high': 13}	{'High': 1, 'Intermediate high': 4, 'Unclear/Pending Echo': 1}	0.453
D Dimer value	15.2 [12.7 - 17.9]	3.9 [2.1 - 5.7]	0.009
Troponin Value	306.6 [139.5 - 746.1]	120.7 [63.4 - 350.6]	0.199
BNP value	7553.0 [5147.2 - 9745.8]	5934.0 [5643.0 - 13979.0]	0.943
PESI score on admission	125.0 [91.5 - 139.5]	138.0 [114.8 - 140.2]	0.664
Highest PESI score (prior to thrombolysis)	137.0 [112.0 - 143.8]	139.5 [114.8 - 141.0]	1
Time from initial admission to thrombolysis (min)	342.5 [207.2 - 549.0]	6168.5 [2917.8 - 12843.0]	<0.001
Time between CTPA and thrombolysis (min)	131.0 [82.2 - 231.8]	1480.5 [1340.5 - 3144.5]	<0.001
Timing of thrombolysis (day, night)	{'Day': 5, 'Night': 13}	{'Day': 5, 'Night': 1}	0.050
Length of stay (hrs)	177 [84.2 - 270.0]	253 [129.2 - 593.5]	0.271
Clinical outcome (Death, discharged)	{'Death': 2, 'Discharged': 16}	{'Death': 4, 'Discharged': 2}	0.018

P36 THE VALUE OF PDE5I THERAPY IN PATIENTS WITH GROUP 3 PULMONARY HYPERTENSION: REAL WORLD DATA FROM A PULMONARY HYPERTENSION CENTRE

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Introduction Phosphodiesterase 5 inhibitors (PDE5i) have been demonstrated to be beneficial for patients with pulmonary arterial hypertension (PH) (Group 1). Despite conflicting data, PDE5i are being increasingly used for this patients with PH associated with lung disease (Group 3) with aim to improve their symptoms and large cohort data suggest improved survival.

Aims and Objectives This service evaluation looked at the clinical outcomes of patients with Group 3 PH treated with PDE5i in a UK pulmonary hypertension specialist centre.

Methods Retrospective data collection of patients with Group 3 PH treated with PDE5i at Imperial College Healthcare NHS Trust, a designated PH specialist centre. Patients were identified using an in-house research database. All patients were included who had baseline and 3 month follow up data. Paired changes were assessed using Wilcoxon signed-rank testing, with exploratory subgroup analysis according to underlying respiratory diagnosis performed with Kruskal Wallis analysis.

Results 73 patients were included. At diagnosis, mean age 65 (±12) years, FEV1 66.5 (±26.4)%, FVC 77.8 (±27.4) %, mPAP 46.9 (±10.0) mmHg and PVR 10.5 (±4.3) WU. 29 patients had follow up right heart catheterisation at 3 months.

Following treatment with PDE5i, there was a statistically significant reduction in pulmonary vascular resistance, (median change −2.33 WU, p=0.013), echocardiographic TR velocity (median change −0.28 m/s, p=0.010), 6 minute walk distance (median change +16 m, p=0.049) and BNP (median change −44 pg/mL, p=0.017). Changes in mPAP, cardiac index and RVSP did not reach statistical significance. There was no statistically significant difference in treatment response between patients with different underlying respiratory diagnoses. Median survival was poor overall at 22.0 months.

Conclusion In the real-world setting, treatment with PDE5i in patients with Group 3 PH was associated with short-term improvements. No clear difference in response was demonstrated between underlying respiratory diagnoses with heterogeneity seen between diagnoses and different parameters but this subgroup analysis is limited by its small numbers. Ongoing global clinical trials in phase 2 and 3, permitting the use of background PDE5i, are recruiting into these groups, with many UK PH centres currently enrolling.

Abstract P36 Table 1 Median changes in physiological, functional and quality of life score in patients with group 3 PH treated with PDE5i at 3 months

Outcome	Group	Paired n	Baseline median	3-month median	Median change	p-value	p-value between groups
6MWD (m)	All patients	33	126	170	+16	0.049	
	COPD	11	170	170	+30	0.058	0.076
	CPFE	5	190	144	-23	0.418	
	ILD	10	108	188	+55	0.155	
BNP (pg/mL)	All patients	39	449	195	-44	0.017	
	COPD	14	774	346	-190	0.079	0.547
	CPFE	4	677	323	-364	0.584	
	ILD	15	449	293	-3	0.293	
mPAP (mmHg)	All patients	28	50	46	-2.5	0.151	
	COPD	9	51	49	+1	0.766	0.097
	CPFE	4	48.5	45.5	-3	0.461	
	ILD	10	41	36.5	**-5**	**0.009**	
PVR (WU)	All patients	26	11.4	8.5	-2.33	0.01	
	COPD	8	13.8	13.0	-2.05	0.294	0.704
	CPFE	3	9.5	9.5	-3.7	0.371	
	ILD	10	9.66	6.65	-1.78	0.262	
Cardiac Index ($L/min/m^2$)	All patients	22	1.75	1.95	+0.15	0.068	
	COPD	7	1.5	1.6	+0.30	0.075	0.15
	CPFE	3	1.6	1.52	-0.08	0.371	
	ILD	8	1.95	1.95	-0.15	0.623	

P37 CATHETER DIRECTED THERAPY (CDT) OF PULMONARY EMBOLISM: AN OVERVIEW OF THE SERVICE RUN IN A SOUTH WALES DISTRICT GENERAL HOSPITAL

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Introduction and Objectives Management of Pulmonary Emboli (PE) remains an evolving area, with new American Heart Associate (AHA) guidelines published in 2026. Catheter directed therapy (CDT) was developed as a minimally invasive treatment for PE and received NICE approval for use in 2015. Our hospital is a District General covering a population of 600,000 and has run a CDT service since opening in 2020. This case series of patients undergoing CDT aims to examine patient selection, feasibility of a daytime service and the outcomes of treatment.

Methods We retrospectively analysed the data of patients who underwent CDT; reviewing demographics, PE severity based on PESI and ESC guidelines and the outcomes and complications of therapy. The new AHA/ACC guidelines were then applied to see if local practice was consistent with their recommendations and to provide data on areas where the benefits of CDT are unclear.

Results 40 Patients underwent CDT between 2020 and 2025. The median PESI score was 100 (Class III Intermediate risk) and 92.5% met the ESC guideline criteria of 'High-risk or Intermediate high-risk PE'. All patients would be placed in category C or above, according to new AHA Criteria. The median wait for CDT from the time of diagnosis on CTPA was 9.83 hours. Two patients had failed procedures, one required further CDT and one required transfer for endarterectomy. 92.5% patients survived to discharge with a median length of stay of 6.5 days. There were no significant procedure-related adverse outcomes, no major bleeding events and no readmissions.

Conclusions This retrospective analysis shows that CDT is a safe therapy for selected patients with PE. It demonstrates that clinicians favour this treatment modality for patients with intermediate high-risk PE predominantly (AHA class C3/D). The increased usage of CDT locally will allow future analysis of the patient cohort the new AHA/ACC guidance deem unclear (class C2-D2).

Abstract P37 Table 1

TOTAL CASES	N =40	
Sex	Male (*%*)	29 (*72.5%*)
	Female (*%*)	11 (*27.5%*)
Age (years)	Median (***Range***)	63 (*32-79*)
PESI score	Median (***Range***)	106 (*48-204*)
Catheter based intervention	Thrombolysis (*%*)	38 (*95%*)
	Thrombectomy (*%*)	2 (*5%*)
Time from confirmed PE to CDT (hours)	Median (***Range***)	9.83 (*2.05-24.16*)
Post procedure care	Respiratory High Care (*%*)	22 (*55%*)
	ITU (*%*)	16 (*40%*)
	CCU (*%*)	1 (2.5%)
	Respiratory Ward (*%*)	1 (2.5%)
Alive at discharge	Yes (*%*)	37 (*92.5%*)
	No (*%*)	3 (*6.5%*)
Days of Admission	Median	6.5

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P38 CAN DISTRICT GENERAL HOSPITALS DELIVER ADVANCED PULMONARY EMBOLISM CARE? A TWO-YEAR REAL-WORLD EXPERIENCE OF CATHETER-DIRECTED THERAPY

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Introduction and Objectives Acute pulmonary embolism (PE) is a major cause of cardiovascular morbidity and mortality. Catheter-directed therapy (CDT), including catheter-directed thrombolysis and mechanical thrombectomy, is increasingly used for high-risk and intermediate-high-risk PE. However, advanced PE interventions are usually delivered in tertiary centres, with limited real-world evidence from district general hospitals (DGHs). We aimed to assess the safety, feasibility and clinical outcomes of CDT delivered within a UK DGH.

Methods We conducted a retrospective observational study of consecutive patients who underwent CDT for acute PE between August 2020 and November 2022 at a UK DGH. CDT was delivered through a multidisciplinary pulmonary embolism response team model involving interventional radiologists and respiratory physicians. Demographic, clinical, radiological, procedural and outcome data were extracted from electronic medical records. Outcomes included in-hospital mortality, 30-day mortality, bleeding complications, intensive care admission and length of hospital stay. Descriptive analysis was performed using IBM SPSS Statistics.

Results A total of 19 patients underwent CDT. Mean age was 57.2 years (SD 18.8), and 57.9% were male. Severe PE, defined as high-risk or intermediate-high-risk PE, was present in 17/19 patients (89.5%), and 3/19 patients (15.8%) presented following cardiac arrest. Mean RV:LV ratio on CT was 1.82 (SD 0.36), and right ventricular dysfunction was identified in 16/17 patients who underwent echocardiography (94.1%). Median lactate, time from diagnosis to CDT and length of hospital stay were 2.0 mmol/L (IQR 1.4–2.9), 23.5 hours (IQR 14.3–59.4) and 8 days (IQR 6.8–11.0), respectively. Overall, 7/19 patients (36.8%) required intensive care admission. In-hospital and 30-day mortality were both 10.5% (2/19). One death followed cardiac arrest due to massive PE, and one was secondary to sepsis. No deaths were directly related to CDT. No major or minor bleeding complications were observed. Further baseline characteristics, procedural details and clinical outcomes are summarised in table 1.

Conclusions In a cohort largely composed of patients with severe PE, CDT showed a favourable balance of safety and effectiveness, with low mortality and no procedure related

Abstract P38 Table 1 Baseline characteristics, treatment details and outcomes following catheter-directed therapy for acute pulmonary embolism

UK district general hospital cohort, August 2020 to November 2022		
Domain	**Variable**	**Result**
Cohort	Patients treated with CDT	19
	Age, mean ± SD	57.2 ± 18.8 years
	Male sex	11/19 (57.9%)
Clinical severity	High-risk PE	4/19 (21.1%)
	Intermediate-high-risk PE	13/19 (68.4%)
	High-risk or intermediate-high-risk PE	17/19 (89.5%)
	Cardiac arrest prior to CDT	3/19 (15.8%)
	Respiratory support: room air / supplemental oxygen / HFNO / mechanical ventilation	15.8% / 36.8% / 36.8% / 10.5%
Haemodynamics and biomarkers	Systolic BP at presentation, mean ± SD	123.8 ± 19.9 mmHg
	Heart rate, mean ± SD	107.7 ± 24.9 bpm
	Respiratory rate, mean ± SD	25.2 ± 8.9 breaths/min
	Lactate, median (IQR); mean ± SD	2.0 mmol/L (1.4-2.9); 2.79 ± 2.36
	Troponin, mean ± SD	129.8 ± 117.3 ng/L (n=12)
	BNP, mean ± SD	3760 ± 4104 pg/mL (n=5)
	D-dimer, mean ± SD	7739 ± 12521 ng/mL
Imaging	RV/LV ratio on CT, mean ± SD	1.82 ± 0.36
	RV dysfunction on echocardiography	16/17 (94.1%)
Treatment delivered	Catheter-directed thrombolysis only	5/19 (26.3%)
	Mechanical thrombectomy only	3/19 (15.8%)
	Mechanical thrombectomy + catheter-directed thrombolysis	11/19 (57.9%)
	Alteplase dose, mean ± SD	13.8 ± 9.2 mg
Service delivery	Time from diagnosis to CDT, median (IQR)	23.5 hours (14.3-59.4)
	ICU admission	7/19 (36.8%)
	Length of stay, median (IQR)	8 days (6.8-11.0)
Outcomes and safety	Survival to discharge	17/19 (89.5%)
	In-hospital mortality	2/19 (10.5%)
	30-day mortality	2/19 (10.5%)
	Major bleeding	0/19 (0%)
	Minor bleeding	0/19 (0%)
	Procedure-related mortality	0/19 (0%)

Abbreviations: BNP, brain natriuretic peptide; CDT, catheter-directed therapy; HFNO, high-flow nasal oxygen; ICU, intensive care unit; IQR, interquartile range; PE, pulmonary embolism; RV/LV, right ventricular/left ventricular; SD, standard deviation.
Baseline characteristics, PE severity, treatment approach and clinical outcomes in 19 patients undergoing catheter-directed therapy.

bleeding events. These results support the feasibility of delivering CDT as an advanced treatment option within appropriately resourced district general hospitals.

P39 IMPACT OF FRAILTY ON CLINICAL OUTCOMES IN ACUTE PULMONARY EMBOLISM: A UK DISTRICT GENERAL HOSPITAL COHORT STUDY

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Introduction Acute pulmonary embolism (PE) is a common and potentially life-threatening cause for hospital attendance in the UK. National guidelines recommend the Pulmonary Embolism Severity Index (PESI) as a validated risk stratification tool in patients with PE, incorporating factors such as age and comorbidity. However, it does not directly account for frailty (Clinical Frailty Scale, CFS >4). We aimed to assess the impact of frailty on clinical outcomes of patients diagnosed with acute PE in a District General Hospital.

Methods A single-centre retrospective cohort study was carried out by searching electronic data from our radiology reporting system. Of 1,470 CTPAs performed in 2023 with PE identified as the primary diagnosis, 106 patients met inclusion criteria (incidental PE and unconfirmed cases excluded). Data collected included CFS, PESI, ethnicity, length of stay (LOS) and mortality.

Results Mean age was 62.3 years (SD 19.7). Overall 30-day mortality was 11.3% (12/106). 28.3% of patients were frail (CFS >4). The cohort was ethnically diverse (50.9% White, 26.4% Black, 6.6% South Asian). Frail patients were significantly older (median 80 vs 58 years, $p<0.001$), had higher

Abstract P39 Table 1 Key outcomes by frailty status

Outcome	Frail (CFS >4) n=30	Not Frail (CFS ≤4) n=76	p value	
Median age, years (IQR)	80 (72–87)	58 (42–70)	<0.001	
Mean PESI score (SD)	126 (47)	79 (32)	<0.001	
30-day all-cause mortality, n (%)	9/30 (30.0%)	3/76 (3.9%)	0.0005	*
Median LOS, days (IQR)	7 (3–12)	4 (0–8)	0.009	*
Ambulatory management, n (%)	6/30 (20.0%)	26/76 (34.2%)	0.17	

* Statistically significant (p<0.05). LOS=length of stay; IQR=interquartile range; SD=standard deviation; CFS=Clinical Frailty Scale; PESI=Pulmonary Embolism Severity Index.

30-day mortality (30.0% vs 3.9%; OR 10.4; p=0.0005), significantly longer LOS (median 7 vs 4 days; p=0.009) and higher PESI scores (mean 126 vs 79, p<0.001). 30.2% were managed ambulatorily. See table 1.

Conclusion This cohort study highlights significantly increased 30-day mortality and length of stay in frail patients with acute PE. CFS >4 may identify patients requiring closer inpatient monitoring and enhanced post-discharge care, including Virtual Ward pathways. Routine frailty assessment can inform PE risk stratification alongside PESI.

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P40 USING NEWS TO PREDICT OUTCOMES IN ACUTE PULMONARY EMBOLISM: A TWO-CENTRE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.202

Introduction and Objectives Acute pulmonary embolism (PE) is a frequent cause of emergency admission with a widely varying inpatient course. Identifying at the front door which patients will need a longer stay would help early bed and resource planning, yet simple tools for this are lacking. The National Early Warning Score (NEWS) is recorded on every acute admission. We asked whether admission NEWS predicts inpatient length of stay in confirmed PE, comparing two practical strata: NEWS <3 versus NEWS ≥3.

Methods Retrospective two-Centre cohort of consecutive patients with CT-proven PE (2021). Of 245 positive scans, 28 without admission observations and 62 with active malignancy were excluded a priori, leaving 165 patients (88 NEWS <3, 77 NEWS ≥3). The primary outcome was hospital length of stay (LOS); in-hospital mortality was a secondary, exploratory outcome. Age was compared between groups and adjusted for. LOS was analysed with the Mann–Whitney U test and age-adjusted log-linear regression; mortality with Fisher's exact test, in IBM SPSS. p<0.05 was significant.

Results The groups were closely matched for age (64.8 vs 65.1 years; p=0.91). Admission NEWS ≥3 was associated with a significantly longer length of stay (median 6 vs 3 days; p<0.001), and the effect persisted after adjustment for age, with the NEWS ≥3 group staying 1.48 times longer (95% CI 1.17–1.88; p=0.002). In-hospital mortality was higher with NEWS ≥3 (7.8% vs 2.3%) but, based on only eight deaths, did not reach significance (p=0.15) and is reported as exploratory (table 1).

Abstract P40 Table 1 Outcomes by admission NEWS group

Outcome	NEWS <3 (n=88)	NEWS ≥3 (n=77)	p
Age, mean (SD), years	64.8 (15.8)	65.1 (16.3)	0.91
Length of stay, median (IQR), days	3 (1–5)	6 (3–9)	<0.001
Length of stay, age-adjusted ratio	reference	×1.48 (1.17–1.88)	0.002
In-hospital mortality, n (%)	2 (2.3%)	6 (7.8%)	0.15

Conclusions In age-matched patients with CT-proven PE, a single admission NEWS ≥3 identified, at presentation, a group who stayed in hospital roughly twice as long. Because NEWS is universally recorded and available within minutes of arrival, it offers a simple, practical signal to support early bed management and resource planning in acute PE — independent of patient age. The accompanying higher in-hospital mortality is hypothesis-generating and merits testing in larger, multicentre cohorts.

P41 COMPARISON OF CLINICAL AND BIOCHEMICAL PREDICTION TOOLS FOR EXCLUDING PULMONARY EMBOLISM AND REDUCING IMAGING

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10.1136/thorax-2026-BTSabstracts.203

Introduction and Objectives Computed tomographic pulmonary angiography (CTPA) is the gold-standard investigation for suspected pulmonary embolism (PE) but contributes significantly to radiation exposure, contrast administration and increasing radiology workload. We evaluated the diagnostic performance of Wells score, PERC, YEARS criteria and age-adjusted D-dimer, individually and in combination, in patients undergoing CTPA for suspected PE.

Methods A retrospective service evaluation was performed of consecutive CTPAs undertaken for suspected PE at a UK district

Abstract P41 Table 1 Comparison of clinical prediction pathways for the exclusion of pulmonary embolism and potential reduction in CTPA utilisation

Strategy	n	% cohort	PE missed	NPV
PERC = 0	153	10.20%	3	98.00%
YEARS excluded	122	8.10%	2	98.40%
Wells ≤4	678	45.20%	74	89.10%
Wells ≤4 + PERC = 0 (<50y)	125	8.30%	3	97.60%
Wells ≤4 + age-adjusted D-dimer negative (≥50y)	37	2.50%	0	100%
Combined pathway + YEARS	32	2.10%	0	100%

Breaking barriers – improving lives, transforming access to healthcare

general hospital between December 2024 and September 2025. Demographic data, Wells score, PERC score, D-dimer results, age-adjusted D-dimer thresholds, YEARS criteria and CTPA outcomes were extracted from electronic records. Diagnostic performance and potential reductions in CTPA utilisation were assessed for individual and combined rule-out strategies.

Results A total of 1499 CTPAs were performed. PE was confirmed in 206 patients, giving an overall diagnostic yield of 13.7%.

Among 153 patients with PERC score 0, three PEs were identified.

YEARS criteria classified 122 patients as PE excluded, although two PEs were confirmed on CTPA. Wells score ≤4 identified 678 patients with 74 confirmed Pes.

In patients aged ≥50 years, Wells score ≤4 combined with a negative age-adjusted D-dimer identified 37 patients, none of whom had pulmonary embolism on CTPA.

Combining age-adjusted pathways with YEARS criteria identified a cohort of 32 patients with no confirmed pulmonary emboli, achieving 100% negative predictive value.

Conclusions Although individual prediction tools demonstrated high negative predictive value in our real-world cohort, however combined clinical prediction pathways may represent a safe strategy to reduce unnecessary CTPA utilisation. Prospective validation is required before implementation.

Breaking barriers – improving lives, transforming access to healthcare

P42 THE OVERLOOKED RESPIRATORY BURDEN OF HOMELESSNESS: A UK ELECTRONIC HEALTH RECORDS STUDY

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10.1136/thorax-2026-BTSabstracts.204

Introduction People experiencing homelessness (PEH) are among the most marginalised groups in society and experience marked health inequalities. Respiratory conditions account for over 6% of deaths in this population, compared with around 2% in the general population. Yet healthcare and research have largely focused on infectious diseases, mental health, and drug and alcohol use, while respiratory disease has received little attention.

We aimed to estimate the prevalence of respiratory conditions in PEH and compare their respiratory-related healthcare use with that of housed people.

Methods Using Clinical Practice Research Datalink primary care records between 2004 and 2025, we identified adults with a homelessness code and matched them to randomly selected housed patients by age, sex and GP practice. We determined the proportion with chronic respiratory conditions and lower respiratory tract infections (LRTI) using relevant GP codes during follow-up. Within this cohort, we identified patients with Hospital Episode Statistics linkage and determined the proportion with respiratory-related A&E attendance or hospital admission.

Results The study included 148,985 PEH and 566,122 housed patients (median age 40 years, IQR 30–50; 67% male). The proportion with chronic respiratory conditions was higher in PEH than housed patients: asthma (20.3% vs 9.7%), COPD (4.2% vs 1.2%), bronchiectasis (0.3% vs 0.2%), lung cancer (1.4% vs 0.9%) and LRTI (25.9% vs 13.1%).

Among those with HES linkage, 70,161 PEH and 239,988 housed patients were included. Respiratory-related A&E attendance occurred in 11.4% of PEH compared with 4.5% of housed patients. Respiratory hospital admissions were also higher in PEH: pneumonia (9.8% vs 3.1%), LRTI (5.4% vs 1.9%), asthma attacks (3.1% vs 1.3%), COPD exacerbations (3.9% vs 1.0%), pulmonary oedema (1.5% vs 0.8%), pulmonary embolism (1.8% vs 0.7%) and non-specific respiratory symptoms (5.0% vs 2.4%).

Conclusion People experiencing homelessness experience a substantially higher burden of respiratory disease and respiratory-related acute healthcare use than housed people. Respiratory health should be recognised as a major component of homelessness-related health inequality and prioritised within integrated care, prevention and outreach services.

P43 INTRODUCING A NOVEL DAILY MEDICAL NURSE PRACTITIONER (MNP) PLEURAL CLINIC; ASSESSING CLINICAL EFFICIENCY AND PATIENT SATISFACTION

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Background The new British Thoracic Society (BTS) Quality Standards for Pleural Disease recommend diagnostic sampling within 5 working days of referral, with rapid access to pleural procedures and specialist review where required.[1] Delays to pleural intervention can contribute to prolonged inpatient stay, increased pressure on Same Day Emergency Care (SDEC), and poorer patient experience. In July 2025 we introduced a respiratory MNP-led pleural clinic to expand outpatient emergency pleural service capacity. The clinic runs daily, Monday–Friday, providing one appointment per day for rapid access assessment and pleural intervention where required. Introduction of this service has supported recommended metrics in the newly published BTS Quality Standards.

Aim To evaluate the impact of introducing a respiratory MNP-led pleural clinic on service efficiency, access to pleural care, and patient and staff experience.

Methods A retrospective review compared pleural outpatient activity during the 6 months before implementation (1/1/25–30/6/25) with the 6 months after implementation (1/7/25–31/12/25). Data collected included clinic activity, pleural procedures, complications, waiting times, admissions, and SDEC activity. Patient and staff surveys were analysed to assess service impact.

Results Thirty-six patients attended the MNP clinic during the evaluation period. Procedural activity increased by 31%, while complication rates remained similar (7.5% vs 8%). Average referral-to-review time reduced by 7.4 days. Of patients reviewed in the MNP clinic, 47% were seen on the day of referral and mean waiting time was 2.3 days, supporting improved adherence to BTS standards. Pleural aspirations performed through SDEC reduced by 50%. Patient satisfaction was high; 100% rated their experience as 'very good'. Staff reported improved waiting times (93%), positive impact on patient experience (100%), and manageable workload (92%). 43% identified confidence in performing independent thoracic ultrasound as an area for development.

Conclusion Introduction of an MNP-led daily pleural clinic improved service efficiency, reduced waiting times and SDEC reliance, increased procedural activity without increasing complications, and achieved high patient and staff satisfaction, supporting introduction of specialist nurse-led outpatient pleural pathways.

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P44 ABSTRACT WITHDRAWN

P45 TARGETED LUNG HEALTH CHECKS: ARE WE MISSING THE OPPORTUNITY TO DIAGNOSE AND TREAT COPD?

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Background The UK Targeted Lung Health Check (TLHC) programme was established to detect lung cancer earlier in high-risk individuals. However, low-dose CT scans frequently identify co-existing smoking-related lung disease, including emphysema. As the programme increasingly focuses on lung cancer outcomes, opportunities for earlier diagnosis and optimisation of chronic respiratory disease may be overlooked. In our trust, individuals with moderate or severe emphysema detected on screening CT are referred to secondary care for respiratory assessment. We evaluated the clinical value of this pathway and identified which patients benefit most from specialist review.

Methods We conducted a retrospective service evaluation of patients referred from TLHC programme to secondary care COPD clinic following detection of moderate or severe emphysema on screening CT in 2024. Electronic patient records were reviewed for demographics, spirometry, treatment interventions and clinical outcomes.

Results Of 165 referred patients, 145 attended secondary care review. Patients were aged 55–78 years, 61% were male, 76% had moderate emphysema and 19% severe emphysema; 12% reported ≥2 exacerbations in the preceding year.

Following assessment, 31 patients (22%) received a new COPD diagnosis and 92 (63%) had pre-existing COPD diagnosis. Among those with established COPD, management was suboptimal and 36% (n=52) required treatment initiation, stopping or modifying.

Of 40 current smokers, 35 (88%) accepted referral to tobacco dependence services despite previously declining support. 32 patients (22%) were referred to pulmonary rehabilitation (PR).

Most patients (n=126, 87%) were discharged after a single consultation, while 19 (13%) required ongoing secondary care follow-up, including 5 (3.4%) referred for lung volume reduction assessment and others requiring further specialist management. Mean COPD Assessment Test (CAT) scores were significantly lower in discharged patients than in those requiring follow-up (14.6 ± 9.4 vs 24.9 ± 6.0; $p = 3.4 \times 10^{-5}$).

Conclusions Emphysema detected through TLHC identifies a population with substantial unmet respiratory health needs. Secondary care review facilitated new COPD diagnoses, treatment optimisation, tobacco dependence engagement, PR referral, and identification of patients suitable for advanced interventions. These findings highlight the need to optimise TLHC pathways beyond lung cancer detection and ensure smoking-related lung disease is identified and treated early.

P46 THE ROLE OF A NURSE-LED CLINIC IN IMPROVING ACCESS AND EFFICIENCY IN THE MANAGEMENT OF LATENT TUBERCULOSIS INFECTION (LTBI) IN ADULTS AT IMPERIAL COLLEGE HEALTHCARE NHS TRUST TUBERCULOSIS SERVICES.

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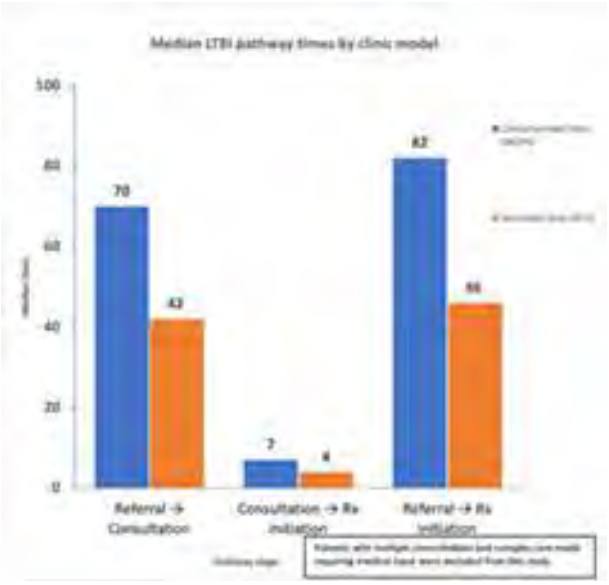
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Background Latent tuberculosis infection (LTBI) carries a 5–10% lifetime risk of progression to active disease without preventive treatment. Timely assessment and treatment are therefore essential. Before July 2023, adults with LTBI were managed through a consultant-led outpatient pathway, with prolonged waiting times from referral to first consultation and treatment initiation. To improve access and streamline care, a nurse-led advanced clinical practitioner (ACP) clinic was introduced.

Aim To evaluate the impact of an ACP nurse-led clinic (NLC) on pathway efficiency, patient experience, and service delivery for adults with LTBI.

Method A retrospective service evaluation compared LTBI pathways in the 12 months before and after NLC implementation. Electronic health records and the local TB database were reviewed to assess referral-to-appointment time, referral-to-treatment time, and efficiency. Patient experience was assessed using a questionnaire on consultation experience and access to the service.

Results The NLC, led by Senior TB Nurse Specialists practising at ACP level, reduced the median referral-to-first consulta-



Abstract P46 Figure 1

tion time from 70 to 42 days (40%), consultation-to-treatment time from 7 to 4 days (43%), and overall referral-to-treatment time from 82 to 46 days (44%). In the consultant-led pathway, 79% of patients rated consultations as excellent, compared with 100% in the NLC. All respondents reported having the opportunity to ask questions. In the NLC, assessment, counselling, and treatment initiation were combined into a single consultation, reducing appointments and freeing up consultant capacity for more complex TB cases.

Conclusion The LTBI NLC is a safe, effective model associated with faster access to preventive therapy. Combining assessment, treatment initiation, and counselling in a single appointment reduced duplication, saved time for patients and healthcare professionals, and improved service efficiency. Given the limited published evidence on nurse-led LTBI clinics, this model contributes to growing evidence base and highlights the important role of ACPs in TB services. If adopted nationally, it could enhance LTBI treatment capacity. Further evaluation should assess treatment completion and long-term outcomes.

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P47 TARGETED EDUCATION IS A POTENTIAL STRATEGY TO IMPROVE THE LIVED EXPERIENCE OF YOUNG ADULTS (16-25YRS) WITH SEVERE ASTHMA

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Background Living with severe asthma (SA) as a young adult is associated with poor health outcomes[1] and can negatively impact upon quality of life (QoL). There exists an evidence gap surrounding the experiences and support needs of young adults with SA, which needs addressing to increase understanding, facilitate age-appropriate care delivery and improve outcomes.

Aim To explore the experiences and support needs of young adults (16-25yrs) with SA, through delivering 1-1 interviews and focus group and to deliver a theory of change (ToC) workshop.

Methods 1-1 interviews using photo-elicitation and interpretative phenomenological analysis, along with a focus group, analysed using thematic analysis, were delivered to explore experiences and support needs of young adults with SA. A ToC workshop concluded the studies, identifying how to improve care and outcomes in this cohort.

Results 15 young adults, with a diagnosis of SA took part in the 1-1 interviews and/or focus group, 80% were female with a median (IQR) age of 22(19-25) yrs, Asthma Control Questionnaire 2.3(1.9-3.0), Asthma Quality of Life Questionnaire 3.9(2.3-5.4), 8(53%) of participants were receiving biological therapy.

The ToC (n=10) included healthcare professionals (HCPs) involved in delivering SA care and patient experts.

Both studies, and the ToC, highlighted that living with SA imposes a multifaceted burden (table 1). Poor education and misunderstanding of SA (including severity, management and QoL impact) amongst some HCPs, patients, and the public was recognised as a root cause of identified issues, with areas for improvement being highlighted (table 1).

The ToC agreed that focussing on improving education to raise awareness of SA and the impact on QoL may decrease patient burden, improve healthcare experiences, outcomes, QoL and support prompt referral.

Conclusion Living with severe asthma in young adulthood can carry a significant physical and psychological burden, impacting QoL. We recommend increased education surrounding SA

Abstract P47 Table 1

Impact of living with severe asthma as a young adult	Support needs & recommendations to improve care
Emotional & psychological distress, including fear, frustration, anxiety, isolation, uncertainty and guilt.	• Increased education around the psychological impact of living with SA to all HCPs. • Increased targeted psychological support
Poor healthcare experiences. Not listened to, dismissed, fighting for care, delays in diagnosis/referral, poor management of acute attacks in ED & incorrect treatment.	• Improved HCP education surrounding SA, management & the impact on QoL • Prompt referral to specialist services • Care must be better aligned with young adults' needs.
Poor perception of HCP and the public in understanding how SA differs to milder spheres of asthma, regarding treatment and impacts on QoL	• Increase HCP & public awareness of how SA differs from milder spheres of asthma in its impact on QoL & available treatment.
Daily physical burden, including poor sleep quality & ongoing symptoms impacting on daily functioning, limiting QoL & opportunities	• Increase the HCP & public awareness of the impact of SA as a complex condition and its impact on QoL
Medication concerns, including side effects, dependence, treatment failure & medication burden. Insufficient education to patients around treatments was highlighted	• Increase education to patients, primary care & ED about SA medications. • Prompt referral and access to biologics-Clearer referral pathways.

and the burden of living with the condition, amongst patients, healthcare professionals, and the public and propose further research is required to develop and evaluate this approach as a method to support this cohort.

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P48 PATIENT EXPERIENCE OF A MULTIDISCIPLINARY ASTHMA TRANSITION CLINIC: A RETROSPECTIVE SERVICE EVALUATION

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Background Transition from paediatric to adult asthma services is a critical stage in the care of young people. At our tertiary centre patients with difficult asthma aged ≥16 years attend a dedicated transition clinic involving a joint appointment with paediatric and adult respiratory physicians, alongside specialist nurses and a physiologist.

Aim We aimed to evaluate patient experience, perceived preparedness for adult services, and the effectiveness of this multidisciplinary model.

Methods A retrospective service evaluation was conducted using a structured questionnaire assessing patient experience of transition, including communication, support, self-management confidence, and overall satisfaction. Patients who attended the transition clinic between January 2023 and March 2026 were invited to participate via text messaging and telephone follow-up. Responses were analysed descriptively.

Results Nineteen eligible patients aged 17-22 years were identified, of whom eight completed the questionnaire (response rate 42%). The mean age of respondents was 18.7 years, and all had successfully transitioned to adult services. Overall satisfaction with the transition process was positive, with 62.5% reporting being satisfied or very satisfied. Patients demonstrated high confidence in understanding their asthma medications (mean score 4.75/5), the rationale for transition (4.5/5), and possession of an up-to-date asthma action plan (4.5/5). Key challenges were identified: 37.5% of respondents reported anxiety regarding adult services and 37.5% perceived a lack of information, particularly relating to lifestyle, physical activity, smoking, and transition to university or employment. Half of respondents reported at least one asthma exacerbation following transition. Patients who experienced exacerbations reported lower satisfaction scores than those without exacerbations (mean satisfaction score 2.75/5 versus 4.75/5).

Conclusions Our multidisciplinary asthma transition clinic was associated with generally positive patient experiences and good self-management confidence. However, targeted improvements are required to enhance preparation for adult care, particularly in addressing anxiety and providing more comprehensive lifestyle-related information. Larger prospective studies are needed to improve response rates and further optimise transition pathways.

P49 'NOTHING ABOUT ME WITHOUT ME'. YOUNG PEOPLE WITH ASTHMA AND UPPER AIRWAY DISORDERS TRANSITIONING TO ADULT SERVICES: SERVICE EVALUATION OF A UK HOSPITAL

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10.1136/thorax-2026-BTSabstracts.210

Introduction and Objectives Asthma is the most common long-term condition among children and young people, yet young people with asthma and co-existing upper airway disorders are underrepresented in transition literature and national reviews.

I evaluated participant experiences of transition within respiratory services and benchmarked findings against national recommendations.

Methods

1. Semi-structured interviews and thematic analysis with 4 young people (<25 years) with asthma and co-occurring upper airway disorder(s), and their 4 parents.
2. Stakeholder discussions involved paediatric and adult respiratory consultants and a paediatric-adult Speech and Language Therapist (SLT).
3. A trust-wide staff-survey gathering quantitative and qualitative data

Results Four themes emerged.

Delayed diagnosis and fragmented care: All young people described repeated healthcare encounters and referrals before upper airway disorders were diagnosed (range 2-6 years). Symptoms were frequently attributed to asthma or psychological causes. Participants identified a gap between paediatric respiratory discharge and adult upper airway provision, reducing continuity of specialist care.

Education, Identity and Opportunity: Young people described detriment to education, social participation, exercise, and future aspirations. Absence from school, delayed education progression and reduced participation in extracurricular activities were frequent. All who received SLT intervention reported symptomatic benefit, improving education, careers and mental health.

Transition and continuity of care: Young people valued direct communication, autonomy and continuity of care.

Provision: Participants identified limited access to upper airway specialists and a gap between paediatric and adult provision as barriers to effective transition.

Staff survey responses highlighted that access lost during the COVID-pandemic limited their ability to provide accurate assessment and management, which led to higher healthcare utilisation, longer follow-up times, and patients travelling to other hospitals for assessment. Participants supported integrated service models to improve continuity and flexibility of care.

Conclusions Young people with asthma and upper airway disorders experienced fragmented care and their symptoms were frequently misinterpreted. This contributed to frustration, anxiety and disruption to education, socialising, independence and future aspirations.

National guidance promotes coordinated, developmentally appropriate transition; however, participants frequently described 'transfer' between services rather than preparation for adulthood.

Integrated 0-25 models, dedicated transition support, and improved access to upper airway services may address these unmet needs.

P50 RESEARCH EQUITY BY DESIGN: INSIGHTS FROM THE A-TREC COHORT

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10.1136/thorax-2026-BTSabstracts.211

Background The NIHR INCLUDE framework highlights the need for trial designs that recruit under-represented groups.[1] Traditional hospital-centred models create logistical and financial barriers for vulnerable COPD patients, leading to selection bias and exclusion of those with significant physical disabilities or lower socioeconomic status. A recent systematic review highlighted that social determinants of health are poorly considered in trial design and rarely reported in published outcomes,[2] potentially limiting generalisability.

Objective To evaluate how home-based research follow-up, including point of care testing (Spiro, FeNO, bloods), affects engagement and facilitates the inclusion of housebound and socioeconomically disadvantaged individuals within a COPD research study.

Methods The A-TREC study (NCT06188065) identifies factors associated with admission eosinopenia and examines time to recovery in blood eosinophil count (BEC) following severe exacerbation of COPD. Patients were recruited upon admission and chose home-based or hospital-based follow-up involving six visits over six weeks. No upper age limit was applied. Patients were given the opportunity to provide anonymous feedback upon completion of the study.

Abstract P50 Table 1 Population descriptors of entire A-TREC cohort

Population descriptor	Summary (n = 180)
Age (years)*	73 ± 8.5
Male‡	92 (51%)
IMD†	4 (2 – 6)
Nursing or Residential Home‡	4 (2%)
eMRCD†	4 (3 – 5a)
Rockwood Clinical Frailty Score (CFS)†	5 (4 – 5)
Charlson Co-Morbidity Index†	4 (4 – 5)
Anxiety or Depression‡	63 (35%)
Current smoker‡	68 (38%)
LTOT use‡	19 (11%)
Home NIV‡	11 (6%)
FEV1% predicted*	33 ± 17
Annual rate of exacerbations in preceding year†	3 (1 – 5)

Data presented as mean ± standard deviation*, median (interquartile range)†, number with percentage‡
IMD: index of multiple deprivation, LTOT: long-term oxygen therapy, FEV1: forced expiratory volume in 1 second

Results To date, 180 participants have been recruited (65% consent rate); 179/180 (99%) requested home-based follow-up.

The cohort demonstrated high representation of historically excluded groups:

- 34% (n=61) were housebound (eMRCD 5a/5b)
- 48% (n=86) resided in the lowest three IMD deciles, with 19% (n=34) in the lowest decile

Qualitative feedback was overwhelmingly positive, with a common theme of improved access: *'I would not have been able to take part if the research team had not visited me at home.'* Participants also reported reassurance from community-based follow-up and an altruistic desire to improve COPD outcomes.

Discussion The 99% preference for home-based follow-up demonstrates a disconnect between traditional trial design and patient needs. Moving research into the community is a strategy for achieving research equity. This model bridges the gap for housebound and socioeconomically disadvantaged populations, ensuring findings are generalisable to the entire COPD population.

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P51 SAME-DAY TEXT MESSAGING FOLLOWING LUNG MDT DOWNGRADE DECISIONS: IMPROVING COMMUNICATION EQUITY IN A DEPRIVED, MULTILINGUAL POPULATION.

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Background In socioeconomically deprived and linguistically diverse urban populations, a substantial proportion of residents do not report English as their main language and many have limited English proficiency. This may reduce the effectiveness of traditional postal communication following multidisciplinary team (MDT) decisions. Postal correspondence may also result in delayed communication of outcomes and therefore delayed patient reassurance. We introduced same-day text messaging following lung MDT downgrade decisions and evaluated patient experience and communication equity.

Methods This quality improvement project included all patients downgraded following lung MDT discussion over a three-month period (June–September 2025). Patients received a same-day text message confirming no evidence of lung cancer and outlining follow-up where required. Telephone follow-up assessed recall, timing relative to postal letter, comprehension, need for translation support, and acceptability. Free-text feedback was also collected.

Results Forty patients received text messages. Twenty-two were contactable for feedback. Of these, 17 (77%) recalled receiving the message and 14 (64%) received it before the corresponding letter. All respondents supported ongoing use of text messaging. Patients reported reduced anxiety due to earlier reassurance compared with postal correspondence. Those with limited English proficiency highlighted improved accessibility, including use of smartphone translation tools and ability to share messages with family or friends. Two patients aged over

90 years preferred telephone communication due to difficulty using mobile phones. Eighteen patients could not be contacted, highlighting a potentially vulnerable cohort less likely to benefit from digital communication pathways. The intervention was associated with zero breaches of the 28-day Faster Diagnostic Standard during the study period.

Conclusion Same-day text messaging following lung MDT downgrade decisions was highly acceptable and improved timeliness of reassurance. The intervention enhanced communication equity in a deprived, multilingual population by reducing delays and improving accessibility of information. This low-cost intervention is scalable and offers a model for improving communication equity and pathway efficiency in similar healthcare settings.

P52 DEVELOPING A BLUEPRINT FOR USING CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) TO ILLUSTRATE BEST NEIGHBOURHOOD HEALTH AND OUTCOMES ACROSS THE UK

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10.1136/thorax-2026-BTSabstracts.213

Introduction Neighbourhood Health is a key element of NHS reform following the *Darzi Report*[1] and the *NHS 10-year Health Plan*,[2] calling for better integration across all healthcare sectors. COPD, which affects approximately 1.7 million diagnosed patients in the UK[3] makes it an ideal test case to illustrate how effective neighbourhood health models can reduce hospitalisations, improve quality of life and address inequalities. In July 2025, the Primary Care Respiratory Society (PCRS) launched a national project to explore this and develop a blueprint for COPD neighbourhood health.

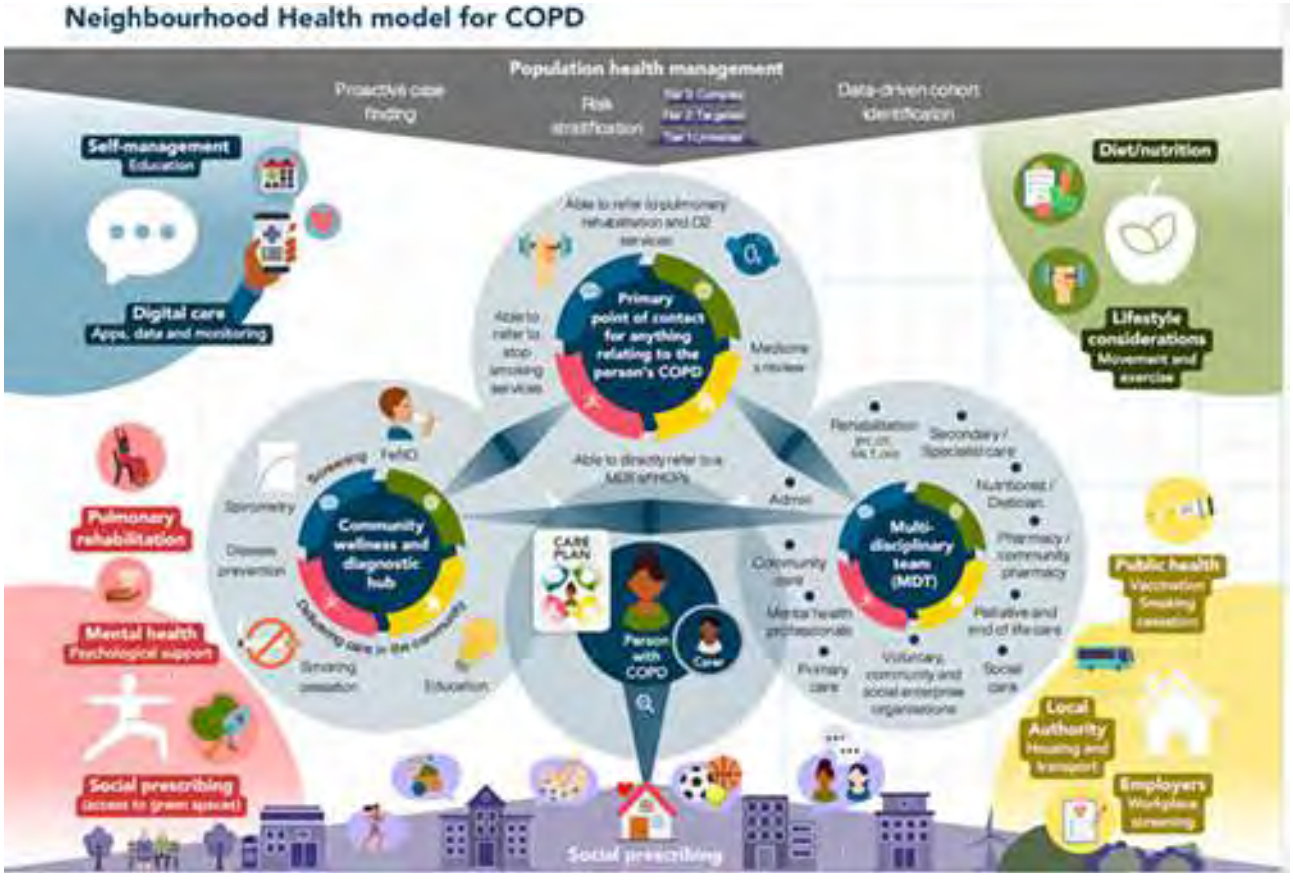
Methods

The project was delivered in two phases:

1. Gathering national examples/case studies of current innovative COPD neighbourhood practice. Initial entries (20) were reviewed and scored, with highscoring examples forming a national repository to support shared learning.
2. Convening a multidisciplinary group of experts (23) and patient representatives for a one-day meeting in October 2025. Using a modified Esther Model and structured sessions, the group explored lived patient experience, system barriers and gaps, opportunities for improvement, and what an effective neighbourhood response could look like.

Results Case studies showed that respiratory neighbourhood health models, delivering better COPD outcomes, were already being introduced across the UK and that a national repository hosting these examples was welcome (Link: PCRS COPD neighbourhood health repository).

Multidisciplinary group discussions identified core features of an effective neighbourhood response: integrated leadership; strengthened workforce capability; embedding prevention; shared and personalised care planning; single points of contact; multidisciplinary team access; proactive casefinding and data sharing, and strong community partnerships. Using an iterative production and review process, these features were used



Abstract P52 Figure 1

to produce a blueprint for COPD neighbourhood health (figure 1) which is supported by case study graphics and information resources to aid implementation.

Conclusion The project demonstrates that neighbourhoodlevel COPD care requires coordinated leadership, patientcentred design, and integrated working across healthcare sectors. The October 2025 meeting produced clear principles for a neighbourhood COPD blueprint, emphasising prevention, early intervention, personalised support, and shared accountability. These insights were used to develop a full blueprint suite to support national adoption and drive improvements for people living with COPD (Link: https://www.pcrs-uk.org/resource/current/blueprint-chronic-obstructive-pulmonary-disease-copd-neighbourhood-health **UNDER EMBARGO**).

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P53 FINDINGS FROM THE HERON NEEDS PRIORITISATION PARTNERSHIP: EXPLORING AND MAPPING UNMET NEEDS AND POTENTIAL HEALTH TECHNOLOGY SOLUTIONS IN CHRONIC RESPIRATORY DISEASE

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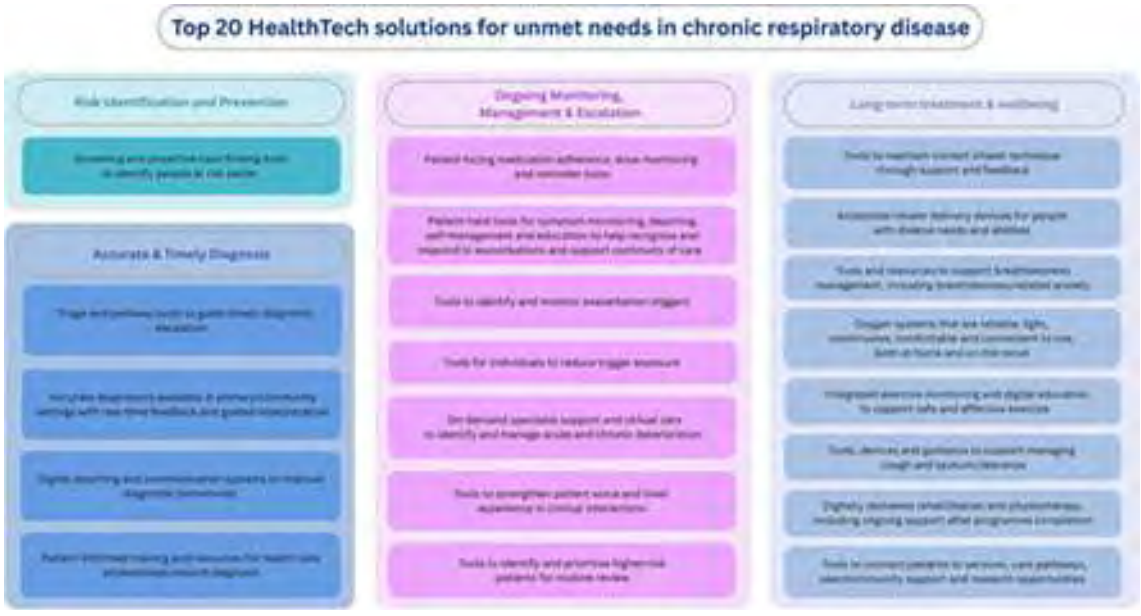
10.1136/thorax-2026-BTSabstracts.214

Background Chronic respiratory disease (CRD) carries high symptom burden and frequent emergency admission, disproportionately affecting disadvantaged populations. Advances in health technology (HealthTech) offer new opportunities to improve early detection, diagnosis, monitoring, and management of CRD and are increasingly prioritised within NHS and UK Government strategy. However, innovation is often developed without sufficient alignment to the priorities of patients, carers, and healthcare professionals, limiting its potential benefit. HERON aimed to identify and prioritise unmet needs in CRD and assess the extent to which existing and emerging technologies address these priorities.

Methods HERON is a Priority Setting Partnership guided by the James Lind Alliance methodology. UK-wide digital surveys were completed by patients, carers, people at risk of CRD (n=136), and healthcare professionals (n=103). Nine in-person engagement group discussions (n=75) were conducted to minimise digital exclusion. Responses were thematically analysed to generate a longlist of unmet needs. Targeted searches across eight databases identified 5715 relevant records, from which 306 technologies were mapped to unmet needs and evaluated using an adapted Technology Readiness Level framework. A multi-stakeholder prioritisation workshop involving patients, carers, and healthcare professionals finalised a Top 20 list of potential HealthTech solutions to address unmet needs.

Results Seventy-three unmet needs were identified across the chronic respiratory disease care pathway. The final Top 20 HealthTech priorities focus on earlier identification and diagnosis, self-management and symptom control, digitally enabled rehabilitation, psychological and community support, remote monitoring and virtual care, and optimisation of inhaler use, medication adherence, and oxygen therapy (figure 1). The technology scoping review identified areas of active innovation but also important gaps, particularly in integrated care pathways, continuity of support, and technologies addressing the broader physical, psychological, and social impacts of chronic respiratory disease.

Conclusions HERON provides an evidence-informed, stakeholder-prioritised assessment of unmet needs across the chronic respiratory disease care pathway and the extent to which they



Abstract P53 Figure 1

are addressed by current HealthTech innovation. These findings can help ensure that future respiratory technologies are aligned with real-world clinical and lived-experience priorities, supporting more targeted research, innovation, funding, and implementation activity.

P54 EXPERIENCES OF SPECIALIST NURSE REVIEW DELIVERED REMOTELY IN PROGRESSIVE PULMONARY FIBROSIS

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10.1136/thorax-2026-BTSabstracts.215

Introduction and Objectives Remote review is often framed by communication modality. In rare chronic respiratory disease, scheduled and patient-initiated telephone contact may be better understood as a way of sustaining continuity, allowing concerns arising between in-person assessments to be reviewed and carried forward. This study examined how people living with progressive pulmonary fibrosis and specialist interstitial lung disease (ILD) nurses experience this form of review over time.

Methods This qualitative study used a phenomenology informed critical realist design. Semi-structured interviews were conducted with 15 patients and 14 ILD nurses. Interviews incorporated participant chosen objects to support their reflection on remote care. Transcripts were analysed using reflexive thematic analysis. Participant accounts were then integrated to examine recurring patterns in experience and the conditions shaping remote review.

Results Remote nurse reviews were primarily telephone-based and most valued when situated within a continuing thread of care. Patients initiated contact after noticing change, weighing its significance and deciding whether it merited specialist attention. Patients felt more able to re-contact when they had a sense of being known to the service and confidence that concerns would be treated as warranting attention. Remote review could address concerns when it helped patients make sense of change; where it led to a fundamental reappraisal of illness and future life, timely face-to-face review was needed.

Patient and nurse work was connected but asymmetrical. Nurses translated patient accounts into clinical judgement by drawing on investigation results, previous encounters and shared team knowledge, locating concerns within the person's illness trajectory, social situation and connection with the service. Remote review was strongest where team support and professional agency enabled nurses to connect spoken accounts to the person's wider care context and shape a proportionate clinical response.

Conclusions Remote ILD nurse review is a longitudinal form of specialist care, sustained across scheduled calls, ad-hoc contact and post-encounter work. Rather than simply providing convenient access, it offers a route through which concerns arising between appointments can be brought back into an established nursing relationship. A history of concerns being heard, interpreted and acted on helped build patients' sense of safety beyond any single review.

P55 THE VALUE OF A MULTIDISCIPLINARY BREATHLESSNESS SERVICE TO MANAGE EXTRA PULMONARY TREATABLE TRAITS IN PATIENTS WITH CHRONIC RESPIRATORY DISEASE

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10.1136/thorax-2026-BTSabstracts.216

Introduction Patient centred management, targeting specific treatable traits (TT) and multi-morbidity clusters, is recommended in international GOLD COPD guidelines and GINA Asthma guidelines. This approach is reliant on a strong multi-disciplinary team (MDT). The assessment of breathing clinic (ABC) consists of a primary care physician led MDT assessment of patients with chronic breathlessness.

Aims To evaluate the frequency of treatable traits in respiratory patients, referred to and managed by the ABC MDT.

Methods A database of patients referred to the ABC team was screened. Patients referred from respiratory teams over a five-month period were included in the review. Electronic records were screened. Key demographic data, data from ABC assessments and ABC MDT were collected.

Results 112 patients were reviewed. Mean (SD) age 61 (16), mean FEV1% predicted 73 (22), mean exacerbations 12 months prior to referral 2 (2). 69% female, median index of multiple deprivation 2 (1-3).

98% of patients had TT identified, shown in table 1, with a median of 3 per patient

80% patients received physio input, 71% Speech and language therapy, 36% social prescribing and 22% talking therapies.

Conclusion TT were almost ubiquitous in this highly symptomatic cohort, with over half of patients having 3 or more TT identified by the ABC MDT.

The higher number of asthma patients referred likely reflects the TT management approach embedded in regional asthma biologic pathways.

Abstract P55 Table 1 Table showing total treatable traits in study cohort and in most common primary diagnoses

Treatable traits	Total number (n = 112)	Asthma (n = 49)	COPD (n=27)	COPD/Asthma (n=17)
Breathing pattern disorder	87 (78%)	39 (80%)	24 (89%)	14 (82%)
Upper airway	87 (78%)	43 (88%)	23 (85%)	11 (65%)
CVD	12 (11%)	5 (10%)	3 (11%)	3 (18%)
Anxiety/ depression	72 (64%)	36 (73%)	21 (78%)	11 (65%)
Obesity	48 (43%)	26 (53%)	10 (37%)	7 (42%)
Physical deconditioning	17 (15%)	5 (10%)	6 (22%)	3 (18%)
Sleep disorder	32 (29%)	15 (31%)	7 (26%)	5 (29%)
Reflux	20 (18%)	16 (33%)	1 (4%)	2 (12%)

Following the recent NICE approval of biologics in COPD, chronic breathlessness teams must be integrated within COPD biologics service to allow early identification and optimisation of extra-pulmonary treatable traits in this high-risk group.

P56 RIGHT CARE, EVERY TIME: DESIGNING PATIENT AND HEALTHCARE PROVIDER CHECKLISTS TO SUPPORT NON TUBERCULOUS MYCOBACTERIA PULMONARY DISEASE (NTM-PD) MANAGEMENT ACROSS THE UK

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Background There is a lack of high-quality evidence to help people with NTM-PD and their healthcare providers engage in shared decision making. The limited availability of patient-specific resources is a barrier to patient self-management and active engagement in care. Furthermore, consistently addressing all relevant pharmacological and non-pharmacological aspects of care in accordance with the recent UK NTM Standards of Care represents an important but often hard to implement priority for clinicians. Freely available, user-friendly checklists for both patients and clinicians could help to address these challenges, promoting high-quality care, improved patient engagement and educational opportunities.

Methods We established a multidisciplinary working group comprising NTM-PD patients, clinicians, physiotherapists, pharmacists and nurses to co-produce four patient and two clinician checklists to optimise NTM-PD care. To inform checklist design and avoid resource duplication, an assessment of existing resources for NTM-PD and other respiratory conditions was conducted using a systematic literature search plus review of websites of relevant professional respiratory clinical and research societies with a plausible interest in NTM-PD. A core writing group drafted the six checklists which were reviewed by the working group for accuracy, relevance and usability.

Results Our search identified limited existing information. The new patient checklists cover key milestones in the NTM patient journey, namely: 'after my diagnosis', 'my review appointment', 'commencing treatment for NTM', and 'my patient passport'. The two clinician checklists address early post-diagnosis care, and review appointments. Two key themes emerged from patient contributors during evaluation: the need for resources that reflect and accommodate individual patient priorities, and use of checklists to support navigating the healthcare system in the context of their disease. Document evaluation and real-world validation are ongoing through NTM Network UK and NTM Patient Care UK.

Conclusions We identified limited practical resources currently available to assist NTM patients and their healthcare providers' management decisions. Addressing this through co-produced, focused information is an important first step.

Acknowledgements The authors acknowledge the other members of NTM Network UK working party who have contributed to checklist development.

TB: from diagnosis to care

P57 EVALUATING OUTCOMES OF ANTI-TB THERAPY IN OLDER PEOPLE

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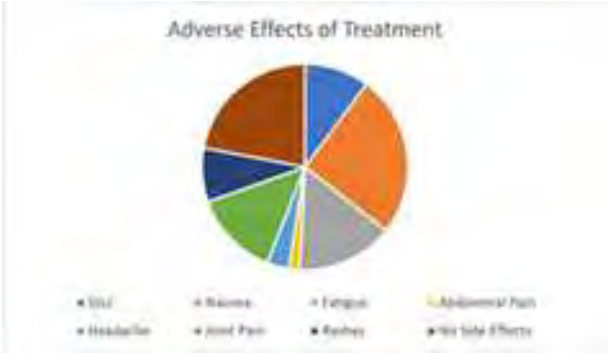
Background Increasing age and co-morbidities can make anti-tuberculosis treatment (ATT) less well tolerated in older people, and therefore potentially harder for them to complete curative treatment. There are limited published data regarding whether outcomes of active TB differ compared to younger groups. We sought to describe the management and outcomes of active tuberculosis in older people in a low-TB incidence setting.

Methods All people treated for active tuberculosis between 2021 and 2025 in our institution who were aged over 65 at the time of diagnosis were identified and reviewed using a standardised data collection proforma.

Results 59 cases of active tuberculosis in people over the age of 65 were identified. Median (IQR) age 73 (68-83) years, 42 (71%) were male and 29 (49%) had pulmonary disease. 48 (81%) were culture-positive for *Mycobacterium tuberculosis*, of whom 6 (12.5%) had isoniazid resistant infection (none had rifampicin resistance).

Patients were on a median of 4 other medications before starting anti-TB therapy; 42 (71%) were taking medications with significant drug-drug interactions with rifampicin a the start of ATT. All patients were started on standard treatment regimens. Adverse drug effects occurred in 78% of patients (figure 1). Interruptions or changes to initial regimens were required in 19 (32%) patients.

38 (64%) were confirmed to have completed ATT;12 (20%) died before completing treatment (in 5 TB directly contributed to death, for 7 death was from another identifiable cause); 7 (12%) either did not complete ATT or this could not be evaluated and 2 were lost to follow-up.



Abstract P57 Figure 1

Conclusions Older patients with active tuberculosis frequently have significant drug-drug interactions with other medications taken at the start of ATT. Adverse drug reactions are common and often lead to interrupted or changed therapy. Outcomes are worse than in nationally reported data with a higher proportion not completing treatment or dying during ATT.

P58 CHARACTERISING NON-TUBERCULOUS MYCOBACTERIAL PULMONARY DISEASE (NTM-PD): HOW DOES THIS COMPARE WITH BRONCHIECTASIS? AN OBSERVATIONAL CROSS-SECTIONAL STUDY

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Background Treatment for NTM-PD includes antimicrobial therapy and non-pharmacological interventions, which are largely extrapolated from bronchiectasis, despite limited evidence supporting their use in NTM-PD. Although bronchiectasis shares several clinical characteristics with NTM-PD, it is unclear whether differences exist in disease burden, and patients' care needs.

This study aimed to develop a clinico-physiological picture of NTM-PD and to better understand whether this differs from people with bronchiectasis and no NTM pulmonary infection.

Methods We conducted a single-site, cross-sectional observational study comparing adults with NTM-PD diagnosed according to British Thoracic Society (BTS) guidelines and not on anti-mycobacterial treatment to people with bronchiectasis and no NTM pulmonary infection. Clinical characteristics, radiological findings, exercise capacity, respiratory muscle strength, body composition, psychological status, and health-related quality of life were assessed.

Results Forty-three participants were recruited (21 NTM-PD, 22 bronchiectasis), with female proportions of 90.5% and 68.2%, respectively.

The mean age was 70.7 years (NTM-PD) and 65 years (bronchiectasis). *Mycobacterium avium complex* (MAC) was the commonest NTM species (n=15/21, 71%). Chronic cough was highly prevalent in both groups (81% vs 91%, p=0.41). Fatigue (90% vs 68%, p=0.13) and chest pain (52% vs 23%, p=0.06) were more often reported in NTM-PD, alongside a higher frequency of night sweats (71% vs 27%, p=0.006), and poor appetite (52% vs 18%, p=0.02).

NTM-PD participants reported significantly worse quality of life across the QoL-B domains, including physical functioning (52.1% vs 72.1%, p=0.03), and role functioning (64.8% vs 81.5%, p=0.007).

Compared with bronchiectasis, participants with NTM-PD had lower BMI (22.1 kg/m2 vs 24 1 kg/m2, p=0.07), fat-free mass index (12.8 vs 14.5 kg/m2, p=0.005), total body water (28.5 vs 31.2 L, p=0.02), and exercise capacity measured by six-minute walk distance (365.5 vs 427 m, p=0.03). Respiratory muscle strength using Sniff Nasal Inspiratory Pressure (54.1 vs 61.1 cmH2O, p=0.22) and psychological status using Patient Health Questionnaire-9 (5 vs 3.86, p=0.25) were similar between groups.

Conclusion Although NTM-PD group demonstrated a greater impairment in key functional and physiological measures, similarities with bronchiectasis were also observed. These findings highlight the potential relevance of non-pharmacological interventions used in bronchiectasis for people with NTM-PD alongside disease-specific treatment.

P59 AN INTERIM EVALUATION OF PHARMACY SERVICE CONTRIBUTIONS TO TUBERCULOSIS (TB) CARE PATHWAYS IN THE UK: A STAKEHOLDER PRACTICE AND PERCEPTION SURVEY

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Introduction and Objective TB infection remains a leading cause of mortality and morbidity, with rising notifications across UK. The Getting It Right First Time 2025 (GIRFT) review[1] emphasises optimising TB services nationally and highlights the key role of pharmacy, which was absent in 43% of reviewed services. Analysing pharmacy contributions and barriers is essential to identify gaps and improve care.

Abstract P59 Table 1

Percentage of respondents in agreement to GIRFT Recommendations	
A senior pharmacist of bands 7 or greater should be attached to and support the TB service on a pro rata basis for the potential workload	92% (36/39)
Recognition of pharmacy role in XDR/MDR and NTM should be included together with time in job plans to attend internal MDTs and cohort reviews.	90% (35/39)
The role of pharmacy support for regional MDTs needs to be included in the future development and funding of such services.	92% (36/39)
Percentage of respondents that agree that the following barriers currently limit the engagement/ involvement of pharmacy professionals with TB services.	
Lack of specialist TB training and expertise in TB management for pharmacists	2. 62% (24/39)
Lack of funding/ resources	92% (36/39)
Limited integration of pharmacy into TB multidisciplinary teams	62% (24/39)
Lack of clear, formalised roles and responsibilities for pharmacists in existing TB care pathways.	82% (32/39)

Methods The most relevant pharmacy professional involved in TB clinical care at each service was invited to complete a survey via Google Forms from February 2026, distributed through the national Chief Pharmacists network. Interim data reported up to June 2026 are presented.

Results Response to the survey has been slow, possibly indicating limited direct involvement of pharmacists in TB services across the UK. Thirty-nine pharmacists across UK and Scotland completed the survey: 25 (64%) were infectious disease specialists and 13 (33%) respiratory specialists and [1] clinical service lead. Fifteen worked in tertiary or teaching hospitals, 24 in district general hospitals.

Pharmacists contributed to TB services in 72% (28/39) of sites, of which 89% (25/28) were unfunded. Pharmacists contributing to TB services provided support in the management of active TB (100%, 28/28), latent TB infection (93%, 26/28), multidrug-resistant (MDR)TB (71%, 20/28), and non-tuberculous mycobacterial (NTM) disease (86%, 24/28).

Respondents strongly supported GIRFT pharmacy recommendations, but barriers to pharmacy involvement included lack of funding (92%, 36/39) and absence of TB training (51%, 20/39). Other challenges were poor integration with TB multidisciplinary teams and undefined TB pharmacist roles (table 1). Opportunities for workforce development include a TB pharmacist training programme, standards of care, dedicated funding, and a peer-support pharmacy TB network.

Conclusions Interim results show pharmacy integration in TB care is highly valued, yet barriers such as funding and lack of structured training persist. Addressing these gaps is essential to achieve national GIRFT objectives and combat antimicrobial resistance. These findings highlight barriers to meeting GIRFT recommendations and inform near-future strategies to strengthen TB pharmacy services.

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P60 INTRODUCING ANTIMICROBIAL STEWARDSHIP WITHIN TB SERVICES - A REVIEW OF INFECTION PHARMACY-LED INTERVENTIONS AT A LONDON TERTIARY CENTRE

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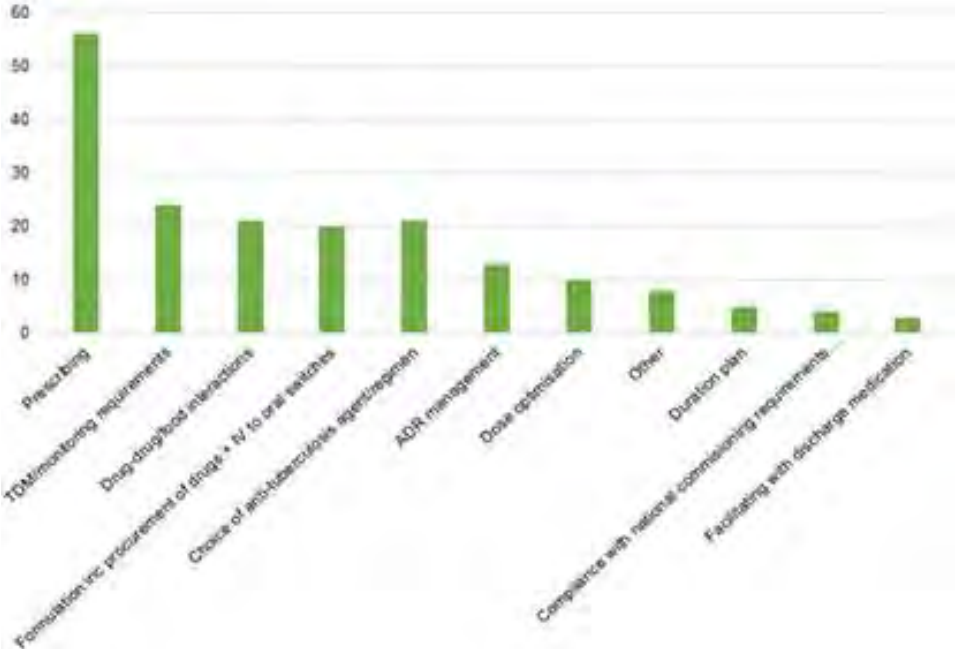
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Background Tuberculosis (TB) is a global concern, with rising notification rates in UK, especially in London. A recent report by Getting It Right First Time (GIRFT) on TB[1] and a national patient safety alert regarding TB drug supply[2] have highlighted unwarranted variation in how clinical pharmacy services are utilised. AMS programmes, through their design and delivery, are ideally positioned to support TB services alongside specialist clinicians to optimise therapies.

At our large tertiary academic TB centre, Infection pharmacists provided regular input to optimise patient-centred TB care. This review describes hospital Infection pharmacy-led interventions to highlight pharmacists' role in TB management.

Methods For a 5-month period (May to October 2025), all pharmacist-led or supported interventions for active and latent TB treatment were recorded at the time of intervention. Routine practice involves Infection pharmacists, who are all independent prescribers, attending all TB multidisciplinary team (MDT) meetings, MDRTB MDTs, TB ward rounds, and offering virtual support via email and phone to medical and nursing colleagues five days a week.

Results A total of 185 interventions were carried out over a 5-month period, with the majority, 48% (89/185), occurring during an MDT, followed by 39% (72/188) through email or phone queries from the TB team. 92% of the interventions (171/185) involved managing active TB, of which 56% (95/171)



Abstract P60 Figure 1 Types of interventions made by infection pharmacists during the study period

were in patients with MDR/XDRTB. The interventions were categorised accordingly, as shown in figure 1.

Conclusion Findings suggest that pharmacists can play a significantly broader antimicrobial stewardship role in overall TB services than currently described in the literature. Their skill sets enable the wider MDT and patients to benefit from pharmaceutical expertise, including drug selection, offering supportive therapies for ADR management, particularly in complex cases like MDRTB- and aiding the implementation of national guidelines. This has led to our centre being recognised as a model for pharmacy services by GIRFT.

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P61 DRUG-INDUCED LIVER INJURY IN ACTIVE AND LATENT TUBERCULOSIS TREATMENT: A RETROSPECTIVE EVALUATION OF MONITORING STRATEGIES

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Introduction Drug-induced liver injury (DILI) is a common complication in patients undergoing treatment for tuberculosis (TB). Current liver function test (LFT) monitoring guidelines rely on pre-treatment risk factors and hepatitis-like symptom onset during treatment, and primarily consider active TB treatment. This study evaluated the effectiveness of universal LFT monitoring strategy compared with existing guidelines in identifying cases of DILI in patients undergoing treatment for active or latent TB infection.

Methods A retrospective cohort study was conducted including patients aged ≥16 treated for active or latent TB between October 2015 and March 2020. LFTs were monitored regularly using a universal surveillance strategy and DILI cases were diagnosed using established criteria. Detection rates were compared with those expected under guidelines published by the American Thoracic Society (ATS), British Thoracic Society (BTS), and National Institute for Health and Care Excellence (NICE).

Results Of 133 patients treated for active TB, 20 (15.0%) met criteria for biochemical or borderline DILI. All cases would have been identified using existing guidelines. Of 279 patients treated for latent TB, 20 (7.2%) met criteria for biochemical or borderline DILI. 25% (ATS) and 55% (BTS/NICE) of patients with DILI or borderline DILI undergoing latent TB treatment would not have been identified by guideline-based LFT monitoring strategies. A substantial proportion of patients in this cohort developed DILI in the absence of risk factors and hepatitis-like symptoms. Time to DILI onset ranged from 0-92 days (active TB) and 7-94 days (latent TB). No statistically significant associations were found between guideline-based risk factors and DILI occurrence. No cases of acute liver failure or mortality were observed in either cohort.

Conclusion Universal LFT monitoring in patients undergoing anti-tuberculous treatment merits consideration for both active and latent TB. While existing guidelines appeared effective in identifying cases of DILI in active TB in this study, their reliance on symptom-driven investigation may delay identification of DILI, increasing the risk of acute liver failure, irreversible liver damage and death. Our results question the predictive utility of risk factors used in established guidelines in identifying patients who are likely to develop DILI.

P62 LOCAL, BUT NOT SYSTEMIC, NEUTROPHIL-DRIVEN INFLAMMATION ASSOCIATES WITH LUNG PATHOLOGY IN TUBERCULOSIS

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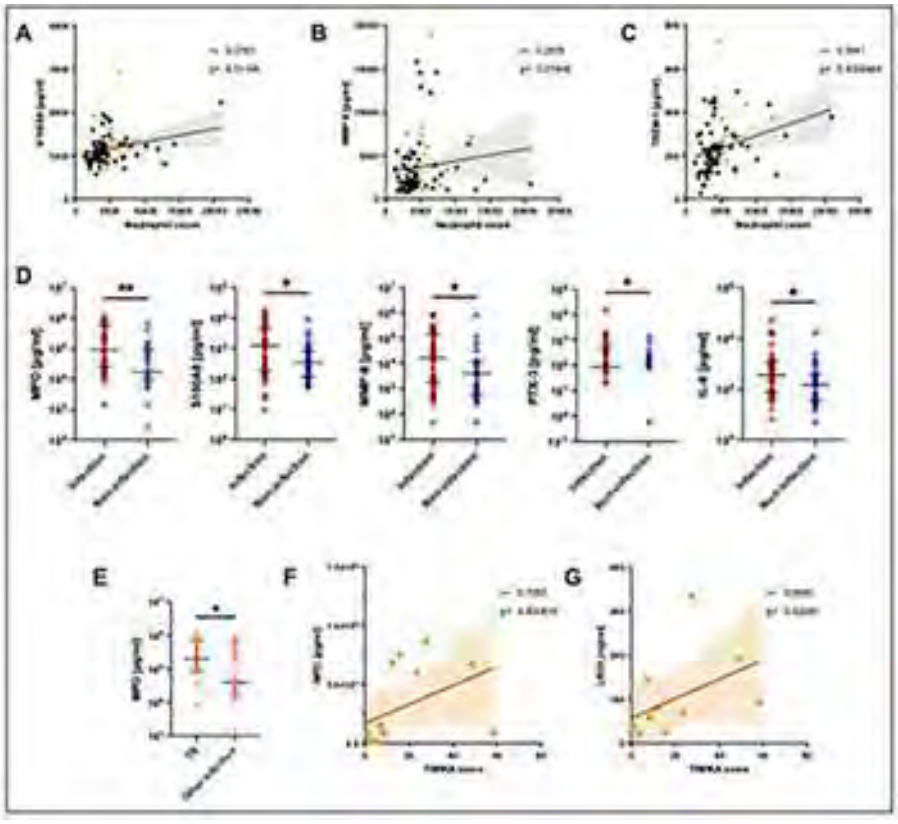
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Background Bronchoalveolar lavage fluid (BALF) is not routinely obtained from patients with suspected TB, therefore airway-level immunology remains understudied. Neutrophils are increasingly recognised as drivers of TB immunopathology.[1] We investigated neutrophil-associated signatures in plasma and BALF from bronchoscopy patients.

Methods Clinical and radiological data, plasma, and BALF were collected from 82 consecutive patients undergoing diagnostic bronchoscopy in Lima, Peru. BALF underwent TB culture, and neutrophil-associated mediators MPO, dsDNA, S100A8, MMP-8, MMP-9, PTX-3, IL-8, TREM-1, LRG1, CXCL2 and CXCL7 were quantified by Luminex or ELISA. Patients' medical records were reviewed after 2 months to ascertain the diagnosis of their presenting respiratory illness. TB patient chest x-rays were analysed for radiological severity using Timika scoring by two radiologists.

Results 17 patients were diagnosed with TB based on microbiological, clinical, and radiological criteria. Other patients were diagnosed with non-TB lung infection (n=22), chronic lung disease (n=13), cancer (n=6), or other diagnosis (n=13). Patients where no diagnosis was reached were excluded from the analysis (n=11). Peripheral neutrophil counts significantly correlated with plasma levels of S100A8, MMP8 and TREM-1 (figure 1A-C), but not with the other measured mediators nor with any BALF levels. Concentrations of MPO, S100A8, MMP-8, PTX-3, IL-8, and TREM-1 were significantly higher in BALF (figure 1D), but not plasma from bronchoscopy patients with pulmonary infections (TB and other) compared to non-infectious patients. Only MPO concentrations were elevated in BALF from TB patients versus other patients with infection (38.8 μg/ml vs 20.6 μg/ml, p=0.04, figure 1E). Finally, BALF MPO and LRG1 show a significant positive correlation with lung pathology severity (figure 1F-G).

Conclusions Taken together, our data show that key neutrophil-associated mediators were elevated locally in the lungs in patients with pulmonary infections, but not in the blood. Specifically, MPO was disproportionately raised in the lungs of TB patients compared to patients with other infections or individ-



Abstract P62 Figure 1 Correlation of plasma concentrations of (A) S100A8, (B) MMP-8, and (C) TREM-1 with peripheral blood neutrophil counts in bronchoscopy patients (n=76). Orange dots represent TB patients (n=17); black dots represent non-TB patients. Spearman's correlation coefficient (r) and p values indicated in graphs.(D) BALF concentrations of MPO, S100A8, MMP-8, PTX-3, and IL-8 comparing bronchoscopy patients with chest infection (n=39) versus patients without infections (n=37), Mann-Whitney test, *p<0.05, ** p<0.01. (E) BALF MPO concentrations of TB patients (n=17) versus patients with other chest infections (n=22), Mann-Whitney test, 'p<0.05. Correlation of (F) BALF MPO and (G) BALF LRG1 concentrations with chest X ray severity TIMIKA scores, linear regression, Spearman's correlation coefficient (r) and p values indicated on graph

uals with non-infectious lung diseases, and local concentrations of MPO and LRG1 were associated with lung pathology, indicating a potential role in TB immunopathology.

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P63 MAPPING THE DYNAMICS OF HOST TRANSCRIPTIONAL RESPONSES AND SPUTUM BACTERIOLOGICAL CLEARANCE DURING TREATMENT FOR PULMONARY TUBERCULOSIS

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Monitoring antimycobacterial treatment response in pulmonary tuberculosis disease relies on *Mycobacterium tuberculosis* (*Mtb*) sputum load quantification. However, patients frequently stop producing sputum and/or the *Mtb* load drops below the limit of detection within the first weeks of treatment despite evidence from previous studies indicating *Mtb* remains within the lung. This limits the ability to accurately monitor antituberculosis treatment response for both clinical management and drug development. We investigated host immune response dynamics alongside sputum bacillary load to assess their potential as complementary treatment response markers.

Longitudinal data were available from two independent and parallel cohorts: adults with drug-sensitive pulmonary tuberculosis (n = 48; TB-PKPD study) receiving standard therapy in London, UK, and adults with rifampicin-resistant tuberculosis (n = 260; SHIFT-TB study) receiving bedaquiline, clofazimine, linezolid and levofloxacin for nine months in East London, South Africa. Sputum, for bacterial load quantification, and whole blood, for host immune response marker quantification, were collected from each patient for as long as they were on treatment. Most samples were collected at baseline, two weeks and two months after treatment initiation, when reductions in overall bacterial numbers and changes in host immune response were hypothesised to be greatest.

Sputum bacillary load was quantified using Mycobacteria Growth Indicator Tube time to positivity (MGIT-TTP). Expression of 35 pre-defined host genes was measured using NanoString, and eleven published transcriptional signatures were derived (Sweeney3, Suliman4, Risk6, Gliddon3, Roe3, Zak11,

BATF2, FCGR1ABC, GBP2, SERPING1 and ETV7). Correlations between published gene signatures ranged from 0.44 to 0.96 in SHIFT-TB and 0.20 to 0.97 in TB-PKPD.

Across cohorts, host transcriptional responses showed heterogeneous patterns over time; some patients demonstrated little change in z-score trajectories while others displayed marked declines. The Suliman4 signature showed pronounced early dynamics; patients with larger decreases generally had higher baseline z-scores. However, changes in host transcriptional responses were not associated with baseline sputum bacillary load, smear status, or bacillary load dynamics over time.

The findings in two separate cohorts suggest that bacteriological and host-immune response measures do not closely align during treatment, supporting the potential role of complementary biomarkers to improve assessment of treatment response in tuberculosis.

P64 TB PCR TESTING IN NON-SPUTUM SAMPLES TO REDUCE DIAGNOSTIC DELAYS: A RETROSPECTIVE CASE SERIES

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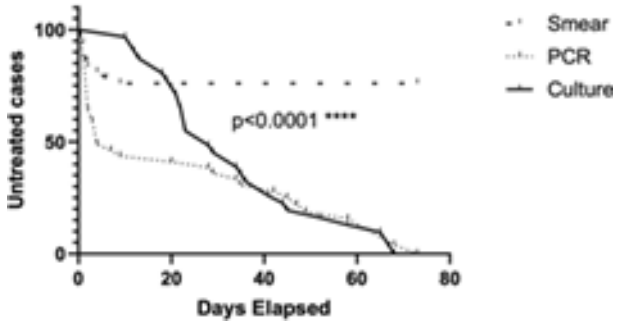
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Introduction Tuberculosis (TB) incidence is increasing in the UK.[1] Earlier diagnosis and treatment of TB is an immediate public health need. TB PCR offers rapid TB diagnosis. National Institute for Clinical Excellence guidelines recommend TB PCR testing in HIV-negative adults if it will change management.[2] However, this test is not available in all UK hospitals, especially for non-sputum samples, potentially contributing to treatment delays.

Methods We reviewed 49 positive TB cases from 1st January 2025 to 1st October 2025, to compare the time to treatment for those who had initial TB PCR testing (Cepheid GenexpertÒ) with other methods of diagnosis in sputum and non-sputum samples.

Results TB PCR was positive in 41 cases (83.7%). Smear and culture positive cases were 24.5% and 67.3%, respectively. In 34 non-sputum samples, 27 cases (79.4%) were TB PCR positive. This was the only positive result in 29.6% of samples.

Initial TB PCR testing in all samples (n=19) had a significantly shorter time to treatment than those without PCR testing (4.7 days *vs* 21.6 days, *t test* p = 0.03). This was also true for non-sputum samples (7.0 days *vs* 37.4 days, *t test* p = 0.009). Kaplan-Meier analysis (figure 1) demonstrates significantly reduced time to treatment when using TB PCR in all samples compared to culture and smear (*log rank* p<0.0001).



Abstract P64 Figure 1 Time to commencing TB treatment

Conclusion We demonstrate reduced time to anti-tuberculous therapy commencing when TB PCR testing is utilised on the initial sample in both sputum and non-sputum samples. This study highlights the need for more UK hospitals to have immediate access to TB PCR testing across all sample types in order to help reduce rising TB incidence.

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P65 SURVEY OF ADENOSINE DEAMINASE (ADA) TESTING PRACTICES FOR PLEURAL TUBERCULOSIS ACROSS HOSPITALS IN UK

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Introduction Pleural adenosine deaminase (ADA) is a validated biomarker for pleural tuberculosis infection, however implementation across NHS trusts is unclear, with variation in assay types, reference ranges and testing processes between centres. We therefore sought to characterise national practice and consider whether current approaches are fit for purpose in terms of diagnostic utility.

Methods A cross-sectional survey was emailed to clinicians in respiratory medicine, infectious diseases, clinical laboratory, and TB services across NHS trusts in UK, identified via regional respiratory networks. Questions addressed ADA availability, methodology, and perceived clinical utility.

Results 29 responses were received from 27 trusts. 17 (59%) reported currently using ADA testing for pleural TB diagnosis, whilst 12 (41%) did not. Among users, 11 (65%) sent samples to an external laboratory and 6 (35%) performed testing in-house, with 1 non-responder. Many external samples were directed to a small number of specialist centres. Notably, no respondents could confirm the assay methodology used, reflecting the predominance of external referral. Reporting units varied with 14 (74%) reporting in IU/L and 2 (11%) in U/L. The most cited reference range was ≤35 IU/L, though multiple respondents were unable to confirm their trust's threshold. Turnaround time was approximately two days for in-house users; however, delays of several weeks were reported for externally referred samples. Annual testing volumes were low, with 45% of trusts sending 0–50 samples per year.

Perceived clinical utility was modest: 32% rated ADA as moderately useful as a supplementary tool, 27% as highly useful, 18% as having limited utility, and 14% as not useful. Themes identified by respondents included a preference for thoracoscopy and PCR-based diagnosis, low local TB incidence limiting test utility, high external assay costs, and sample failures due to haemolysis.

Conclusion ADA testing availability and practice for pleural TB is highly variable across English NHS trusts. Logistical challenges and assay heterogeneity make it challenging to determine

whether ADA has a useful role in UK TB diagnostics. Our findings highlight the need for services to understand what tests are available locally and how they fit within the diagnostic pathway for pleural TB, particularly in higher-volume centres.

P66 GENOMIC CHARACTERISTICS OF MYCOBACTERIUM TUBERCULOSIS AMONG SMALL-SCALE MINERS IN NORTHERN TANZANIA

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10.1136/thorax-2026-BTSabstracts.227

Tuberculosis prevalence is high among small-scale miners and surrounding communities. Previous studies suggest that, although clustering is considered common in high TB incidence settings, it is often difficult to identify epidemiological links. Whole genome sequencing (WGS) allows accurate identification of transmission events. For the first time in a mining setting, we applied WGS to describe circulating strains of *Mtb* and identify clustering according to miner, ex-miner and community member status.

Mtb isolates from a cross-sectional study of small-scale miners in Mererani, Northern Tanzania, along with stored samples from another community-based TB study in the same area, were sequenced on a Oxford Nanopore Technologies (ONT) MinION Mk1B device using R10.4 flow cells. Bioinformatics was performed in Tanzania using a tailored pipeline; samples meeting a defined quality criteria were included in the analysis. Clustering was defined by isolates that differed by 12 or fewer single nucleotide polymorphisms (SNPs).

Of 52 unique *Mtb* culture isolates with extractable DNA that underwent WGS, three failed to meet quality criteria and one was found to be a novel Nocardia species. Of 48 remaining isolates, most (33/48, 68%) belonged to lineage 3, followed by lineage 4 (10/48, 21%), lineage 2 (3/48, 6%) and lineage 1 (2/47, 4%). All isolates were fully sensitive except one isolate with streptomycin resistance (gid c.102delG mutation). Eight clusters, each including two or three participants, included 20/48 (42%) samples (figure 1). No cluster included participants from the same mine and 6/8 (75%) of clusters included participants from more than one study group. Twenty-five percent of cases were estimated to be due to recent transmission.

Despite incomplete sampling, clustering appeared relatively common. However, epidemiological links between clustered *Mtb* cases were not identified. These results suggest generalised high levels of transmission between and within miners and the surrounding community and implies that community level approaches are needed to reduce TB in Mererani.

P67 CAN AN RD-1 BASED SKIN TEST STRATEGY IMPROVE THE TB INFECTION CARE CASCADE WHERE IGRA IS STANDARD?

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Background Achieving TB elimination requires expanded testing and treatment for TB infection (TBI). TB-specific skin tests, such as Cy-Tb (SIILTIBCY), have accuracy comparable to interferon-gamma release assays (IGRA). However, their acceptability, patient preference, and impact on patient outcomes and routine-care implementation remain unclear.

Abstract P67 Table 1 Primary and secondary outcomes

	Cy-Tb arm	Standard-of-care IGRA arm
Participants enrolled	76	50
Primary outcome (LTBI treatment initiation within 12 weeks)		
Primary analysis, n/N (%, 95% CI)	3/76 (3.9%, 1.4-11.0%)	0/49 (0%, 0-7.3%)
Process outcomes		
Acceptance of Cy-Tb testing, n/N (%)		
– Fixed testing strategy	15/15 (100%)	
– Opt-out testing strategy	10/61 (16%)	
Failure to have Cy-Tb test read within 2-3 days among participants receiving Cy-Tb test, n/N (%)	0/25 (0%)	
Positive LTBI test result among participants receiving the allocated LTBI test, n/N (%)	10/76 (13%)	1/49 (2%)
Initiating LTBI treatment within 12 weeks among participants receiving Cy-Tb test, n/N (%)	3/25 (12%)	
Initiating LTBI treatment within 12 weeks among participants testing Cy-Tb positive, n/N	3/4	
Acceptance of LTBI treatment among participants, determined by verbal agreements, n/N (%)	4/76 (5%)	1/49 (2%)
Loss to follow up among participants testing LTBI positive	3/10 (30%)	0/1

Methods We conducted a randomised comparison of Cy-Tb against standard-of-care IGRA in individuals eligible for TBI testing and treatment in UK. Two Cy-TB diagnostic testing strategies were used sequentially: (i) *Fixed*, where participants were directed to receive Cy-TB without the option to decline (October 2021 to November 2023); (ii) *Opt-out*, allowing participants to choose IGRA instead (December 2023-September 2024). Outcomes included treatment initiation, process measures, and safety.

Results Of 353 individuals invited, 126 were enrolled. 76 were randomised to Cy-Tb and 50 to IGRA. 15 participants were offered Cy-TB during the *Fixed* strategy period and 61 in the *Opt-out* period. Ten of 61 participants (16%) accepted Cy-Tb testing.

Overall, 10/76 participants (13%) in the Cy-Tb arm had reactive tests, including four tested with Cy-Tb and six who opted for IGRA. Three participants with reactive Cy-Tb tests initiated preventive treatment (3.9%, 95% CI 1.4–11.0%); one was ineligible due to active TB.

None of the six participants who chose IGRA initiated treatment. Among those offered IGRA, one participant tested positive but did not initiate treatment.

The commonest reason for opting out of Cy-Tb was unwillingness to return for test reading (33/51, 64%).

Conclusions Cy-Tb uptake was low in a setting where IGRA is routine for TB infection testing; unwillingness to return for test reading impeded implementation. However, all individuals who accepted Cy-Tb completed reading, pre-treatment assessment and initiated treatment. Innovations such as remote reading should be explored to improve Cy-Tb uptake. Given its low cost and potential to increase treatment initiation, Cy-Tb warrants further implementation and cost-effectiveness evaluation.

P68 TUBERCULOSIS INFECTION CARE IN MIGRANTS NEEDS IMPROVED ACCESS TO ADEQUATE ONLINE EDUCATIONAL RESOURCES TO IMPROVE OUTCOMES

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Background Tuberculosis infection (TBI) screening and treatment in migrant populations can be complex due to language barriers, low health literacy, and limited access to adequate educational resources.

Methods An online survey was distributed through existing networks to healthcare professionals involved in TBI screening and treatment across the UK. The questionnaire explored the use of educational resources, perceptions of resource quality, and barriers to communicating TBI information to migrants. Descriptive statistical analyses were performed using Stata 19MP. Comparisons between healthcare professional were undertaken using Chi-square or Fisher's exact tests where appropriate.

Abstract P68 Table 1 Online educational resources used by healthcare professionals for migrant populations

Online resources	TB consultants	TB nurses and pharmacists	p-value
Active TB			
Explain TB	9/27 (33%)	14/23 (61%)	0.05
WHO TB fact sheets	10/26 (38%)	6/23 (26%)	0.36
CDC fact sheets	3/26 (12%)	2/23 (9%)	0.74
The truth about TB (TB Alert)	18/27 (67%)	21/23 (91%)	0.04
There are no adequate resources	2/27 (7%)	3/23 (13%)	0.51
TB infection			
WHO TBI guidelines	2/27 (7%)	3/23 (13%)	0.51
NICE TB guidelines	0/27 (0%)	2/23 (9%)	0.12
TB Alert latent TB toolkit	11/ 27(41%)	9/23 (39%)	0.91
UKSA Latent TB testing and treatment: leaflet	8/27 (30%)	10/23 (43%)	0.31
There are no adequate resources	8/27 (30%)	7/23 (30%)	0.95

Results Fifty healthcare professionals completed the survey, including 27 TB consultants and 23 allied healthcare professionals (TB nurses and pharmacists). 40/ 50 respondents (80%) had worked in TBI services for more than 10 years. One third of respondents reported that no adequate resources were available for TBI but 40/50 (80%) felt that there were resources available for active TB. Use of educational resources varied between professional groups. TB nurses and pharmacists were significantly more likely than TB consultants to use the resource '*Explain TB*' (14/23 vs 9/27, p=0.05) and *the TB Alert resource The Truth About TB* (21/23 vs 18/27, p=0.04). (Table 1) Overall, TB Alert resources were the most frequently used educational materials by healthcare professionals (39/50, 79%)), whereas resources from the WHO, CDC, UKHSA and NICE were rarely used. Routine use of educational resources in clinical practice was however uncommon, reported by only 10/23 (43%) of TB nurses and pharmacists and 4/27 (15%) doctors. The most frequently reported barriers to explaining TBI to migrants were cultural beliefs/ misconceptions (33/50, 66%), language barriers (32/50, 64%), limited access to interpreters (31/50, 62%), low health literacy (25/50, 50%), and lack of adequate educational materials (24/50, 48%).

Conclusions Online educational resources for migrants with TBI are perceived as limited and are infrequently used in routine clinical practice. Language barriers, cultural beliefs, poor interpreter access, and low health literacy remain significant obstacles to effective migrant care. Interventions including multilingual and culturally appropriate educational resources may improve patient understanding, increase uptake and completion of TBI treatment.

P69 DECISION-MAKING IN LATENT TUBERCULOSIS TESTING: QUALITATIVE INSIGHTS ON NOVEL TB-SPECIFIC SKIN TESTS

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10.1136/thorax-2026-BTSabstracts.230

Background Novel TB-specific skin tests (TBST) might offer a cost-effective alternative to the blood-based QuantiFERON-TB Gold Plus assay (IGRA) for diagnosing latent tuberculosis infection (LTBI). However, TBST are not currently included in routine clinical practice in the UK. This study therefore examined how healthcare service users and providers make decisions when both IGRA and TBST are available.

Methods We carried out a cross-sectional qualitative study embedded within a diagnostic randomised controlled trial. Service users who were offered TBST through an opt-out approach, choosing either to accept TBST or decline and undergo IGRA instead, participated in individual semi-structured interviews along with healthcare providers. Reflexive thematic analysis was used to examine decision-making processes, which were then interpreted using the Perceptions and Practicalities Approach.

Results Fourteen participants were interviewed, including seven service users (TBST acceptors [n = 4] and decliners [n = 3]) and seven healthcare providers. Service users highlighted the rapid turnaround of TBST results and the option to avoid needle-related anxiety as advantages; however, concerns about the test's trustworthiness and safety reduced their willingness to choose it when offered. The requirement to return to the clinic two to three days after the initial intradermal injection also contributed to some participants declining TBST.

Healthcare providers expressed mixed opinions about offering TBST. While some were uncertain about its clinical value, others viewed the need for a follow-up visit to receive results as an added burden within already busy TB clinics. In contrast, some clinicians saw this follow-up requirement positively, as it enabled the provision of additional testing options and supported more person-centred care.

Conclusions If TBST were introduced into routine clinical care, it would be important to address both the perceptual and practical barriers experienced by service users and healthcare providers. Future studies should focus on developing patient-centred communication approaches, co-designed with patients and service providers, to improve the acceptability of new TBST.

P70 WHAT IS THE IMPACT OF RADIOLOGY REFERRER ALERTS ON TUBERCULOSIS DIAGNOSES? EXPERIENCE FROM A SINGLE CENTRE

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10.1136/thorax-2026-BTSabstracts.231

Background Tuberculosis (TB) GIRFT ('Getting It Right First Time') aims to optimise TB care nationally. To reduce time to diagnosis, and thereby interrupt TB transmission, GIRFT recommends direct referral from Radiology to TB services when images are suspicious for active TB. Our trust implemented Radiology referrer TB alerts (RRA) in June 2022.

Aims

1. To describe the number of RRA and diagnostic yield for cases of active TB.
2. To examine the impact of RRA on time to diagnosis and treatment for pulmonary TB.

Methods Single-centre retrospective observational cohort study of all patients referred to the TB clinic from June 2022 to March 2026.

Results 240 RRA were sent for 227 individual patients.

Of these, 113/240 (47%) were chest x-ray alerts. 115/240 (48%) were chest CT scans, including combinations of chest, abdomen and pelvis acquisitions. 4/240 (2%) were MRI scans (3 neck, 1 spine). 4/240 (2%) were neck ultrasound scans.

51/240 (21%) led to diagnosis of active TB: 37/51(73%) with pulmonary TB; 7/51(14%) with extrapulmonary TB; and 7/51(14%) with both pulmonary and extrapulmonary TB. 2/51(4%) were on TB treatment at the time of alert. 9/44 pulmonary cases (21%) were acid fast bacilli (AFB) sputum smear positive. 42/44 (95%) had fully sensitive TB; 2/44 (5%) had multi-drug-resistant (MDRTB); none had extremely-drug-resistant (XDRTB).

Concurrently, there were 205 notified cases of pulmonary TB (201 fully sensitive, 4 MDRTB, 0 XDRTB). Therefore, 42/205 (20%) were connected to care via RRA including 2/4 (50%) MDRTB cases.

Likely reflecting study power, we found no significant difference (p=0.47) in the median time from symptom onset to pulmonary TB treatment between RRA (66 days (interquartile range (IQR) 35, 121)) and non-RRA groups (75 days (IQR 41, 149)).

Conclusion RRA effectively connects patients to the TB service with a significant diagnostic yield of 21%. Substantial proportions of pulmonary cases detected via RRA had AFB sputum smear positive TB and MDRTB. Larger, multicentre studies should evaluate the impact of RRA on time from symptom onset to pulmonary TB treatment.

Biologics galore!

P71 A GLOBAL CLINICIAN SURVEY OF OCS STEWARDSHIP IN ASTHMA EXACERBATIONS: TIME FOR T2-GUIDED TREATMENT?

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10.1136/thorax-2026-BTSabstracts.232

Background Oral corticosteroids (OCS) are widely used for acute asthma exacerbations but are associated with cumulative morbidity. Emerging evidence suggests treatment response may differ by inflammatory phenotype, with patients with type 2 (T2)-low disease potentially deriving less benefit. Understanding clinician perspectives is important to inform OCS stewardship and future trials.

Aim To evaluate global clinician views on OCS prescribing for acute asthma exacerbations and attitudes towards T2 biomarker-guided treatment.

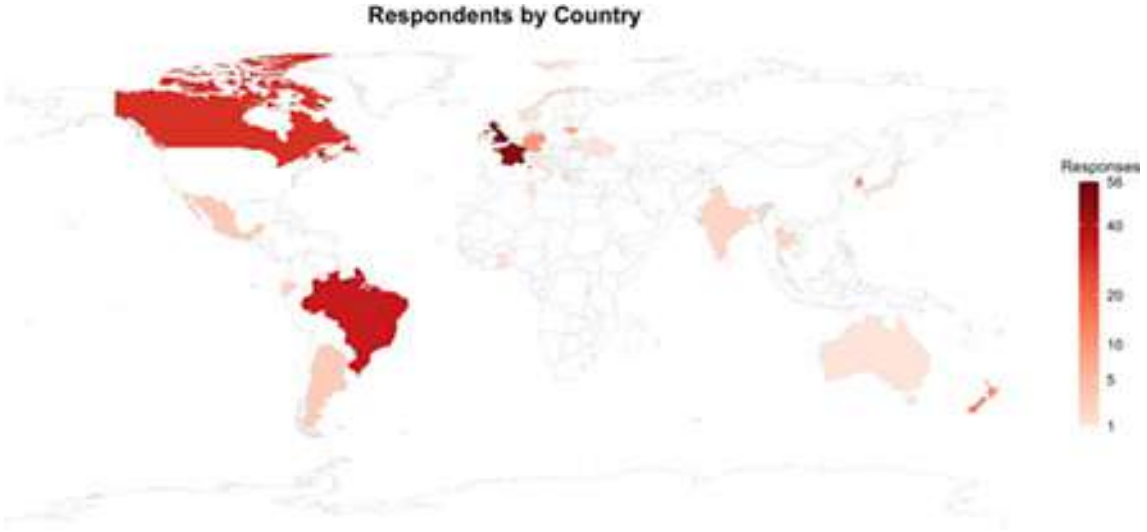
Methods A 24-item online survey was distributed internationally to healthcare professionals involved in asthma care. Questions explored clinical factors, lung function, T2 biomarkers and comorbidities influencing OCS prescribing. A Portuguese-language version was combined with the English survey. One non-consenting respondent was excluded.

Results Overall, 227 consenting respondents from 26 countries were included; 52% (n=119) were female and 57% (n=129) were consultant/attending pulmonologists. Most had substantial asthma experience, with 68% (n=154) caring for asthma patients for more than 10 years, and 66% (n=149) managing exacerbations at least weekly.

Views on universal OCS prescribing were divided. For patients presenting to primary care with asthma exacerbations, 45% (n=104) agreed or strongly agreed that *all exacerbations* should receive OCS, while 46% (n=105) disagreed or strongly disagreed. A higher proportion agreed with this statement for secondary care presentations (54%, n=123), although 37% (n=83) disagreed or strongly disagreed.

Patient-reported symptom intensity was the highest-ranked clinical factor guiding OCS initiation, followed by wheeze and peak flow. When asked if PEFR or FEV1 guided treatment decisions 59% (n=134) reported they did, but 33% (n=76) reported not using lung function thresholds for definite OCS prescription. Most respondents did not use a fixed blood eosinophil cut-off (58%, n=129); among those who did use a cut-off, >300 cells/μL was the commonest, utilised by 61% (n=58). 78% (n=176) of respondents agreed that some exacerbations could be safely managed without OCS. There was strong support for further evaluation, with 91% (n=205) stating that a clinical trial of T2-guided treatment is needed.

Conclusion International practice varies substantially, but there is broad support for OCS stewardship and biomarker-informed management of acute asthma exacerbations. These findings support prospective trials of T2-guided exacerbation treatment.



Abstract P71 Figure 1

P72

REAL-WORLD EXACERBATION PHENOTYPING IN SEVERE ASTHMA TREATED WITH ANTI-IL5/5RA, ANTI-IL4RA AND ANTI-TSLP

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10.1136/thorax-2026-BTSabstracts.233

Background Biologic therapies are highly effective at reducing exacerbations in severe asthma. Several target type 2 (T2) inflammation but act on different cytokines, IL-5, IL-4 and TSLP, with different downstream effects.

Aim To characterise exacerbation phenotypes in real-world severe asthma patients treated with anti-IL-5/5Ra, anti-IL-4Ra or anti-TSLP.

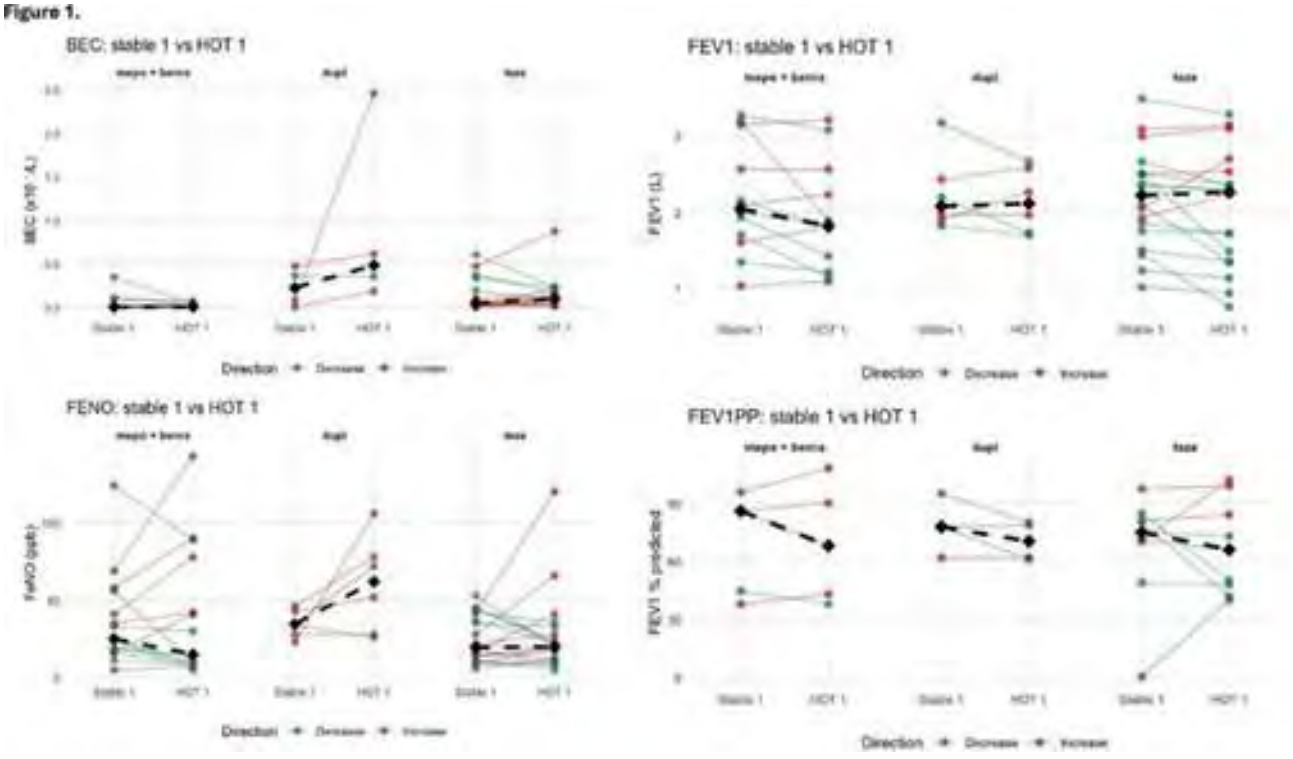
Methods Severe asthma patients on biologic therapy at a single tertiary centre were included if they attended a stable assessment after ≥3 months of biologic treatment plus ≥1 exacerbation visit. Patients were advised not to start or increase oral corticosteroids or antibiotics before exacerbation visits. Demographics, clinical characteristics, biomarkers (FeNO, blood eosinophil count [BEC]) and spirometry were recorded when stable and repeated at exacerbation. T2 exacerbations were defined as FeNO ≥50 ppb or a rise in BEC. Univariable linear regression assessed associations between biomarker change (ΔBEC, ΔFeNO) and FEV1 change (ΔFEV1), stratified by biologic class.

Results Of 67 patients who attended for both stable and exacerbation visits, median age was 56 (17–82), 63% were female and median BMI 29 (19–48). At baseline, median BEC was 0.0 (0–0.06), FeNO 28 (5–124), CRP 4 (1–16) and FEV1 78% predicted (40–100). Group-level biomarkers and spirometry were similar at exacerbation (BEC 0.1 [0–2.5], FeNO [5–144], CRP 3 [1–26], FEV1 72% predicted [40–134]).

56 (84%) patients had paired biomarkers from stable and exacerbation visits: 24 (43%) were on anti-IL-5/5Ra, 24 (43%) were on anti-TSLP and 8 (14%) were on anti-IL-4Ra. Biomarker changes were highly heterogeneous at the individual level within each class (figure 1). According to FeNO, 17%, 8% and 50% of exacerbations on anti-IL5/5Ra, anti-TSLP and anti-IL-4Ra were T2, whereas according to BEC rise, 4.2%, 38% and 38% respectively. In patients on anti-IL-5/5Ra, neither ΔFeNO nor ΔBEC was associated with ΔFEV1. In patients on anti-TSLP, ΔBEC was negatively associated with ΔFEV1 (β −1.82 [−3.44 to −0.20], p=0.03), but ΔFeNO did not correlate with ΔFEV1.

Conclusion In this real-world cohort, exacerbation phenotypes differed by biologic class. These were predominantly T2 on anti-IL4Ra, partly T2 on anti-TSLP (identified using blood eosinophils rather than FeNO), and rarely T2 on anti-IL-5/5Ra.

Figure 1.



Abstract P72 Figure 1

P73

BENRALIZUMAB DOES NOT ALTER THE AIRWAY MICROBIOME IN SEVERE ASTHMA, BUT EXACERBATIONS ARE ASSOCIATED WITH INCREASED DYSBIOSIS IDENTIFIED BY THE LOG2 PSEUDOMONADOTA:BACILLOTA RATIO

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10.1136/thorax-2026-BTSabstracts.234

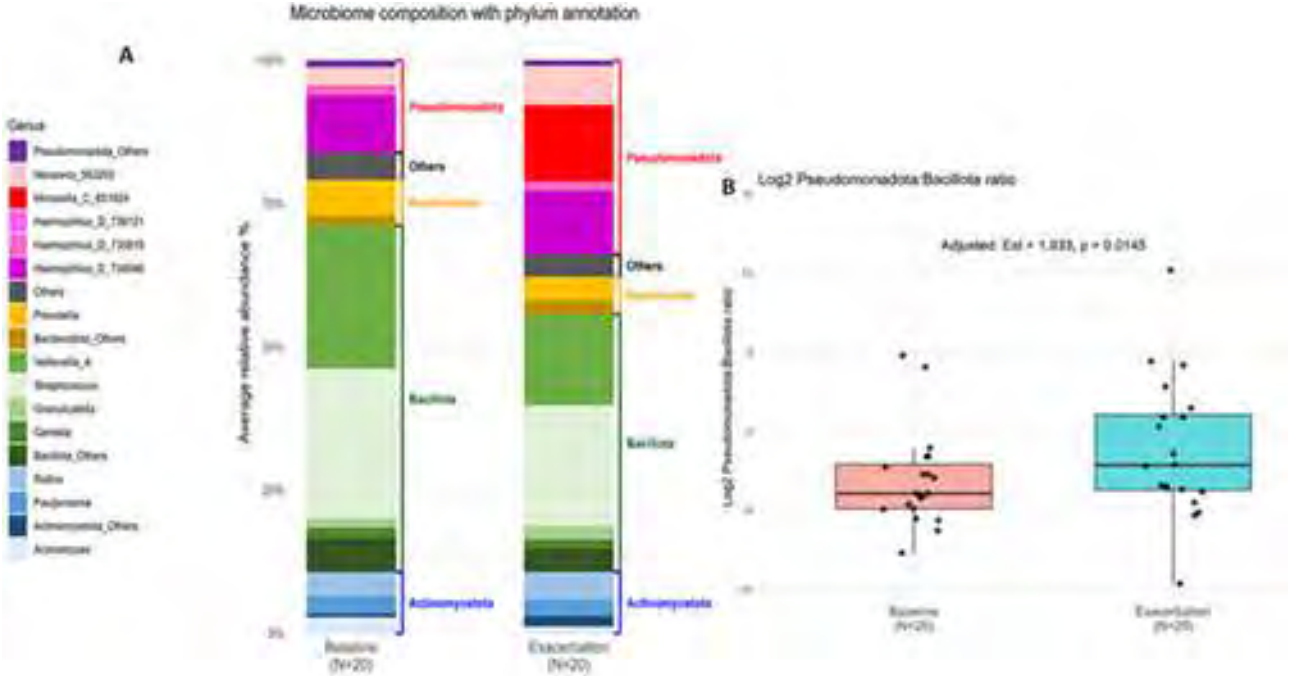
Background The airway microbiome may contribute to disease activity in severe asthma, although the impact of benralizumab on microbial communities remains unclear. The $\log_2$ Pseudomonadota:Bacillota (P:B) ratio has emerged as a potential marker of airway dysbiosis.

Objectives To assess longitudinal changes in the airway microbiome during benralizumab therapy and evaluate the utility of the $\log_2$ P:B ratio as a marker of exacerbation-associated dysbiosis.

Methods Airway microbiome composition at baseline, one year, and exacerbations was analysed using mixed-effects models, diversity metrics, differential abundance testing, and application of the ratio between the phyla Pseudomonadota:Bacillota (P:B), a measure of bacterial dysbiosis.

Results 86 baseline, 40 exacerbation samples (n=20 paired), and 38 one-year samples (n=30 paired) were included in the analysis. The airway microbiome remained stable over one year of benralizumab treatment, with no changes in the $\log_2$ P:B ratio (estimate = 0.2, p = 0.625), Shannon diversity (estimate = 0.009, p = 0.891), Simpson diversity (estimate = 0.015, p = 0.551), or Pielou's evenness (estimate = 0.003, p = 0.894). Beta diversity analysis demonstrated no temporal shift in community composition (PERMANOVA R^2 = 0.007, p = 0.333). There was no change in the relative abundance of any taxa from baseline to 1 year. In contrast, the $\log_2$ P:B ratio increased during exacerbation. This shift was more pronounced in the paired analysis (figure 1, estimate ≈ 1.93, p ≈ 0.015) than in the full sample set (estimate = 1.126, p = 0.0439). Similarly, beta diversity analysis identified modest but significant compositional differences during exacerbation in both datasets (all samples: R^2 = 0.018, p = 0.022; paired samples: R^2 = 0.039, p = 0.012).

Conclusion The stable airway microbiome in severe asthma was not affected by benralizumab therapy, but changes were observed at exacerbation. The $\log_2$ Pseudomonadota:Bacillota ratio captured these changes and may represent a biomarker of exacerbation-associated microbial imbalance in severe asthma.



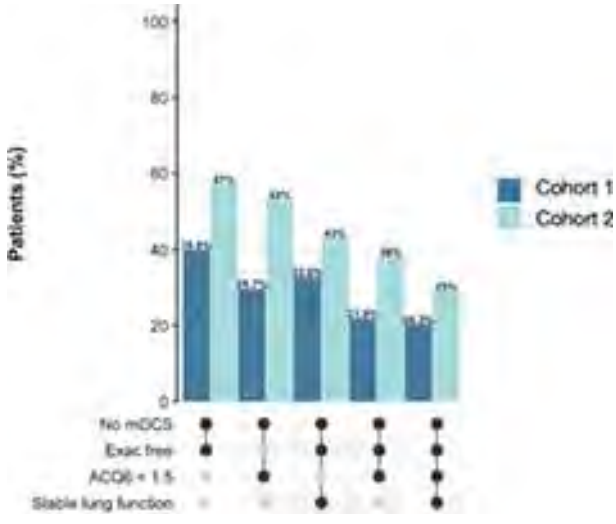
Abstract P73 Figure 1

P74 BENRALIZUMAB IN THE MODERN BIOLOGIC ERA: COMPARISON OF CLINICAL OUTCOMES FOLLOWING THE EXPANSION OF BIOLOGIC TREATMENT OPTIONS IN SEVERE ASTHMA

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10.1136/thorax-2026-BTSabstracts.235

Background Benralizumab is an anti-IL5 receptor monoclonal antibody licensed for severe eosinophilic asthma (SEA). Clinical trial and large real-world effectiveness analyses of benralizumab were published prior to the availability of other anti-T2 therapies including anti-IL4R and anti-TSLP. Whether current response rates to benralizumab have changed due to altered patient selection and additional treatment options is unknown.
Method We performed a comparison of clinical response rates with benralizumab in two large cohorts of patients with SEA under the same regional severe asthma centre. Our previously published 'Cohort 1' (n=130) received benralizumab prior to availability of alternative biologic therapies. 'Cohort 2' (n=100) received benralizumab following physician selection despite availability of other anti-T2 treatment options. Baseline characteristics and treatment outcomes were recorded including clinical remission (defined as no maintenance OCS for asthma, ACQ6<1.5, no exacerbation, and <5% FEV1 decline from baseline). Multivariable logistic regression to adjust for differences in baseline characteristics between the two cohorts was performed.
Results Compared with cohort 1 (patients initiated May 2018 – April 2019), cohort 2 (initiated June 2023 – Oct 2024) was older (59.4±15.5 vs 52.8±14.0 years, p=0.001), had lower BMI (29±6.0 vs 31.1±7.1 kg/m^2, p=0.004), lower maintenance OCS use (29% vs 56.9%, p<0.001), higher baseline FEV1 (2.01±0.73 vs 1.76±0.69 L, p=0.008) and no prior biologic exposure (0% vs 49%, p=0.001). Numerically superior response rates were observed for cohort 2 including exacerbation-free status (57% vs 43%), being off mOCS (97% vs 81%), achieving ACQ<1.5 (54% vs 35%) and clinical remission (31% vs 19%). However, after adjustment for differences in baseline characteristics, only odds of achieving ACQ<1.5 and weaning off mOCS remained significant (both p<0.05).
Conclusions Clinical response rates for benralizumab in SEA including the ability to achieve clinical remission are numerically superior to those first reported. This appears to reflect clinically relevant differences in baseline characteristics as more treatemnt options have become available.



Abstract P74 Figure 1 Remission outcomes in historical (Benra 1) and contemporary (Benra 2) severe eosinophilic asthma cohorts treated with benralizumab. Proportions of patients achieving individual and composite remission domains at 48 weeks are shown. Remission domains included absence of mOCS, exacerbation-free status, symptom remission and stable lung function. Composite outcomes are represented by connected dots beneath each bar pair

P75 TEZEPELUMAB IN PATIENTS RECEIVING BENRALIZUMAB FOR EGPA - STEROID SPARING EFFECT OF DUAL BIOLOGIC THERAPY

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10.1136/thorax-2026-BTSabstracts.236

Background Eosinophilic granulomatosis with polyangiitis (EGPA) is a multisystem inflammatory vasculitic disorder characterised by severe eosinophilic asthma (SEA), eosinophilic tissue infiltration and organ damage. Benralizumab, an anti-interleukin-5 receptor α monoclonal antibody licensed for refractory EGPA has demonstrated efficacy in reducing disease activity and corticosteroid exposure. However, approximately one-third of patients fail to achieve clinical remission, suggesting the involvement of additional inflammatory pathways. Thymic stromal lymphopoietin (TSLP), an epithelial-derived cytokine involved in type 2 inflammation, is elevated in EGPA and genetic variants in the TSLP pathway have been associated with disease susceptibility, making it a potential therapeutic target.
Methods We conducted a retrospective review of patients with EGPA receiving benralizumab who additionally commenced tezepelumab for uncontrolled severe eosinophilic asthma between November 2025 and March 2026. Clinical outcomes including Birmingham Vasculitis Activity Score (BVAS), Asthma Control Questionnaire-6 (ACQ-6), fractional exhaled nitric oxide (FeNO), and maintenance oral corticosteroid (mOCS) requirements were assessed at baseline and after four months of treatment.
Results Twelve patients (4 female; 44±10 years) completed ≥4 months of tezepelumab treatment. All received benralizumab for EGPA (30 mg every 4 weeks) for ≥1 month prior to tezepelumab initiation. At baseline, patients remained symptomatic despite treatment, with a median prednisolone dose of 10 mg/day (IQR 7.5–14), ACQ-6 of 2.3±1.3, BVAS of 1±1, and FeNO of 101 ppb (IQR 45–181). After four months, 6/12 patients achieved a >50% reduction in prednisolone dose, with the median dose falling to 5 mg/day (IQR 2.5–9; p=0.04). Improvements in ACQ-6 (−1±1) and BVAS (−0.75±0.7) were observed, while FeNO decreased significantly to 28 ppb (IQR 22–66; p=0.001). Combination therapy was well tolerated, with no treatment-limiting adverse events.
Conclusion In this small observational cohort, the addition of tezepelumab to ongoing benralizumab therapy was associated with improvements in oral corticosteroid requirements in patients with active EGPA. These findings support the hypothesis that TSLP-mediated pathways may contribute to persistent

disease activity despite eosinophil depletion and suggest that dual targeting of IL-5R and TSLP warrants further investigation in larger prospective studies.

P76 EFFECTIVENESS OF TEZEPELUMAB TREATMENT IN PATIENTS WITH SEVERE ASTHMA: REAL WORLD OUTCOMES FROM A SEVERE ASTHMA NETWORK

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10.1136/thorax-2026-BTSabstracts.237

Introduction Tezepelumab, a monoclonal antibody targeting thymic stromal lymphopoietin (TSLP), is licensed for severe asthma irrespective of inflammatory biomarkers. Pivotal trial data (NAVIGATOR) demonstrated significant reductions in exacerbations; however, real-world evidence in heterogeneous populations remains limited. This study evaluated real-world effectiveness across T2 inflammatory subgroups and identified predictors of clinical remission.

Methodology This multicentre retrospective cohort study was conducted across a regional severe asthma network (one tertiary centre; nine spoke hospitals). Adults meeting ERS/ATS severe asthma criteria fulfilling NICE prescribing criteria received tezepelumab 210 mg subcutaneously every four weeks. Data were collected prospectively over 12 months (2024–2026). Patients were stratified by T2 profile: T2-low (FeNO <25 ppb and BEC $<0.15\times10^9$/L), T2-intermediate, and T2-high (FeNO ≥25 ppb and BEC $\geq0.30\times10^9$/L). Endpoints were classified as positive (≥50% reduction in exacerbation frequency or mOCS dose), negative (including early discontinuation), or clinical remission (no severe exacerbations, off mOCS, and ACQ-6 <1.5).

Results 167 patients were included (113 female; mean age 50.0 years; median BMI 31.3 kg/m^2): 29 T2-low, 28 T2-intermediate, and 110 T2-high. Comorbidity burden was high; GORD (66%), rhinosinusitis (50%), and psychological comorbidity (37%) were most prevalent.

In patients with paired data (n=150), tezepelumab significantly improved all outcomes: AER fell from 5.0 to 1.0 (p<0.0001), ACQ-6 from 3.70 to 2.19 (p<0.0001), FEV_1% predicted from 68.5% to 79.5% (p<0.0001), and mOCS from 10.0 to 4.0 mg (p<0.0001). Blood eosinophils, FeNO, and IgE were significantly reduced (all p<0.001). Overall, 80% achieved a positive response and 20% (n=34) met remission criteria. Response rates were comparable across subgroups: 86% (T2-low), 88% (T2-intermediate), and 76% (T2-high). Logistic regression identified lower baseline ACQ-6 (OR 0.44, 95%CI 0.27–0.72, p=0.001) and BMI (OR 0.87, 95%CI 0.80–0.95, p=0.002) as independent predictors of remission. Early discontinuation occurred in 10%, predominantly due to rash and musculoskeletal adverse events.

Conclusion Tezepelumab demonstrated high positive response rates and significant improvements in OCS use, ACQ-6 score, and AER, comparable across all T2 phenotypes. Twenty percent achieved clinical remission; lower baseline ACQ-6 and BMI were positive predictors. These data support the efficacy of tezepelumab across a real-world severe asthma network.

Abstract P76 Table 1 Baseline clinical and phenotypical characteristics. *Values reflect available data; n is indicated where complete data were not obtained for the full cohort (N=167)'*

	N=167
Female n (%)	113 (68%)
Age mean (SD)	50.0 (47.8-52.2)
BMI median (IQR)	31.3 (27.5-38.0) (n=164)
Smoking **Mean pack years (SD)**	Non = 18, Ex/Current = 66 20.8 (95%CI 13.0-28.7) (n=50)
Nasal Polyps n (%)	29 (17)
Rhinosinus Disease n (%)	84 (50)
GORD n (%)	111 (66)
ILO n (%)	27 (16)
BPD n (%)	34 (20)
Psychology n (%)	61 (37)
OSA n (%)	27 (16)
Eczema n (%)	25 (15)
High-dose ICS n (%)	85 (51)
Medium-dose ICS n (%)	75 (45)
Low-dose ICS n (%)	7 (4)
Inhaled LAMA n (%)	127 (76)
Oral LRTA n (%)	131 (78)
Oral Methylxanthine n (%)	66 (40)
Previous Biologic n (%)	Total 53 (32) Xolair 23 (14), Mepolizumab 9 (5), Benralizumab 5 (3), two previous biologics 8 (5), three previous biologics 8 (5)
IgE pre median (IQR)	204.5 (68.5-732.5) (n=120)
FeNO pre median (IQR)	26.0 (12.0-58.0) (n=157)
Eosinophils pre median (IQR)	0.27 (0.11-0.50)
FEV1%p pre median (IQR)	69.0 (54.3-86.8) (n=144)

P77 MALIGNANCY OUTCOMES IN SEVERE EOSINOPHILIC ASTHMA PATIENTS TREATED WITH BENRALIZUMAB ACROSS TWO UK CENTRES

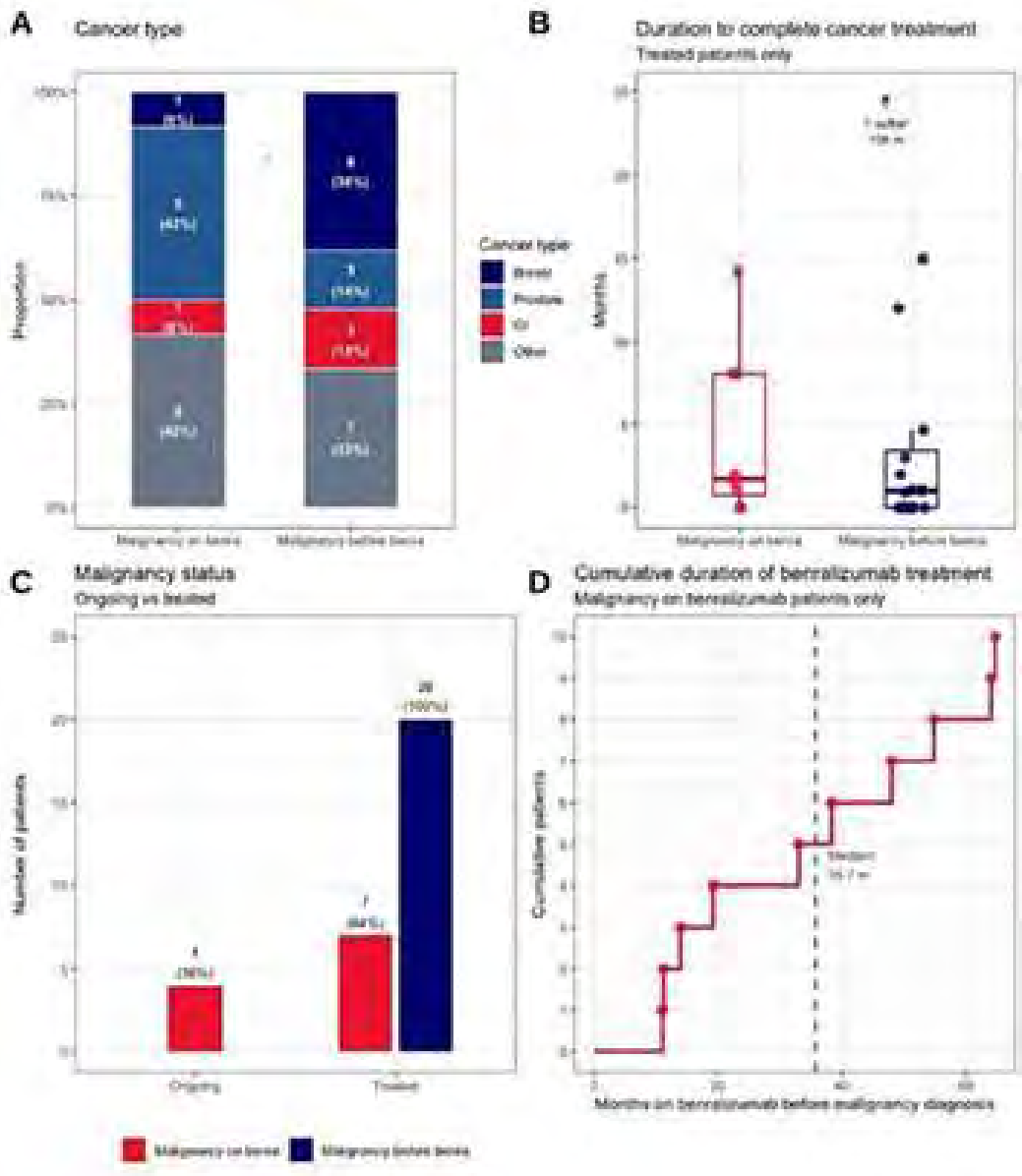
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10.1136/thorax-2026-BTSabstracts.238

Background Benralizumab, an anti-IL-5Rα antibody, is a highly effective treatment in severe eosinophilic asthma (SEA) and produces near-complete eosinophil depletion. Animal models have suggested a role for eosinophils in tumour surveillance. Whether this homeostatic function exists in humans remains unclear as the clinical impact of benralizumab on concurrent malignancy has not been studied in large patient cohorts.

Aim To evaluate malignancy outcomes in SEA patients diagnosed with malignancy whilst on benralizumab compared with those diagnosed prior to treatment initiation.

Methods Retrospective observational study of SEA patients attending two UK severe asthma centres who received



Abstract P77 Figure 1 Malignancy profiles and treatment outcomes according to timing of diagnosis relative to benralizumab exposure. (A) Malignancy type distributions in patients diagnosed before versus whilst receiving benralizumab, (B) Time to completion of malignancy treatment in successfully treated patients, (C) Current malignancy treatment status, and (D) Cumulative benralizumab exposure prior to malignancy diagnosis in patients diagnosed whilst receiving benralizumab

benralizumab and had a malignancy diagnosis. Patients were classified as malignancy 'before benralizumab' or 'on benralizumab' if diagnosed within 12 months of starting treatment, Outcomes included malignancy type, treatment status and time to curative treatment. Fisher's exact test and Mann-Whitney U test with Hodges-Lehmann estimation were used for categorical and continuous variables, respectively.

Results Of 1,750 patients treated with benralizumab, 44 (2.5%) had a malignancy diagnosis. Complete data were available for 33: 12 with malignancy on benralizumab and 21 before benralizumab. Groups were comparable for sex (67% male on benralizumab vs 33% before benralizumab, p=0.083) and smoking status (ex-smokers 25% vs 43%, p=0.457). Patients with malignancy on benralizumab were significantly older (65 vs 54 years, p=0.002) and had longer treatment exposure (5.1 vs 2.6 years, p=0.014). Prostate malignancy was more common in the on benralizumab group (42% vs 14%), while breast cancer was less prevalent (8% vs 38%). All 33 patients are alive. Curative treatment had been completed in all patients with malignancy before benralizumab, compared with 64% in the on benralizumab group, with 36% still under active treatment. This may reflect the significantly shorter follow-up from malignancy diagnosis in the latter group (3 vs 14 years, p<0.001).

Conclusion In a large real-world cohort, malignancy was identified in 2.5% of benralizumab-treated patients with no malignancy-related deaths. Differences in cancer treatment status maybe attributable to follow-up duration. The higher prevalence of prostate cancer in the on-benralizumab group warrants further investigation, given previous evidence linking tissue eosinophilia with favourable prostate cancer outcomes.

P78 EFFECT OF TEZEPELUMAB ON TYPE 2 INFLAMMATORY BIOMARKER LEVELS IN THE NASAL LINING FLUID OF PATIENTS WITH CRSWNP AND COMORBID ASTHMA

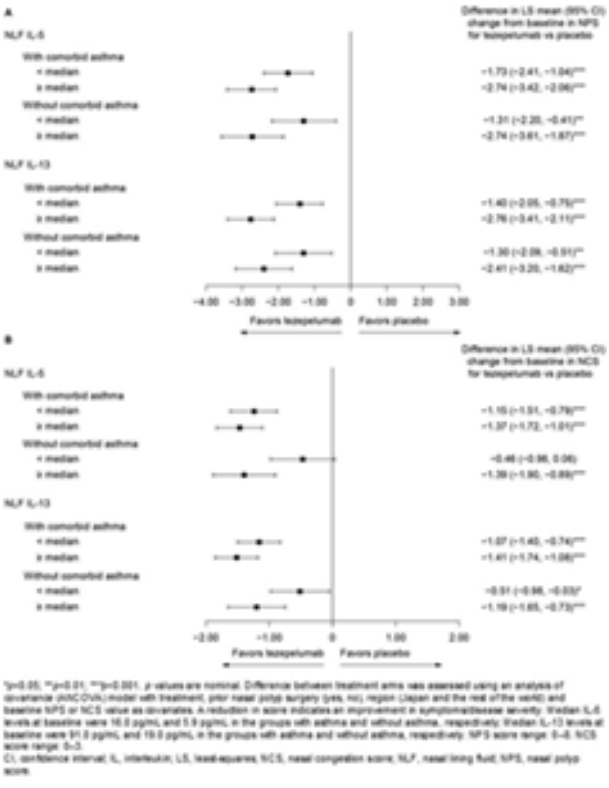
[1]BJ Lipworth, [2]C Marchese, [3]C Chen, [4]A Foster, [5]SS Ponnarambil, [6]J Reddy, [7]JR Parnes, [8]J Publicover, [9]JD Sherrill. [1]*Scottish Centre for Respiratory Research and ENT Rhinology Clinic, Ninewells Hospital and Medical School, University of Dundee, Dundee, UK;* [2]*Biometrics, Respiratory and Immunology, BioPharmaceuticals R&D, AstraZeneca, Barcelona, Spain;* [3]*Biometrics, Respiratory and Immunology, BioPharmaceuticals Medical, AstraZeneca, Gaithersburg, MD, USA;* [4]*Respiratory and Immunology, BioPharmaceuticals Medical, AstraZeneca, Gaithersburg, MD, USA;* [5]*Respiratory and Immunology, BioPharmaceuticals R&D, AstraZeneca, Cambridge, UK;* [6]*Global Clinical Development – Medical, Amgen, Thousand Oaks, CA, USA;* [7]*Early Development, Amgen, Thousand Oaks, CA, USA;* [8]*Precision Medicine, Amgen, South San Francisco, CA, USA;* [9]*Translational Science and Experimental Medicine, Research and Early Development, Respiratory and Immunology, BioPharmaceuticals R&D, AstraZeneca, Gaithersburg, MD, USA*

10.1136/thorax-2026-BTSabstracts.239

Background In WAYPOINT (NCT04851964), tezepelumab significantly reduced nasal polyp (NP) size and improved sino-nasal symptoms versus placebo in adults with uncontrolled chronic rhinosinusitis with NP (CRSwNP).

Objective This *post hoc* analysis evaluated the effect of tezepelumab on type 2 (T2) inflammatory biomarker levels in nasal lining fluid (NLF) and clinical outcomes among patients with comorbid asthma.

Methods WAYPOINT was a phase 3, multicenter, randomized, double-blind, placebo-controlled, parallel-group study. Adults (≥18 years) with uncontrolled CRSwNP were randomized (1:1) to tezepelumab 210 mg or placebo subcutaneously Q4W for 52 weeks. Change from baseline to weeks 24 and 52 in NLF IL-5 and IL-13 levels and relationships between baseline NLF cytokine levels and total NP and nasal congestion score at week 52 were explored.



Abstract P78 Figure 1 Difference in LS mean change from baseline to week 52 in NPS (A) and NCS (B) for tezepelumab versus placebo by median cytokine levels at baseline

Results This analysis included 345 patients (tezepelumab, n=174 [comorbid asthma, n=109]; placebo, n=171 [comorbid asthma, n=108]). The least-squares mean (95% CI) reductions in NLF IL-5 and IL-13 levels for tezepelumab versus placebo at week 52 were 90.4% (85.0%, 93.8%) and 93.1% (89.9%, 95.3%), respectively. Participants with comorbid asthma had higher median baseline levels of NLF IL-5 (16.0 vs 5.9 pg/mL) and IL-13 (91.0 vs 19.0 pg/mL) than those without comorbid asthma. For those with and without comorbid asthma, mean (95% CI) reductions in IL-5 at week 24 were 94.2% (90.0%, 96.6%) and 83.6% (67.6%, 91.7%), respectively, whereas reductions in IL-13 were 94.8% (91.5%, 96.9%) and 86.9% (75.1%, 93.1%), respectively. For those with and without comorbid asthma, reductions in IL-5 at week 52 were 90.4% (83.1%, 94.6%) and 90.1% (79.8%, 95.2%), respectively, and reductions in IL-13 were 94.0% (90.2%, 96.3%) and 91.2% (83.6%, 95.3%), respectively. In both groups, improvements in clinical outcomes at week 52 were more pronounced among patients with cytokine levels at or above the median at baseline (figure 1).

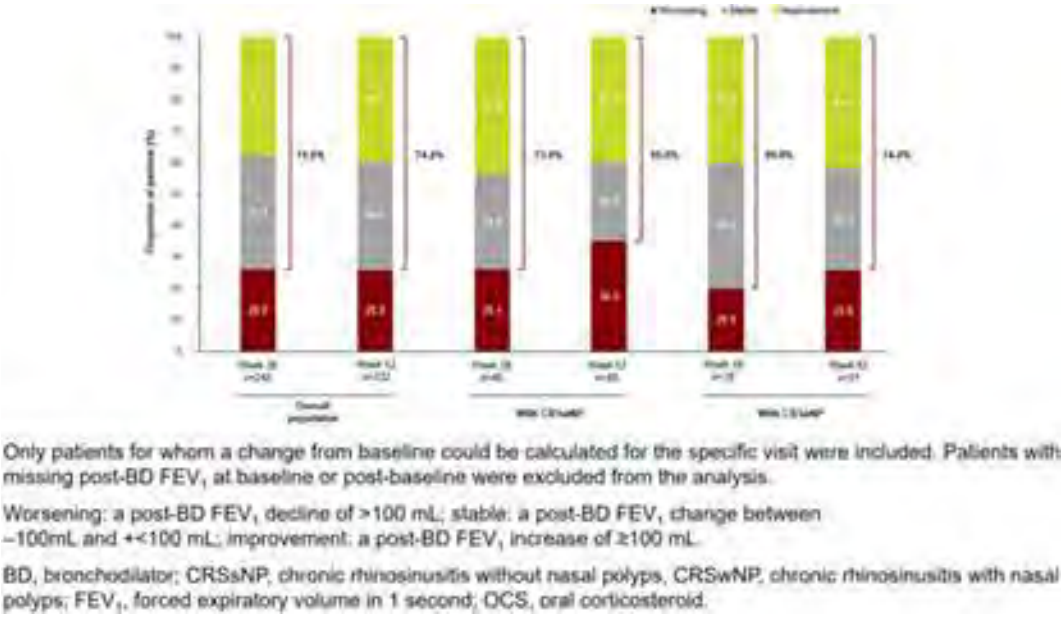
Conclusions Patients with CRSwNP and comorbid asthma had higher expression of NLF T2 cytokines at baseline than those without asthma, and tezepelumab reduced cytokine levels in both groups. Tezepelumab significantly improved clinical responses across subgroups, with numerically greater improvements in patients with CRSwNP and comorbid asthma versus without asthma or with higher versus lower baseline NLF T2 cytokine levels.

P79 EFFECT OF TEZEPELUMAB ON LUNG FUNCTION IN ORAL CORTICOSTEROID-DEPENDENT PATIENTS WITH SEVERE ASTHMA AND COMORBID CRSWNP OR CRSSNP IN THE PHASE 3B WAYFINDER STUDY

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10.1136/thorax-2026-BTSabstracts.240

Background In the phase 3b WAYFINDER study of tezepelumab in oral corticosteroid (OCS)-dependent patients with severe asthma (NCT05274815), ~90% of patients, including those with chronic rhinosinusitis (with/without nasal polyps [CRSwNP/CRSsNP]), achieved a maintenance OCS dose of ≤5 mg/day prednisone/prednisolone or discontinued OCS while



Abstract P79 Figure 1 Proportions of OCS-dependent patients with severe asthma and comorbid CRSwNP or CRSsNP who had worsening, stable or improved lung function at weeks 28 and 52 compared with baseline

maintaining asthma control. Lung function remained stable in the overall cohort (mean [95% confidence interval] change from baseline in post-bronchodilator [BD] FEV_1 at week 52: +70 mL [20–110]).

Objective This *post hoc* analysis assessed lung function in WAYFINDER participants with CRSwNP/CRSsNP.

Methods WAYFINDER was an open-label, single-arm study in adults with severe asthma receiving maintenance OCS. Patients received tezepelumab 210mg subcutaneously every 4 weeks for up to 52 weeks. Lung function was measured at baseline and at weeks 28 and 52. Improvement was defined as a post-BD FEV_1 increase of ≥100mL from baseline, stability as a change between −≤100mL and +<100mL, and worsening as a decline of >100mL.

Results Of the 298 tezepelumab recipients, 50 (16.8%) had CRSwNP and 39 (13.1%) had CRSsNP. At weeks 28 and 52, lung function improvement or stability was seen in 73.9–80.0% and 65.0–74.2% of patients across the overall, CRSwNP and CRSsNP groups, respectively (figure 1).

Conclusions Tezepelumab treatment resulted in improved or stable lung function over 28 and 52 weeks in over 65% of patients with OCS-dependent severe asthma and CRSwNP/CRSsNP, despite OCS tapering.

P80 TREATMENT RESPONSE AND CLINICAL REMISSION AFTER ONE YEAR OF TEZEPELUMAB IN BIOLOGIC-NAIVE SEVERE ASTHMA: A REAL-WORLD STUDY

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Background Tezepelumab has shown to reduce severe asthma exacerbations and improve clinical outcomes across asthma phenotypes (PMID:33979488). Clinical remission is emerging as an important treatment target in severe asthma, but real-world data on remission following Tezepelumab therapy remains limited. We evaluated treatment response and clinical remission after one year of Tezepelumab in biologic-naive severe asthma cohort.

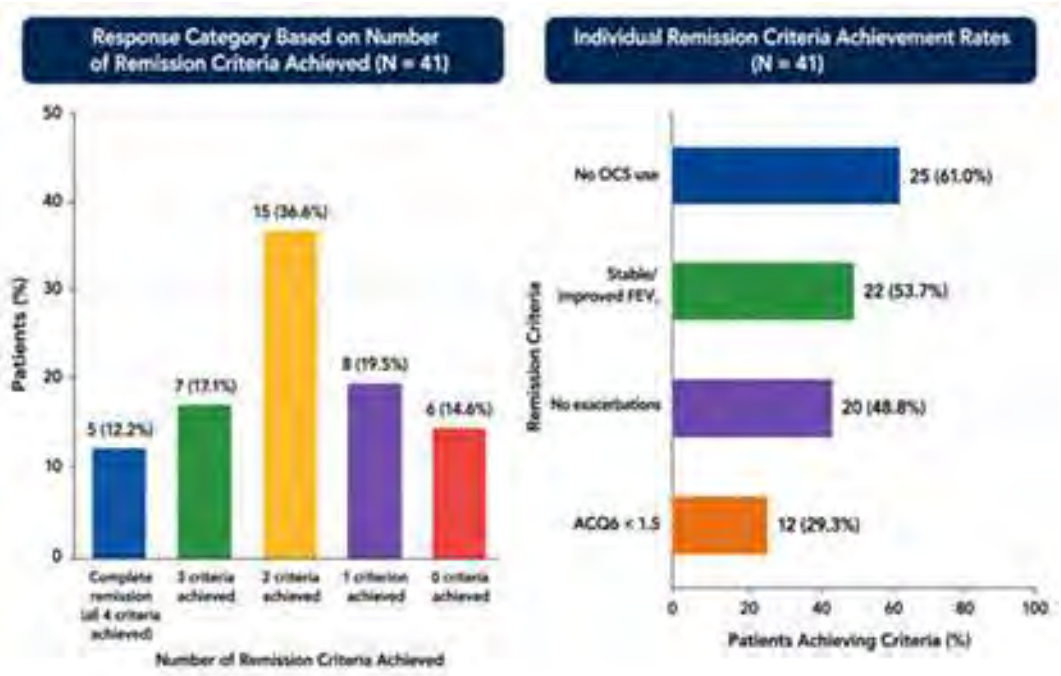
Methods We conducted a retrospective observational analysis of adults with severe asthma who were initiated on Tezepelumab between January 2024 and February 2025 and completed one year of treatment. Patients previously exposed to biologic therapy were excluded. Outcomes assessed at baseline and one year included annual exacerbations, mOCS dose, ACQ-6, FEV1, FeNO, and blood eosinophil count.

Clinical remission was defined as achievement of all four criteria: (1) no exacerbations, (2) no mOCS use, (3) ACQ-6 <1.5, and (4) stable or improved FEV1 compared with baseline.

Results Forty-one patients completed one year of therapy. Significant improvements were observed in asthma control, exacerbation burden, OCS exposure, and FeNO. ACQ-6 improved from a mean 2.99 to 1.33 ($p<0.001$), annual exacerbations decreased from median 5 to 0 ($p<0.001$), mOCS dose fell from mean 5.5 to 2.6 mg ($p<0.001$), and FeNO declined from median 28.5 to 21 ppb ($p<0.001$). Mean FEV1 improved (1.87 to 1.98 L, +110 mL; $p=0.31$), while median blood eosinophil counts were unchanged (0.14 vs 0.10 $\times 10^9$/L).

Complete clinical remission was achieved in 5/41 patients (12.2%), 7/41 (17.1%) achieved three remission criteria, 15/41 (36.6%) achieved two, and 8/41 (19.5%) achieved one criterion. Overall, 85.4% achieved at least one remission criterion. The most frequently achieved components were no mOCS use (61.0%), stable/improved FEV1 (53.7%), no exacerbations (48.8%), and ACQ-6 <1.5 (29.3%).

Conclusions Tezepelumab was associated with significant clinical improvement and reduced OCS dependence in biologic-naive severe asthma. Complete clinical remission was achieved in 12.2% of patients, while most achieved partial remission, supporting remission as a meaningful treatment outcome.



Abstract P80 Figure 1

P81 **TEZEPELUMAB TO IMPROVE SYMPTOM AND TREATMENT BURDEN IN SEVERE ASTHMA: AN ENGLAND BASED SINGLE-CENTRE RETROSPECTIVE COHORT STUDY**

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10.1136/thorax-2026-BTSabstracts.242

Tezepelumab is a monoclonal antibody (MAB) acting against thymic stomal lymphopoietin (TSLP), showing potential in trials,[1] but with limited real-world data and patient feedback. This study will aim to review whether Tezepelumab can reduce symptom and treatment burden posed by steroid use, alongside good patient tolerability.

This retrospective cohort study looked at 66 patients who between September 2023 and April 2026 completed a minimum of a 12-month trial, were on maximal treatments prior to commencing Tezepelumab and required three-or-more rescue oral steroids (OCS) in the 12 months pre-trial and/or were on regular maintenance steroids.

95% (63) had successful trials, defined as a reduction of 50% or more in their rescue OCS or a reduction / cessation of their maintenance steroid. 42% (28) required 0 rescue OCS during trials.

Whole cohort pre-trial average number of rescue OCS was 4.5(SD 2.2), with average rescue OCS from the 12 months of the trial being 1.0 (SD 1.4). This accounts for a 78% reduction in rescue OCS compared to baseline, with a paired t-test (65) of 11.3 ($p<0.001$).

This was replicated across asthma subtypes

- Atopic asthma patients mean change of rescue OCS from 4.4(SD1.7) to 1.3(SD1) and paired t-test(20) of 7.8 ($p<0.001$).

Abstract P81 Table 1 Cohort Data (total number of patients unless otherwise stated)

Total Cohort		**66**
Age – Mean (SD)		52 (SD 14)
Gender	Male	25
	Female	41
BMI – Mean (SD)		32 (SD 7.6)
Co-Morbidities:	Hay-fever	19
	Eczema	11
	GORD	14
	Diabetes Mellitus	9
	Obstructive Sleep Apnoea	11
	Chronic Rhinosinusitis	5
Smoking Status	Current Smokers	6
	Ex-Smokers	26
	Never Smokers	34
Asthma Subtype	Atopic Asthma	21 (20 successful trials, 1 unsuccessful trial)
	Eosinophilic Asthma	3 (3 successful trials, 0 unsuccessful trials)
	Atopic and Eosinophilic Asthma	30 (29 successful trials, 1 unsuccessful trial)
	No Atopy and normal or low eosinophils	12 (11 successful trials, 1 unsuccessful trial)
No of patients previously trialled on MABs		14
Previously Trialled Respiratory Associated MABs	Mepolizumab	8
	Benralizumab	6
	Omalizumab	4
	Dupilumab	1

- Neither atopic or eosinophilic patients mean change of rescue OCS of 4.3(SD2.7) to 1.3(2.3) and paired t-test(11) of 3.1 (p=0.010).

Of the 13 patients on maintenance steroids, 23%(3) stopped completely, 54%(7) were on steroid replacement for adrenal insufficiency.

Pre-trial Juniper Asthma Control (JAC) average score was 20(SD 8), with a post-trial average score of 12(SD 10.1). Paired t-test(65) was 7.2 (p<0.001). Patient survey data collected frequently commented on improved quality of life (QOL), mainly achieved through reduced symptom impact on their lifestyles.

Overall, this trial shows that Tezepelumab is an effective and well tolerated method to reduce treatment and symptom burden in severe asthma across different subtypes.

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P82 A COMPREHENSIVE ASSESSMENT OF THE IMPACT OF ANTI-IL5 THERAPY ON WELLBEING, ILLNESS PERCEPTION AND COGNITION IN SEVERE ASTHMA: LONGITUDINAL DATA FROM THE CONTENTED STUDY

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Introduction and Objectives Asthma biologics have transformed outcomes for patients with T2-high severe asthma, including significant reductions in exacerbations and oral corticosteroids (OCS). However, less is known about the broader impacts on patient wellbeing, illness perception and cognition. We aimed to examine longitudinal changes across a comprehensive set of 12 validated measures in patients following initiation of mepolizumab (100mg SC) anti-IL-5 therapy as per NICE guidance.

Methods 100 people with T2-high severe asthma completed 12 validated questionnaires prior to starting mepolizumab, and again after 6 months. 50 controls with mild/moderate well-controlled asthma provided comparator baseline data. Longitudinal outcomes were compared using paired t-tests. Mepolizumab responders were defined as having a ≥50% reduction in annualised OCS-requiring exacerbation rate, or maintenance OCS, at the follow-up visit compared to baseline. Differences in outcomes between clinical responders and non-responders were compared using analysis of covariance (ANCOVA), adjusting for baseline score.

Results At baseline, participants with severe asthma had significantly worse wellbeing (WEMWBS-14 -4.90, p = 0.003), anxiety (GAD7 3.32, p <0.001), depression (PHQ9, 5.38, p <0.001), insomnia (AIS 5.10, p <0.001), breathlessness perception (MDP 4.30, p <0.001), illness perception (BIPQ 22.5, p <0.001), and MoCA (-1.44, p <0.001) when compared to controls.

After 6 months of mepolizumab we observed improvements, approaching or exceeding the minimally clinically important differences, in all measures except for cognition (see table 1). Greater improvements were observed in those with a clinical response (n=74) to mepolizumab in most domains (see table 1). Improvements in anxiety (GAD7 -0.63, p = 0.532), wellbeing (WEMWBS 1.87, p = 0.360) and insomnia (AIS -0.75, p = 0.554) did not differ by clinical responder status.

Abstract P82 Table 1 Longitudinal differences in wellbeing patient-reported outcome measures after 6 months of mepolizumab therapy. Difference in mean change was analysed using paired samples t-tests. The difference in the change between mepolizumab responders and non-responders was compared using ANCOVA adjusting for baseline score

Patient report outcome measure	Baseline outcome, Mean (SD)	Mean change (bootstrapped 95%CI)	p-value	Adjusted mean difference in change between responders (n=74) and non-responders (n=19), adjusted mean (95%CI)	p-value
Asthma control, ACQ-7	2.64 (1.04)	-1.25 (-1.48, -1.01)	<0.001	-0.89 (-1.37, -0.42)	<0.001
Asthma-related quality of life, mAQLQ	3.74 (1.31)	1.58 (1.28, 1.88)	<0.001	0.90 (0.25, 1.55)	0.007
Health status, SGRQ	55.7 (18.1)	-20.8 (-24.8, -17.2)	<0.001	-17.5 (-26.7, -8.25)	<0.001
Anxiety, GAD-7	8.11 (6.55)	-3.31 (-4.32, -2.44)	<0.001	-0.63 (-2.63, 1.37)	0.532
Depression, PHQ-9	9.85 (6.98)	-3.76 (-4.96, -2.75)	<0.001	-3.09 (-5.56, -0.63)	0.015
Wellbeing, WEMWBS	44.7 (12.3)	4.77 (3.30, 6.43)	<0.001	1.87 (-2.17, 5.91)	0.360
Insomnia, AIS	11.3 (5.40)	-2.90 (-3.95, -1.81)	<0.001	-0.75 (-3.25, 1.76)	0.554
Breathing discomfort, MDP	5.39 (2.40)	-2.76 (-3.39, -2.11)	<0.001	-2.11 (-3.39, -0.83)	0.002
Dyspnoea sensory quality, MDP	4.51 (2.53)	-2.62 (-3.17, -2.04)	<0.001	-1.69 (-2.73, -0.64)	0.002
Emotional response to dyspnoea, MDP	4.65 (2.81)	-2.07 (-2.62, -1.50)	<0.001	-1.34 (-2.61, -0.07)	0.038
Illness perception, BIPQ	46.4 (11.3)	-12.7 (-15.7, -9.94)	<0.001	-12.2 (-18.7, -5.70)	<0.001
Cognition, MoCA	26.1 (3.23)	0.28 (-0.27, 0.85)	0.316	0.87 (-0.29, 2.03)	0.138

Follow-up data were available for 93 participants, missing data were excluded from the analyses. *WEMWBS-14, Warwick-Edinburgh Mental Wellbeing Scale; GAD7, Generalised Anxiety Disorder Assessment 7; PHQ9, Patient Health Questionnaire 9; SGRQ, St George's Respiratory Questionnaire; AIS, Athens Insomnia Scale; MDP, Multidimensional Dyspnoea Profile; BIPQ, Brief Illness Perception Questionnaire; ACQ7, Asthma Control Questionnaire 7; mAQLQ, mini-Asthma Quality of Life Questionnaire; MoCA, Montreal Cognitive Assessment*

Conclusions People with T2-high severe asthma had significantly worse wellbeing and illness perception compared with mild/moderate well-controlled asthma. Following 6 months of mepolizumab, there were clinically meaningful improvements in wellbeing and illness perception, with some measures improving regardless of the exacerbation-defined clinical response. These findings highlight the broader impact of IL-5 inhibition beyond exacerbation reduction and the value of considering patient-centred wellbeing outcomes when evaluating treatment response.

P83 DUPILUMAB IMPROVES SPIROMETRY- AND OSCILLOMETRY-DERIVED RATIOS INDICATING LARGE AND SMALL AIRWAY FUNCTION IN PATIENTS WITH MODERATE-TO-SEVERE TYPE 2 ASTHMA: VESTIGE

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Introduction Spirometry- and oscillometry-derived ratios can be used to assess airflow in large and small airways, guide treatment decisions, and indicate biologic response in asthma. Dupilumab, improves lung function in patients with moderate-to-severe type 2 asthma. We investigated dupilumab effects on airway function and persistent airflow obstruction using spirometry- and oscillometry-derived.

Methods VESTIGE (NCT04400318) was a phase 4, randomized, placebo-controlled study in adults with uncontrolled, moderate-to-severe, type 2-high asthma (baseline blood eosinophils ≥300 cells/μL and fractional exhaled nitric oxide ≥25 ppb) who received dupilumab 300 mg or placebo every 2 weeks for 24 weeks. At Week 24, we assessed least squares mean (LSM) change from baseline in the pre-bronchodilator forced expiratory volume in 1 second (FEV_1)/forced vital capacity (FVC) ratio indicating large airway function and the forced mid-expiratory flow at 25–75% FVC ($FEF_{25–75\%}$)/FVC and resistance in small airways (R5–R20)/total airway resistance (R5) ratios as indicators of small airway function. We also assessed the proportion of patients with persistent airflow obstruction (pre-bronchodilator FEV_1/FVC z score <-1.64) at baseline who improved this obstruction (FEV_1/FVC z score ≥-1.64) at Weeks 4 and 24.

Results This analysis included 109 patients (dupilumab, n=72; placebo, n=37). At Week 24, dupilumab vs placebo significantly improved the pre-bronchodilator FEV_1/FVC ratio for an LSM difference of 6.8% (95% CI 3.4, 10.2; $P<0.001$). Dupilumab vs placebo also significantly improved $FEF_{25-75\%}$/FVC and R5–R20/R5 ratios at Week 24, with LSM differences of 12.2% (95% CI 5.2, 19.1; $P<0.001$) and −8.1% (95% CI −13.7, −2.5; $P<0.01$), respectively. Of 85 patients with persistent airflow obstruction at baseline, a greater proportion receiving dupilumab vs placebo improved obstruction at Week 4 (27.3% vs 14.3%; $P>0.05$), and a significantly greater proportion improved obstruction at Week 24 (35.7% vs 11.5%; $P<0.05$).

Conclusions In patients with moderate-to-severe asthma and type 2 inflammation, dupilumab significantly improved spirometry- and oscillometry-derived measures of large and small airway function and was associated with a greater proportion of patients improving persistent airflow obstruction.

Clinical Implications Assessment of spirometry- and oscillometry-derived airway ratios may help clinicians assess and monitor meaningful airway-level responses to dupilumab in adults with moderate-to-severe type 2 asthma.

P84 TWICE-YEARLY DEPEMOKIMAB MAINTAINS EXACERBATION REDUCTION AND CLINICAL REMISSION OVER 2 YEARS IN PATIENTS WITH ASTHMA, REGARDLESS OF BASELINE ASTHMA DURATION: SWIFT/AGILE INTEGRATED ANALYSIS

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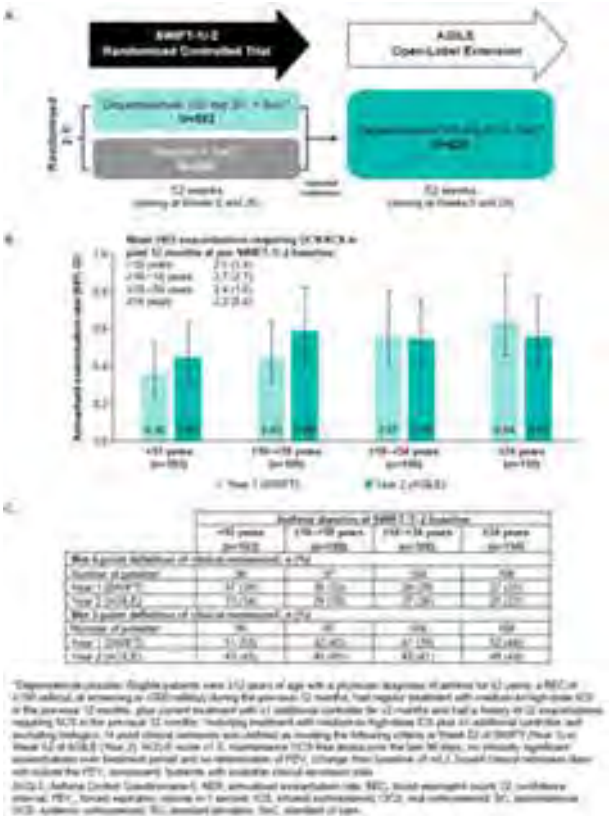
Background Depemokimab is the first ultra-long-acting biologic, with enhanced IL-5 binding affinity, high potency and extended half-life, enabling twice-yearly dosing. In the Phase III SWIFT-1/-2 studies, patients with type 2 (T2) asthma and shorter disease duration showed enhanced depemokimab response versus placebo over 1 year.

Aims Assess depemokimab's efficacy over 2 years in the SWIFT-1/-2 and AGILE studies in patients with T2 asthma, split by asthma duration.

Methods In SWIFT1/2, patients on medium/highdose ICS were randomized 2:1 to depemokimab (N=502) 100 mg or placebo (N=206) every 26 weeks for up to 52 weeks. Completing patients entered AGILE OLE, continuing or switching to depemokimab (N=629) for up to 2 years.This post hoc analysis focused on patients receiving depemokimab for 2 years. Outcomes included annualised exacerbation rate (AER) and clinical remission (4- and 3-point definition; see figure footnote) split by baseline asthma duration (<10, ≥10–<18, ≥18–<34, ≥34 years).

Results In total, 419 patients received 2 depemokimab doses in SWIFT-1/-2 and ≥1 dose in AGILE over a 2-year period. Patients maintained AER with depemokimab over 2 years regardless of asthma duration, with trends of lower exacerbation rates observed in patients with baseline asthma duration <10 years compared with those with longer asthma duration (figure). Clinical remission rates (4point) were higher in patients with shorter asthma duration at Year 1 (39% for <10 years vs 25% for ≥34 years) and remained numerically higher at Year 2 (34% vs 23%); similar trends were observed using the 3-point definition (Year 1: 53% vs 48%; Year 2: 45% vs 44%).

Conclusions Twice-yearly depemokimab maintained efficacy over 2 years across asthma duration subgroups.



Abstract P84 Figure 1 SWIFT-1/-2 and AGILE study flow are shown in (A). In patients who received depemokimab for 2 years, patients maintained AER regardless of asthma duration (B), with a trend of lower AER and higher rates of clinical remission in patients with shorter asthma duration at baseline, compared with those with longer asthma duration (C)

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P85 NEVER TOO OLD FOR BIOLOGICS? REAL-WORLD OUTCOMES IN SEVERE ASTHMA PATIENTS AGED 75 YEARS AND OLDER

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10.1136/thorax-2026-BTSabstracts.246

Older adults with severe asthma are under-represented in clinical trials of biologic therapies. Consequently, evidence regarding the effectiveness and safety of biologics in patients aged 75 years and older remains limited.

Method We conducted a retrospective observational study of patients aged 75 years and over who were initiated on Benralizumab or Tezepelumab within our severe asthma service.

Data collected included, age of starting biologic, FeV1, exacerbation frequency, maintenance oral corticosteroid use, and co-existing Chronic Obstructive Pulmonary Disease.

Clinical outcomes following biologic initiation included annualized exacerbation rate, long term prednisolone use, Asthma Control Questionnaire (ACQ), improvement in FeV1 and current steroid inhaled treatment.

Results A total of 43 patients completed a year's trial; 35 Benralizumab, and 8 Tezepelumab. The median age at biologic initiation was 77 years (75-88). Of these, 36 patients were biologic naive; 7 had been switched; from Omalizumab (1), Mepolizumab (2), Benralizumab (4). All switches were for biologic failure.

Following treatment initiation, median annual exacerbations decreased from 4 to 0 (11-3). Of the 11 patients on baseline maintenance prednisolone (median dose: 10mg OD), 9 weaned off completely, 2 needed long-term steroids for adrenal suppression. Of those who required IM steroid injections, 1 required a dose of steroids in the year of post commencement.

Mean FEV1 improved by 0.182L (0.97 to-0.34), 9 patients' lung function reverted to normal FeV1>80% 6 (Benralizumab), 3 (Tezepelumab). ACQ improved by 1.7(5.6 to-0.7). In those admitted to hospital 9 out of 13 had no admissions in the year after commencing biologics.

One patient failed their biologic trial; there were no adverse events in this population.

Conclusion Thisdataindicatesthat biologics are successfulwithinanolder adult population as per NICE criteria.With discontinuation oflong-termprednisolone beingpossible. Theygainsymptomatic benefit with aclinically meaningful improvement in ACQ(>0.5)and FeV1.

As this was carried out within a small cohort, further research is needed to ascertain if this is a true effect. More research is needed to explore the benefits of biologics in older adults, such as improvement in all causes of admissions and clinical frailty.

Respiratory infections: from disease burden to outcomes

P86 PUBLIC AWARENESS AND UNDERSTANDING OF HOSPITAL-ACQUIRED PNEUMONIA: A CROSS-SECTIONAL SURVEY

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Introduction Hospital-acquired pneumonia (HAP) develops at least 48 hours after admission or within 7-10 days of discharge and is associated with mortality, morbidity, antibiotic exposure and antimicrobial resistance. Prevention depends partly on patient, visitor and staff behaviours, yet UK public awareness is not well described. We assessed awareness of HAP, understanding of differences from CAP, knowledge of risk, prevention, treatment, and support for research.

Methods An anonymous questionnaire was developed using published guidance on survey design. The survey was drafted iteratively, piloted with nine participants of varying ages and educational backgrounds, revised, and re-shared. Feedback and resulting revisions were captured in a formal activity tracker and change log, including changes to improve clarity, reduce free text, and standardise response options. The final survey was distributed online and via QR-code poster. Responses were analysed descriptively; healthcare workers, including clinical

Abstract P86 Table 1 Key survey findings to accompany the abstract

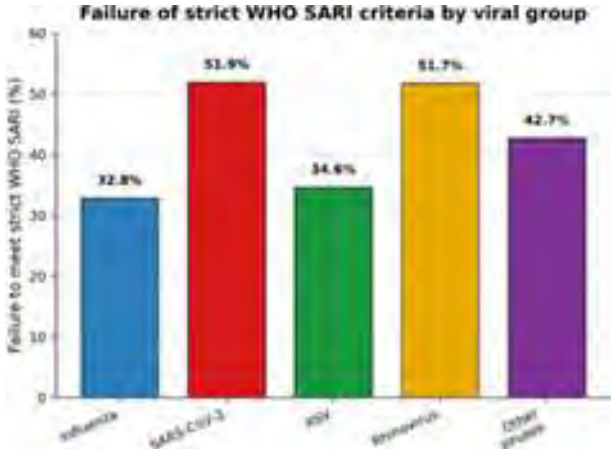
Domain	Survey finding	Overall n/N (%)	By respondent type n/N (%)
Awareness	Previously heard of HAP	142/270 (52.6)	HCW: 21/25 (84.0); non-HCW: 122/245 (49.8)
Understanding	Knew a HAP-CAP distinction beyond location of acquisition	35/270 (13.0)	HCW: 10/25 (40.0); non-HCW: 25/245 (10.2)
Risk recognition	Identified older/severely ill patients as higher risk	244/270 (90.4)	HCW: 25/25 (100); non-HCW: 219/245 (89.4)
Treatment/prevention	Incorrectly stated antibiotics can always prevent HAP	45/270 (16.7)	HCW: 0/25 (0); non-HCW: 45/245 (18.3)
Prevention knowledge gap	Unsure whether toothbrushing/oral care reduces risk	155/270 (57.4)	HCW: 11/25 (44.0); non-HCW: 144/245 (58.8)
Research support	Supported prioritising HAP diagnosis/treatment research	246/270 (91.1)	Not reported by subgroup

HCW = healthcare worker; CAP = community-acquired pneumonia; HAP = hospital-acquired pneumonia.

and non-clinical healthcare-associated roles, were compared with other respondents.

Results There were 270 responses; mean age was 50.4 years (range 18–90), 201 (74.4%) were female and 257 (95.2%) were UK residents. The survey achieved broad UK geographical spread, with responses from 47 counties; the largest county groups were Merseyside (n=43), Nottinghamshire (n=25) and Lincolnshire (n=25). Table 1 shows that, overall, 142/270 (52.6%) had previously heard of HAP, but only 35/270 (13.0%) knew any distinction between HAP and CAP beyond place of acquisition. Awareness was higher in healthcare workers than other respondents (21/25, 84.0% vs 122/245, 49.8%). Recognition of high-risk groups was good, but knowledge gaps remained: 155/270 (57.4%) were unsure whether toothbrushing and oral care reduced risk, and 45/245 (18.3%) non-healthcare respondents thought antibiotics could always prevent HAP. After brief information, 246/270 (91.1%) supported prioritising research into HAP diagnosis and treatment.

Conclusion In this geographically broad UK survey, about half of respondents had heard of HAP, but detailed understanding was limited. Education should explain how HAP differs from CAP, why oral care and mobilisation matter, and why antibiotics are not a guaranteed solution. Brief, accessible information may help patients engage with prevention and generate strong support for HAP research.



Abstract P87 Figure 1 Failure to meet strict WHO severe acute respiratory infection criteria by viral group. Bars show the proportion of adults with polymerase chain reaction-confirmed single-virus respiratory viral infection who did not meet strict WHO severe acute respiratory infection criteria. Strict criteria required cough and fever-related symptoms, with both symptom durations ≤10 days before admission. Fever- related symptoms comprised chills/shivering, feeling cold, feeling hot or sweating; cough comprised dry, wet/productive or other cough. Failure was most frequent in SARS-CoV-2 and rhinovirus/enterovirus admissions and least frequent in influenza and respiratory syncytial virus admissions

P87 FEVER-DEPENDENT WHO SARI CRITERIA INCOMPLETELY ASCERTAIN ADULT HOSPITALISED RESPIRATORY VIRAL INFECTION

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10.1136/thorax-2026-BTSabstracts.248

Introduction and Objectives The World Health Organization severe acute respiratory infection (WHO SARI) definition enables standardised hospital respiratory-virus surveillance, but encodes a febrile, recent-onset cough syndrome and may not reflect the breadth of adult acute respiratory illness (ARI) requiring admission. We evaluated pathogen-specific WHO SARI capture among adults hospitalised with clinician-confirmed ARI and polymerase chain reaction (PCR)-confirmed respiratory viral infection (RVI).

Methods Adults in a prospective multicentre UK cohort were eligible if aged ≥18 years, admitted as medical inpatients within 36 hours, and had clinician-confirmed ARI. ARI was defined as acute upper or lower respiratory tract illness, including rhinitis, rhinosinusitis, pharyngitis, pneumonia, bronchitis or influenza-like illness, or acute exacerbation of chronic lung disease, diagnosed by the treating clinician. We analysed PCR-confirmed single-virus RVI: influenza (n=260), SARS-CoV-2 (n=252), rhinovirus/enterovirus (n=221), respiratory syncytial virus (RSV; n=115) and other respiratory viruses (n=122). Strict WHO SARI required cough and fever-related symptoms, both with maximum duration ≤10 days before admission. Loose WHO SARI required cough and fever-related symptoms, with at least one duration ≤10 days. Penalised logistic regression examined strict WHO SARI failure.

Results Lower-respiratory symptoms were present in 955/961 adults (98.5%), but strict WHO SARI captured only 514/909 (56.5%), leaving 395/909 (43.5%) uncaptured. Capture was highest for influenza (67.2%) and RSV (65.4%), intermediate for other viruses (57.3%), and lowest for SARS-CoV-2 (48.1%) and rhinovirus/enterovirus (48.3%). Loose WHO SARI captured 634/920 (68.9%), but still left 286/920 (31.1%) uncaptured. Among strict WHO SARI failures, absence of fever-related symptoms contributed to 43.5%–55.0%, cough duration >10 days to 51.6%–68.3%, fever-related symptom duration >10 days to 31.8%–47.1%, and absence of cough to only 3.7%–

17.5%. Compared with influenza, rhinovirus/enterovirus and SARS-CoV-2 had the strongest associations with strict WHO SARI failure (OR 2.40 and 2.03).
Conclusions In adults admitted with clinician-confirmed ARI, WHO SARI under-ascertained PCR-confirmed RVI despite near-universal lower-respiratory symptoms. Non-capture was driven principally by fever dependence and the 10-day duration threshold. Hospital viral testing should use broad ARI criteria rather than WHO SARI alone to ascertain adult inpatient respiratory viral burden.

P88 VIRAL AETIOLOGY OF ARDS IN NEUTROPENIC PATIENTS WITH MALIGNANCY: A RETROSPECTIVE COHORT STUDY

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Introduction Acute respiratory distress syndrome (ARDS) carries high mortality in patients with malignancy. Viral respiratory infections are a recognised trigger, yet whether confirmed viral aetiology independently worsens prognosis — beyond overall illness severity — remains unclear. Neutropenia, common in haematological malignancy, may further impair viral defence and modify outcomes. The prognostic value of the Berlin ARDS severity criteria in this immunocompromised population is also uncertain.
Methods Retrospective observational cohort study of 111 adult patients with malignancy admitted to a tertiary ICU with ARDS. The primary exposure was confirmed viral infection on respiratory testing. Neutropenia was defined as absolute neutrophil count $<0.5 \times 10^9$/L at ARDS onset; severity was classified by the Berlin definition. The primary outcome was ICU or hospital mortality. Multivariable logistic regression adjusted for age, neutropenia, severe Berlin ARDS, invasive mechanical ventilation, and organ failure.
Results Overall mortality was 48.6% (54/111). Viral infection was confirmed in 30 patients (27.0%); crude mortality was higher in the viral group (60.0% vs 44.4%, $p = 0.199$). After multivariable adjustment, viral infection was not independently associated with mortality (aOR 1.02, 95% CI 0.38–2.74; $p = 0.965$). This null finding was consistent across all sensitivity analyses, including a pathogenic respiratory virus-restricted definition (aOR 1.58, 95% CI 0.52–4.76), and was supported by Bayesian estimation (posterior probability of harm 52%). Organ failure was the strongest predictor of mortality (aOR 3.87, 95% CI 1.56–9.61; $p = 0.004$). Neutropenia showed a clinically important but statistically uncertain association with mortality (aOR 2.21, 95% CI 0.91–5.36; $p = 0.080$). Berlin oxygenation categories demonstrated poor mortality discrimination (AUC 0.53–0.54), substantially outperformed by a composite model incorporating organ failure and ventilator support (AUC 0.747).
Conclusions In malignancy-associated ARDS, confirmed viral infection was not independently associated with mortality. Prognosis was driven primarily by organ failure and host vulnerability, not by viral aetiology or Berlin oxygenation severity. Neutropenia emerged as a clinically meaningful prognostic factor. These findings argue against relying on viral PCR result or oxygenation category alone when making prognostic or triage decisions in this population.

P89 HEALTHCARE UTILISATION AFTER HOSPITALISATION FOR INFLUENZA IN UK: A POPULATION-BASED STUDY.

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10.1136/thorax-2026-BTSabstracts.250

Introduction and Objectives Prolonged recovery was highlighted in people recovering from COVID-19. What is not clear is the recovery and morbidity after other respiratory viruses and the impact on primary and secondary healthcare usage. This study aimed to identify the rate and reason for primary care consultation, hospital readmission and death after hospitalisation with influenza in UK using Clinical Practice Research Datalink (CPRD) linked with Hospital Episode Statistics (HES).
Methods Adults with a first episode of hospitalised influenza as the primary cause for admission between 01/07/2004 - 01/03/2021 based on ICD-10 codes were included. Patients were followed from one day after discharge till either acute primary care consultation, readmission, death or study end. Logistic regression models estimated adjusted odds ratios for respiratoryrelated consultations and readmissions.
Results Among 16,929 patients, 6,865 (40.6%) had 13,039 primary care consultations within 30 days of hospital discharge. Median time to first consultation was 7 days (IQR 4-14). Notably, 26% of patients that consulted had not interacted with primary care in the prior year before hospital admission. Risk factors for respiratory related GP consultations included being female, aged 41-60 years old, preexisting asthma or COPD and requiring critical care during hospital admission.

Within 90 days of hospital discharge, 3,230 (19.1%) people had 4,940 hospital readmissions with a median length of stay of 3 days (IQR 1-8 days). Common readmission reasons included influenza, pneumonia and cardiovascular disease. In the year following discharge, 1,299 (7.7%) patients died. Median time to death was 151 days (IQR 61-260 days) for all-cause mortality and 133 days (IQR 47-256 days) for a respiratory cause of death. Respiratory disorders represented a significant proportion of primary and secondary care encounters often resulting in earlier readmissions and earlier death compared to other diagnoses.
Conclusions Following hospitalisation for influenza a significant number of patients consulted primary care within 30 days and were readmitted to hospital within 90 days. This highlights the ongoing morbidity during recovery from influenza.

P90 IMPLEMENTATION OF DIGITAL TOOLS TO IMPROVE LENGTH OF STAY AND ANTIMICROBIAL STEWARDSHIP IN COMMUNITY-ACQUIRED PNEUMONIA

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10.1136/thorax-2026-BTSabstracts.251

Background Community-acquired pneumonia (CAP) places significant burden at individual and institutional level. At our centre, approximately 1000 adults are admitted annually, with mean length of stay previously exceeding 10 days. Previous audits repeatedly showed poor compliance with risk stratification, resulting in overuse of co-amoxiclav and intravenous ther-

apy in low severity pneumonias and excessive total treatment durations. A pneumonia optimisation pathway was instituted in October 2023 aiming to align institutional priorities (patient flow, length of stay) with antimicrobial stewardship (AMS). Here we describe three digital tools developed to monitor and improve AMS in CAP.

Methods As part of a pneumonia optimisation work, a multidisciplinary team developed, implemented and provided education support for three digital tools: i) a clinical decision support (CDS) tool to facilitate calculation and improve prescribing based on C(U)RB-65 score risk stratification implemented in the electronic health records (Epic); ii) a best practice advisory (BPA) to promote early antibiotic intravenous to oral switch (IVOS) flagging patients in Epic when reaching predefined stability criteria; iii) an interactive dashboard (implemented in QlikView) enabling large-scale near-real-time auditing of AMS parameters using an automated data pipeline, including antibiotic adherence to guidelines, time to IVOS, total length of treatment, uptake of the CDS and BPA, and used for feedback.

Results The CDS launched in January 2024 was associated with significant improvement in overall adherence to guidelines. Average length of stay was reduced by 2.5 days, resulting in significant bed day savings. The dashboard was iteratively developed and validated, including optimising parameters for cohort definition, checkpoint selection (4, 12 and 24h post admission) for dynamic assessment of antibiotic compliance, and compliance rules. Feedback of hospital-wide performance started in summer 2025 with plans to initiate ward-level feedback.

Conclusions Dashboards fully automate the auditing process and enable near-real-time assessment of the impact of quality improvement interventions, including CDS tools. Institutional buy-in and multidisciplinary collaboration were essential elements in this process. The CDS improved length of stay and guideline based antibiotic prescribing.

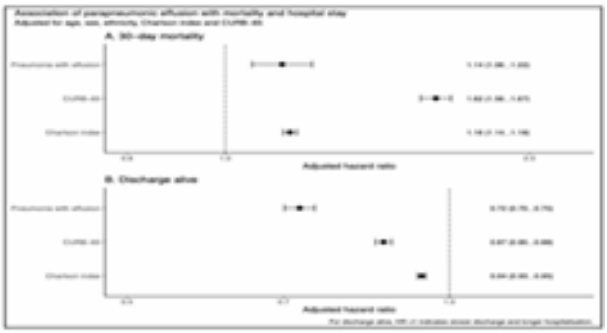
P91 NOT-SO- 'SIMPLE': PARAPNEUMONIC EFFUSION AS AN INDEPENDENT MARKER OF EXCESS MORTALITY AND PROLONGED HOSPITALISATION IN PNEUMONIA

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Background Parapneumonic effusion (PPE) can complicate up to 40% of cases of pneumonia, yet its independent prognostic significance remains poorly characterised, and pleural effusion is not incorporated into standard pneumonia severity scoring (CURB-65). We evaluated the association between PPE and clinical outcomes in a large, hospitalised pneumonia cohort.

Methods We conducted a retrospective analysis of routinely collected data from the Infections in Oxfordshire Database (IORD) on adult pneumonia admissions in Oxfordshire over a 7-year period (2016-2023). PPE was identified using diagnostic coding and a natural language processing algorithm applied to radiology reports. The primary outcome was 30-day mortality; the secondary outcome was time to discharge alive. Cox proportional hazards models were adjusted for age, sex, ethnicity, Charlson Comorbidity Index and CURB-65 score, with robust standard errors clustered by patient identifier. Unadjusted rates are reported for the full cohort; adjusted analyses were conducted in the 21,436 admissions with complete covariate data.



Abstract P91 Figure 1 Forest plots showing adjusted hazard ratios for 30-day mortality (Panel A) and time todischarge alive (panel B) in 21,436 adult pneumonia admissions. All models adjusted for age, sex, ethnicity, Charlson comorbidity index and CURB-65. Parapneumonic effusion was independently associated with both increased mortality (HR 1.14, 95% CI 1.06-1.22) and a 28% reduction in rate of discharge alive (HR 0.72, 95% CI 0.70-0.75)

Results Among 35,807 pneumonia admissions, 9,250 (26%) had PPE. Unadjusted 30-day mortality was higher among patients with PPE than those without (20.3% vs 15.6%). On multivariable analysis, PPE remained independently associated with higher 30-day mortality (HR 1.14, 95% CI 1.06–1.22; $p<0.001$).

Median length of stay was longer in those with PPE (9.7 days [IQR 4.4–19.2] vs 5.8 days [IQR 2.3–13.4]). On multivariable analysis, PPE was independently associated with a 28% slower rate of discharge alive (HR 0.72, 95% CI 0.70–0.75; $p<0.001$), demonstrating a clinically important effect that persisted after adjustment for established severity markers (figure 1).

Conclusions This is the largest study to assess the association of PPE with outcomes. In this real-world cohort, PPE, present in one in four pneumonia admissions, was independently associated with excess mortality and longer hospitalisation, effects not fully explained by age, sex, ethnicity, comorbidity or CURB-65 severity. These findings support recognition of PPE as a distinct, higher-risk pneumonia phenotype and provide the evidence base for prospective evaluation of pleural effusion within pneumonia risk stratification frameworks to improve prognostication and inform clinical decision-making.

P92 IMPACT OF STATINS ON OUTCOMES OF OLDER INPATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA

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Statins are commonly used lipid-lowering drugs that also have immunomodulatory effects. People above the age of 65 have higher mortality with pneumonia, one possible reason being age-related impairments in neutrophil function. Previous studies suggest statins can reverse these impairments in vitro, but it

Abstract P92 Table 1 Baseline characteristics and outcomes of patients aged >65 years with CAP, stratified by inpatient statin use. Values are presented as mean ± SD, median (IQR), or n (%), as appropriate

Characteristics	Statins (n=207)	No statins (n=595)	P
Age (y)	76.1 ± 6.6	77.6 ± 7.6	0.021
Gender – F (%)	81 (39.1%)	283 (47.6%)	0.044
Charlson comorbidity index	6 (5–8)	6 (4–8)	0.221
Admission white blood cell count (1000/μL)	8.9 (6.6–11.7)	9.2 (6.1–12.8)	0.631
Admission neutrophil/ lymphocyte ratio	5.0 (3.2–14.3)	5.7 (2.1–12.4)	0.764
Admission C-reactive protein (mg/L)	114.5 ± 110.4	103.3 ± 49	0.84
Admission urea (mg/dL)	20.7 (15–28.5)	19.0 (13.6–28.9)	0.15
Outcomes			
Primary: 30d mortality (%)	11 (5.31%)	46 (7.73%)	0.313
Secondary: in-hospital mortality (%)	2 (0.97%)	28 (4.71%)	0.026
Secondary: length of stay (day)	4.2 (2.7–7.1)	3.9 (2.7–6.1)	0.095
Secondary: ICU admission (%)	41 (19.8%)	134 (22.5%)	0.474

is uncertain whether statins improve mortality in older patients with pneumonia. We sought to identify the impact of statin administration on clinical outcomes of patients over the age of 65 hospitalised for community-acquired pneumonia (CAP).

Methods A retrospective cohort study using the MIMIC-IV dataset was performed.[1] Subjects over 65 were identified, and those with 'pneumonia' coded as the admission diagnosis who had a sputum sample submitted within 48 hours of admission were considered to have CAP. Statin status was based on the inpatient drug chart. Data processing was performed in R. Chi-square tests were used for comparisons of categorical data and Mann–Whitney U tests for non-parametric data. The primary outcome was 30-day mortality. Secondary outcomes were in-hospital mortality, length of stay and ICU admissions.

Results 802 patients over 65 with CAP were identified. Of these, 207 patients were on statins during their inpatient stay. Patients not on statins were older (77.6 vs 76.1 years) with a higher proportion of female patients (47.6% vs 39.1%) (table). There was no significant difference in 30-day mortality, length of stay and rate of ICU admission between the groups. However, the statin group had significantly lower in-hospital mortality compared to the non-statin group (0.97% vs 4.71%, p = 0.026).

Conclusion This study suggests that among patients over 65 with CAP, statin use was associated with significantly lower in-hospital mortality, despite similar baseline characteristics and no difference in 30-day mortality and ICU admission. These findings show a potential protective effect of statins in older patients with CAP, warranting investigation in prospective studies.

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P93 VALUE OF PROCALCITONIN IN IDENTIFYING ALTERNATIVE DIAGNOSES IN PATIENTS WITH SUSPECTED COMMUNITY-ACQUIRED PNEUMONIA

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10.1136/thorax-2026-BTSabstracts.254

Introduction and Objectives Community-acquired pneumonia (CAP) is a common cause of acute respiratory admission. Procalcitonin (PCT) may help distinguish bacterial infection from alternative causes of respiratory symptoms and support diagnostic assessment in suspected CAP.[1 2] This audit evaluated alternative diagnoses in patients with suspected CAP and a normal PCT level and their impact on clinical management.

Methods A retrospective audit was conducted of patients admitted with suspected CAP in 2023 who underwent PCT testing because of diagnostic uncertainty or suspicion of an alternative diagnosis. A total of 292 patients presenting with signs and symptoms consistent with pneumonia were screened. Of these, 45 adults with a CRP ≥40 mg/L and a normal PCT level (<0.25 ng/mL) met the inclusion criteria. Clinical, radiological and laboratory data were collected, including presenting symptoms, chest radiograph findings, inflammatory markers and microbiological results. Final diagnoses were established following specialist respiratory review. The primary outcome was the proportion of patients ultimately diagnosed with a condition other than CAP.

Results Forty-five patients were included. All had normal PCT levels despite elevated CRP levels ranging from 40–323 mg/L. Following further investigation, 13 patients (29%) were reclassified as having alternative diagnoses. These included interstitial lung disease exacerbation (n=4), heart failure (n=2), asthma exacerbation (n=2), lung adenocarcinoma (n=2), pulmonary embolism (n=1), tuberculosis (n=1) and COVID-19 pneumonitis (n=1). Identification of alternative pathology resulted in important changes to management, including anticoagulation, diuretic therapy, corticosteroid treatment, anti-tuberculous therapy, oncological referral, and reduction or discontinuation of unnecessary antibiotics.

Conclusions Among patients admitted with suspected CAP and a normal PCT level, 13 of 45 (29%) were subsequently found to have an alternative diagnosis following specialist review. These findings suggest that a normal PCT may prompt investigation for non-bacterial causes of respiratory symptoms and support targeted management while reducing unnecessary antibiotic use.

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P94 DIAGNOSTIC ACCURACY OF AN ARTIFICIAL INTELLIGENCE SYSTEM FOR IDENTIFYING PULMONARY TUBERCULOSIS ON CHEST RADIOGRAPHS IN A MODERATE INCIDENCE GLOBAL NORTH SETTING

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10.1136/thorax-2026-BTSabstracts.255

Introduction Accurate identification of tuberculosis (TB) on chest x-rays (CXR) remains a diagnostic challenge. The World Health Organisation (WHO) has recommended artificial intelligence (AI)-based computer-aided detection to support CXR interpretation and TB screening.
Objective To evaluate the diagnostic performance of qXR (Qure.ai) for pulmonary TB detection on CXRs within a large NHS trust covering areas with TB incidence ranging from 18.8-41.4/100,000. To date, qXR performance for TB detection has not been assessed in this setting.
Methods GP-requested CXRs from 1/1/2025-30/6/2025 containing TB in the qXR or radiology report were identified and compared to diagnoses on the national TB surveillance system. Sensitivity, specificity, positive and negative predictive values (PPV, NPV) were calculated.
Results The searches identified 560 CXRs with a 2.9% TB prevalence. qXR had 62.5% (95% CI: 35.4-84.8%) sensitivity and 82.5% (95% CI: 79.1-85.6%) specificity compared to 68.8% (95% CI: 41.3-89%) and 97.8% (95% CI: 96.2-98.9%) for clinicians. Sensitivity was not statistically different ($p=0.1$), whilst qXR had a significantly lower specificity ($p<0.001$). NPV was comparable between qXR (98.7% [95% CI: 97.5-99.3%]) and clinicians (99.1% [95% CI: 98.1-99.6%]). However, qXR had a considerably lower PPV (9.5% [95% CI: 6.5-13.8%]) than clinicians (47.8% [95% CI: 32.4-63.7%]). No significant difference was detected between clinician subgroups (Consultant/Registrar/Reporting Radiographer). qXR had a moderate AUROC of 0.58 (95% CI: 0.54-0.61, $p<0.0001$).
Conclusions Our findings show qXR has a high sensitivity and NPV that are comparable to radiology clinicians, with lower specificity and PPV. In a low incidence setting, AI may have a role in prioritising CXRs for urgent reporting to reduce diagnostic delay; in a higher incidence setting, there may be a risk of overdiagnosis, which should be offset against workforce constraints.

P95 MAC-PD, THE COST OF CARE: A COMPREHENSIVE ASSESSMENT OF ACTUAL HEALTHCARE COSTS ASSOCIATED WITH MYCOBACTERIUM AVIUM COMPLEX (MAC) PULMONARY DISEASE (PD)

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Introduction Non-tuberculous Mycobacteria Pulmonary Disease (NTM-PD), predominantly linked to MAC species, is associated with significant healthcare resource utilisation. With the increasing prevalence of NTM-PD, the cost of care to the NHS remains unknown.
Methods We sought to assess total healthcare costs of MAC-PD through a retrospective cohort study of all patients with positive respiratory NTM isolates in NHS Fife, Scotland, across a 6-year period, 2017-2022. Patient pathways were assessed using electronic patient records and categorised into 'incidental', 'suspected' and 'confirmed/treated' MAC-PD pathways. Patients with confirmed/treatment pathways were analysed for all related healthcare costs over a 5-year period (1 year pre-, 4 years post-treatment). Healthcare unit costs derived from national reference costs were inflation-adjusted to today's costs. Relevant local approvals were obtained.
Results 334 NTM isolates included 211 MAC species-isolates from 81 unique patients. Clinical pathway review identified 44 (54%) as incidental, 12 (15%) suspected and 25 (31%) confirmed/treated for MAC-PD. The treated cohort had a median age of 69 (range 45–93), 10 female/15 male, median treatment 18 months (IQR: 8.8–26.2; range 1.1–61.6), and chronic lung disease in 22/25 patients (19 with COPD (16) and/or bronchiectasis (15)).

Total per-patient healthcare cost of care for treated MAC-PD was £27,600. This was significantly influenced by high-cost therapy with Arikayce, with the median total cost £7,500 (£2,400–£289,000). Category costs were dominated by specialist staffing (mean £3,300, range £600–£7,900) and drug treatments/hospital admissions (£2,200; £380–291,400); other major category-costs included diagnostics including microbiology/imaging (£500; £80–£1,900), support services/adjunctive therapies (£55; 0-£630). Per-patient drug-therapy costs were £80–£230,800 (mean £10,800, median £1,400); 1st line therapy was £31,300; 2nd/3rd line therapies including amikacin IV/nebulised represented very high-cost therapy (£217,000, >85% costs). All values include treatment durations shortened by death/other interruption.
Conclusions We present the first highly characterised cost assessment cohort for MAC-PD in the UK. These data can inform local, regional and national service provision, are anticipated to significantly increase with novel high-cost drug therapies and present a significant and unpredictable cost-burden on individual Health Boards. We recommend investment into enhanced respiratory specialist nursing roles, regional integration of services and a regional/national framework for novel high-cost therapies.

P96 UNDERSTANDING PATIENT AND HEALTH SYSTEM DELAYS IN TUBERCULOSIS DIAGNOSIS

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10.1136/thorax-2026-BTSabstracts.257

Background Timely diagnosis and treatment of tuberculosis (TB) are essential for improving outcomes in the UK. This study aimed to quantify patient and health system delays and identify factors associated with diagnostic delay.
Method We conducted a retrospective analysis of all consecutive patients diagnosed with TB at our institution between September 2021 and September 2025. We measured time from symptom onset to first healthcare presentation (patient delay) and from first presentation to diagnosis (health system delay). Delays were stratified by age, sex, migrant status, time in the UK, symptom status, and site of disease. Comparisons were made using the two-sample Wilcoxon rank-sum test in Stata 19 MP.
Results A total of 1,028 patients were included. Median age was 35 years (IQR 26–51); 662 (64.4%) were male and 868 (84.5%) were migrants. Overall, 402 patients (39.1%) had been in the UK for less than 5 years. Median patient delay was 28 days (IQR 7–67), and median health system delay was 49 days (IQR 16–116). There were 383 had just pulmonary TB alone, 132 of patients had EPTB and PTB, and 513 had EPTB alone.

Patient delay did not significantly differ across any of the examined factors. In contrast, health system delay varied

Abstract P96 Table 1

Factor	Health system delay in days Median (IQR)	p-value
Gender		0.001
Male	14.5(42.5-104)	
Female	21 (60-134)	
Age		0.01
16-35	14 (42-109)	
36- 84	20 (58-126)	
Time since entry		0.01
Less than 5 years in UK	44 (14-104)	
UK born or more than 5 years in UK	55 (19-128)	
Symptoms		0.000
Symptomatic	30 (10-85)	
Asymptomatic	117.5 (50- 205)	
Site of disease		0.000
PTB and EPTB	30 (10-85)	
EPTB	73 (32-153)	
Social risk factors		0.03
No social risk factor	18 (53-110)	
At least one social risk factor	11 (36-111)	

significantly by several characteristics. Longer health system delay was observed in females, symptomatic patients, and those with extrapulmonary TB. Importantly, time in the UK was associated with health system delay ($p = 0.0116$), with patients who had been in the UK for less than 5 years experiencing shorter health system delays compared with those being UK born or resident for 5 years or more.

Conclusion Compared with patient delay, health system delay in TB diagnosis varies by patient characteristics, particularly sex, extrapulmonary disease, and duration of residence in the UK, highlighting the need to strengthen diagnostic pathways in high-incidence settings.

P97 QUANTIFYING THE SECONDARY CARE BURDEN AND SOCIAL COMPLEXITY OF TUBERCULOSIS TO SUPPORT COMMUNITY PATHWAY IMPROVEMENT

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10.1136/thorax-2026-BTSabstracts.258

Background Tuberculosis (TB) places substantial pressure on secondary care services in high incidence settings. Patients often present late, have unmet support needs and require prolonged admissions. The full secondary care burden of TB in the UK including inpatient utilisation, extensive diagnostic workup, delayed discharge and escalation to intensive care remains poorly quantified. Better understanding these pressures is essential for service planning and improving TB pathways.

Aim To quantify secondary care utilisation and system pressures associated with TB, and to examine how social complexities contribute to avoidable admissions, delayed discharge and treatment interruption.

Methods A retrospective review of 100 consecutive TB cases (March 2025–January 2026) was conducted. Patients ranged from 14–87 years (median 30; mean 36.1); 67% were male. Pulmonary TB occurred in 73%, with intrathoracic lymph node involvement in 40% and extrathoracic disease in 33%. Admissions directly attributed to TB accounted for 45% of the cohort. Of these, 68% presented via A&E and 10% required an ITU stay (2% of the full cohort), with a median ITU LOS of 8 days. Mean LOS was 20.96 days (median 16). Care bundles are being reviewed against NHS PLICS datasets to assess resource use and cost pressures. Variables included inpatient utilisation, LOS, repeated admissions (40% of TB related admissions), intensive care use, A&E attendances and causes of delayed discharge. LOS was examined for prolonged admissions, repeat utilisation and links to social complexity. Clinical interventions, diagnostic activity and multidisciplinary involvement (therapies, social care, safeguarding, nutrition and TVN) were recorded. Case examples demonstrated how social and clinical complexity prolonged LOS and delayed discharge.

Results Preliminary findings indicate secondary care utilisation was concentrated among socially complex patients, with prolonged admissions, repeated A&E attendances and a high diagnostic burden. Delayed discharges were frequently linked to housing barriers or unmet social care needs.

Conclusion TB generates a significant and often preventable secondary care burden in high incidence areas. Strengthened community based TB pathways, earlier housing and social care involvement and targeted support for socially complex patients are needed to reduce avoidable admissions and improve continuity of care.

P98 TITLE: MISSED OPPORTUNITIES FOR TUBERCULOSIS PREVENTION: AN AUDIT OF LTBI SCREENING AMONG RECENT MIGRANTS SUBSEQUENTLY DIAGNOSED WITH ACTIVE TUBERCULOSIS

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10.1136/thorax-2026-BTSabstracts.259

Background The vast majority of active tuberculosis (TB) cases in the UK occur among migrants from high-incidence countries and result from reactivation of previously acquired latent tuberculosis infection (LTBI), rather than recent transmission. Barking, Havering and Redbridge has one of the highest TB incidence rates in London. UK Health Security Agency (UKHSA) guidance for the national LTBI screening programme recommends LTBI screening in primary care for eligible recent migrants from high TB prevalence countries. We conducted a retrospective study to evaluate missed opportunities for LTBI screening in primary care among migrants who were later diagnosed with active TB at our centre.

Methods A retrospective audit was conducted of patients diagnosed with active TB between 2021 and 2024. Patients who had entered the UK within the preceding five years from countries with a high TB incidence were identified from local TB service records. Data collected included demographic characteristics, site of TB disease, LTBI screening status, microbiological findings and treatment outcomes.

Results Sixty-nine patients met inclusion criteria. Only 8/69 (11.6%) had undergone LTBI screening prior to their diagnosis of active TB, while 61/69 (88.4%) had no documented LTBI screening.

Among the 61 unscreened patients, the commonest disease sites were lymph node TB (25/61, 41.0%) and pulmonary TB (23/61, 37.7%). Twenty-two patients had fully sensitive TB and four had documented drug-resistant disease. Treatment was

Abstract P98 Table 1



completed successfully in 56/61 (91.8%) patients. Five patients had alternative outcomes, including treatment discontinuation due to adverse effects, loss to follow-up, transfer abroad, or insufficient outcome documentation.

Of the eight screened patients, two had positive LTBI tests and four had negative results; documentation was incomplete in the remainder.

Conclusions A substantial proportion of patients diagnosed with active TB in this cohort had not undergone prior LTBI screening despite recent arrival from high TB burden countries. These findings suggest substantial missed opportunities for preventive intervention and highlight the need to improve identification and screening of eligible migrants in accordance with national guidance. Optimising LTBI screening pathways could substantially reduce progression to active TB and contribute meaningfully to TB elimination efforts in low-incidence settings.

P99 SOCIAL RISK BURDEN AND TREATMENT OUTCOMES AMONG PATIENTS WITH TUBERCULOSIS: A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.260

Background Social risk factors are important determinants of tuberculosis (TB) outcomes and are recorded in TB surveillance as alcohol or drug abuse, homelessness, imprisonment, mental health needs and asylum seeker status.

Methods We conducted a retrospective cohort study using routinely collected TB surveillance data of all consecutive patients who were treated at our institution between 2021 and 2024. The association between social risk factors and TB treatment outcomes was assessed using multivariate logistic regression, adjusting for age, sex, migrant status and mono/ multi drug resistant TB using STATA 19MP.

Results 909 individuals with a diagnosis of active TB were included in the analysis. Median age was 35 years (IQR 26-51), 583 (64 %) were male and 765 (84.1%) were migrants. 68 (7.5%) of patients had drug resistant TB, 695 (76.5%) patients successfully completed treatment, there was no difference in treatment completion in non-migrants compared to migrants. There was a significant association between migrant status and social risk burden ($p < 0.001$), non-migrants were more likely than migrants to have one or more recorded social risk factors (37.9% vs 26.5%). In multivariate logistic regression; social risk burden was independently associated with lower odds of treatment success. Compared with individuals with no social risk factors, those with one social risk factor had 44% lower odds of treatment success (adjusted OR 0.56, 95% CI 0.38–0.82; p=0.003), while those with two or more social risk factors had 53% lower odds of treatment success (adjusted OR 0.47, 95% CI 0.28–0.78; p=0.004). Drug resistance was independently associated with poorer treatment outcomes (aOR 0.28, 95% CI 0.17–0.47; p<0.001). Age, sex, and migrant status were not significantly associated with treatment success.

Conclusions Social risk factors were strongly associated with poorer TB treatment outcomes, with increasing social risk burden associated with lower odds of treatment success. Although migrants were less likely than non-migrants to have documented social risk factors, migrants did not have worse treatment outcome after adjustment. These findings highlight the importance of including other social determinants such as insecure housing, unemployment, financial insecurity, language barriers, and migration-related vulnerabilities in TB risk assessment.

P100 TUBERCULOSIS (TB) RELATED DEATHS:10 YEAR ANALYSIS AND COMPARISON OF OUTCOMES BETWEEN WHITE UK-BORN AND OTHER ETHNIC GROUPS

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10.1136/thorax-2026-BTSabstracts.261

Background & Objectives UKHSA reports deaths rates in TB notifications at ~3-5% with deaths from pulmonary disease (~6%) higher than extrapulmonary (~2.5%). The demographic in semi-rural Shropshire (population ~500,000) over the past decade has seen an increase in ethnic minority groups, collectively now 6-7%. Analysing data in the decade from 2013, objectives were (1) to describe the populations and extent of disease and co-morbidity, and (2) comparing deaths in the White UK-born group from the other ethnic groups collectively.

Methods Retrospective analysis of all adults (age >16 years) with TB from 2013 to 2023. Data were taken from hospital and community GP computer records. Deaths were 'early' if within two years of notification. Comparisons are by Pearson Chi-Square ($\chi 2$) or linear association and regression analysis with statistical significance at $p < .05$.

Results Of 142 (63% males) notifications, 80 (56%) were White UK-born; they were older with mean age 60.4 (SD18.5, range 18-96) years compared to the 62 from other ethnic groups (34 South Asians, 10 Black Afro-Caribbeans, 15 Eastern Europeans, and 3 others) at 38.5 (13.2, 16-87) years, $p < 0.001$. Notifications were 12 times higher among the ethnic minority groups. Comparing co-morbidities identified only excessive alcohol use in the White UK-born group. Pulmonary disease was more represented in the White UK born group (52.5% vs 37.1%) and conversely extrapulmonary disease (26.3% vs 41.9%) among the ethnic minorities with no differences when occurring together (21.3% vs 20.9%). 30/142 (21%) with median

Abstract P100 Table 1

Deaths	White UK-born	Other ethnic groups	Total
Surviving	50	62	112
Early deaths (up to 24 months)	19	0	19
Late deaths (after 24 months)	11	0	11
Total	80	62	142

survival 15.5 (IQR 1-36.25) months had died by 2025; of these, all 30/80 were in the White UK-born group (χ2 29.478 df 1, p <.001) and table 1 further separates early from later deaths confirming statistical difference in mortality (χ2 25.284 df 1, p <.001). Regression showed only increased age and White UK-born (p= 0.002) contributed to early deaths.

Conclusions Data (1) confirm TB is at least a 12 times higher rate among the ethnic minority groups, (2) the low annual death rate <3% overall, (3) proportionately more early deaths in the white UK-born, with (4) contributing older age with more pulmonary disease (52% vs 37%).

Global and public health: future aspects with impact

P101 REUSING INHALERS IN LUNG FUNCTION REDUCES COST AND CARBON FOOTPRINT

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10.1136/thorax-2026-BTSabstracts.262

Introduction The NHS has committed to reduce the environmental impact of healthcare and go Net Zero by 2040. Medicines account for 25% of NHS emissions, inhalers 3%.

Bronchodilator response is assessed as part of lung function testing. Baseline spirometry is performed, salbutamol is administered and spirometry is repeated. ARTP and BTS guidance[1] state that 4x100mcg salbutamol via spacer or 2.5mg via nebuliser can be used for bronchodilation. Patients are asked to bring their own inhaler, but many do not have one, or forget. Widespread UK practice is for a single pressurised metered dose inhaler (pMDI) to be used per patient, resulting in up to 196 doses being wasted per inhaler.

The Chest Unit at King's College Hospital performs 5400 bronchodilator response tests, using 5400 Salamol inhalers a year. Salamol has a high carbon footprint as hydroflurocarbon HFA134a forms the propellant, with a global warming potential 1300 times that of CO2. The carbon footprint of these inhalers is 64530kgCO_2e a year, equivalent to driving 6 times round the equator in an average petrol car.

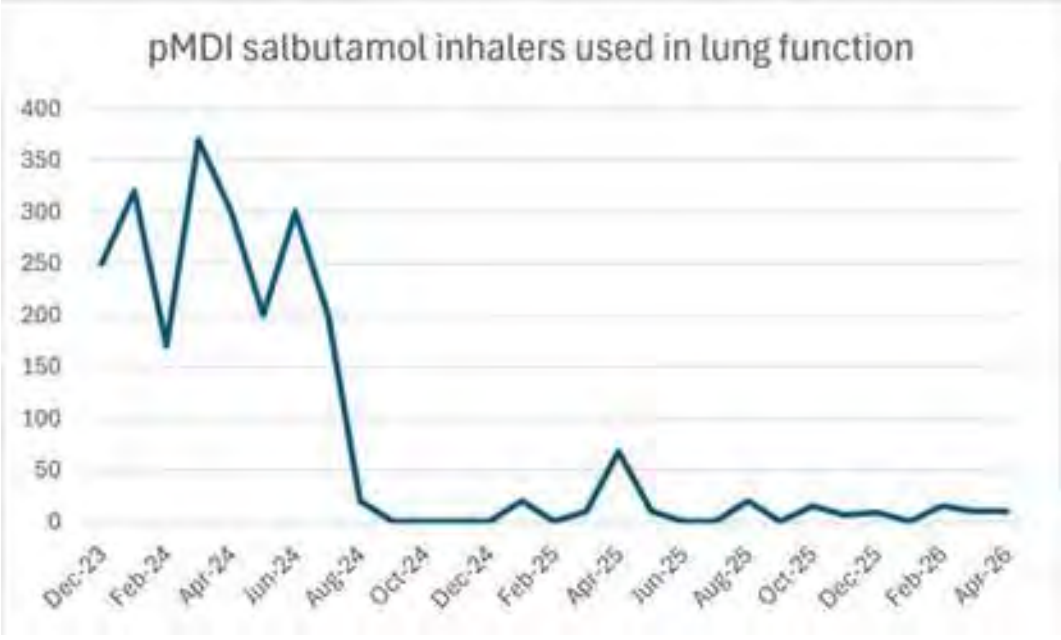
Methods In collaboration with the infection control team, we created a new protocol which allowed for cleaning and re-use of each pMDI inhaler across multiple patients. Factors considered included: cross-infection risk; staff time; staff acceptance; risk of inhaler degradation after multiple cleaning episodes; patient experience; and environmental sustainability. We proposed that at 4 monthly intervals all canisters be disposed via pharmacy for recycling or incineration to prevent inhalers running out of active drug, or of canister breakdown.

Results Prior to the change, monthly pMDI inhaler use was 264 inhalers (mean, Dec 2023 – July 2024), at a financial cost of £398.64 and a CO2e of 3154kg/CO_2e per month. Following change to the new protocol, the monthly pMDI inhaler use was 10 inhalers (mean, Sep 2024 - Apr 2026). An annual cost saving of £4602 and CO2e reduction of 36,414 kg/CO_2e was achieved. No adverse events have been recorded.

Conclusions Re-using pMDI inhalers for multiple patients for bronchodilator response testing is safe, cost saving and reduces carbon footprint.

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Abstract P101 Figure 1

P102 TEMPORAL TRENDS IN MORTALITY DUE TO ALPHA-1-ANTITRYPSIN DEFICIENCY ACROSS 30 EUROPEAN COUNTRIES FROM 2011-2021

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10.1136/thorax-2026-BTSabstracts.263

Background The rare genetic condition, alpha-1-antitrypsin deficiency (AATD) is associated with premature mortality,[1] but contemporary Europe-wide mortality data are limited. This hinders accurate assessment of disease burden in the era of emerging therapies. Here, more up to date mortality statistics across 30 European countries are provided. Comparison of death rates with research outputs (measure of disease expertise) on AATD adds context to the trends and inter-country mortality.

Methods Data were extracted from the EUROSTAT database, using the International Classification of Diseases 10 (ICD-10) code E88.0 from 2011-2021. Age-standardised mortality rates (SMRs) were calculated overall and by country, with temporal trends estimated using Poisson regression. Country-level AATD publication counts, excluding case reports, were extracted from Scopus Analytics (1-Jan-1997 to 31-Dec-2021) and correlated with mortality rates using Pearson's coefficient. Sensitivity analysis, assessing the trends between 2011-2018, was conducted to mitigate the effects of the COVID-19 pandemic and UK leaving the dataset (a noticeably high outlier in SMR). Statistical analyses were carried out in Python or Stata.

Findings The overall SMR in Europe was 5.4 (95% CI: 5.2-5.5) per 10,000,000 person-years, with substantial inter-country variation (table 1). There was a 6-fold difference between countries with the greatest and least SMR, in those that recorded 100 deaths or more. Although an apparent 4% decrease in annual death rates was observed between 2011-2021, this trend was not sustained in sensitivity analysis (2011-2018) [unadjusted mortality rate ratio (95% CI): 0.99 (0.97-1.00]. There was a 3-fold increase in the number of publications from 1997 to 2021, with a moderate positive correlation between publication rates and SMR (R=0.65; p=0.02)

Interpretation The geographic variation likely represents heterogeneities in reporting true disease burden. The positive correlation between publications and deaths suggests that countries with greater awareness of AATD may diagnose and document related deaths more frequently. The absence of a mortality decline despite increased research activity suggests a lag between knowledge generation and clinical impact. These novel observations support the need for coordinated pan-European efforts to raise awareness and implement evidence-based care in AATD.

Abstract P102 Table 1 Number of deaths and mortality rates for alpha-1-antitrypsin deficiency between 2011-2021 stratified by countries reporting 10 or more deaths. CMR: crude mortality rates; SMR: standardised mortality rates; CI: confidence interval

Country	Deaths	CMR per 10,000,000 Population (95% CI)	SMR per 10,000,000 Population (95% CI)
Austria	59	6.2 (4.7-8.0)	5.8 (4.3-7.3)
Belgium	82	6.6 (5.3-8.2)	6.1 (4.8-7.4)
Czechia	15	1.3 (0.7-2.1)	1.4 (0.7-2.1)
Denmark	52	8.3 (6.2-10.9)	7.8 (5.7-9.9)
Finland	41	6.8 (4.9-9.2)	5.9 (4.1-7.7)
France	218	3.0 (2.6-3.4)	2.7 (2.3-3.1)
Germany	482	5.4 (4.9-5.9)	4.5 (4.1-4.9)
Greece	10	1.2 (0.6-2.1)	0.9 (0.3-1.4)
Hungary	10	0.9 (0.4-1.7)	0.9 (0.4-1.5)
Ireland	42	8.0 (5.8-10.9)	9.8 (6.8-12.8)
Italy	788	11.9 (11.1-12.8)	8.8 (8.1-9.4)
Netherlands	112	6.0 (4.9-7.2)	5.8 (4.7-6.8)
Norway	52	9.1 (6.8-11.9)	9.4 (6.8-12.0)
Portugal	91	8.0 (6.4-9.8)	6.9 (5.5-8.3)
Spain	157	3.1 (2.6-3.6)	2.7 (2.3-3.1)
Sweden	152	14.0 (11.8-16.4)	12.8 (10.8-14.9)
Switzerland	56	6.1 (4.6-8.0)	5.8 (4.3-7.3)
Turkey	101	1.2 (1.0-1.4)	2.2 (1.7-2.6)
UK	520	10.1 (10.0-10.1)	10.0 (9.1-10.8)
All Europe	3,040	5.6 (5.4-5.8)	5.4 (5.2-5.5)

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P103 LUNG FUNCTION, ASTHMA, AND RESPIRATORY SYMPTOMS IN NEPALI CHILDREN AND ADOLESCENTS: AN ANALYSIS FROM NEPAL FAMILY COHORT STUDY (NEFCOS)

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10.1136/thorax-2026-BTSabstracts.264

Background Despite the burden of lung diseases being the highest in Nepal, there is a lack of lung function data from children and adolescents. Prior to our study, Nepal had no longitudinal study in children and adults with repeated measures of lung health, including lung function.

Aim To understand the trajectories of lung function in children and adolescents.

Methods This Nepal Family Cohort study (NeFCoS) measured lung function in 8591 participants aged 5 to 14 years, out of a total of 9225 at baseline, using standard ERS/ATS guidelines. We conducted a two-sample t-test in children (5-9 years) and

adolescents (10-14 years) to assess differences in lung function indices (FEV1 and FVC) and to determine whether sex contributed to variation in these parameters. Additionally, we estimated the proportions of pastyear wheeze, wheeze without colds, and everdiagnosed asthma using the ISAACderived questionnaire.

Results The mean and ±SD of lung function indices (in L) were higher in boys [children: (FEV_1: 1.06±0.28, FVC: 1.18±0.31); adolescents: (1.63±0.42, 1.83+0.46)] compared to girls [children: (1.01±0.27, 1.13±0.30); adolescents: (1.60±0.41, 1.77±0.45)]. Wheeze in the previous year was more prevalent in boys (11.8% versus 10.4%) and was associated with reductions in FEV_1 and FVC across strata, particularly among adolescent boys ($p < 0.05$). On the contrary, compared to boys, more girls reported having asthma (4.0% vs 4.7%) and wheeze without colds (5.5% vs 6.4%).

Conclusion The NeFCoS study offers comprehensive lung function data from Nepal, collected from adolescence to adulthood, with the first follow-up currently underway. It will evaluate factors affecting lung health, such as early diagnosis of lung diseases, to inform an early-intervention study aimed at identifying those associated with the initial stages of lung health decline.

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P104 INTEGRATING FIELD SURVEILLANCE AND IMMUNOLOGICAL ASSESSMENT TO ENHANCE TUBERCULOSIS CONTACT TRACING IN THE GAMBIA INSIGHTS FROM THE TBVAC STUDY

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10.1136/thorax-2026-BTSabstracts.265

Background Despite major progress in tuberculosis (TB) control, early immunological mechanisms that define protection versus infection remain poorly understood. The TBVAC Study integrates field-based contact tracing with immunological profiling to identify early immune correlates of protection against Mycobacterium tuberculosis(M.tb) among household contacts in The Gambia.

Methods The study was conducted across 12 health facilities in the Greater Banjul Area. Newly diagnosed GeneXpert-confirmed pulmonary TB index cases were identified, and their household contacts were traced within 14 days of treatment initiation. Adults aged 18-65 years with close exposure (shared household ≤ three months before diagnosis) were enrolled after informed consent. Whole blood and plasma samples were collected for immunological, molecular, and transcriptional analyses. PBMCs were isolated to characterize cytokine and gene-expression signatures to identify early immune correlates of protection. Participants were followed longitudinally at 6 and 12 weeks to monitor infection conversion using IGRA.

Results A total of 112 GeneXpert-confirmed pulmonary TB index cases (70 males, 42 females) were identified across participating facilities. Among these, 39 (35%) had medium and 68 (61%) had high bacterial loads on GeneXpert, indicating substantial transmission potential. Through household tracing, 213 contacts were screened and 200 enrolled in follow-up assessments. Among enrolled contacts, 140 (70%) were female, and 126 (63%) shared the same household with index cases, reflecting high exposure intensity. Baseline immunological assessment showed that 112 (57%) household contacts were QFT-positive, indicating latent TB infection (LTBI). Of the 107 contacts followed up within 12 weeks, 90 completed repeat assessments. During follow-up, 16 (8%) converted from QFT-negative to QFT-positive status, suggesting recent TB infection, while 45 (23%) remained persistently QFT-positive.

Conclusion The TBVAC Study demonstrates the feasibility of integrating community-based TB contact tracing with immunological research in The Gambia. Findings highlight sex differences in exposure, early immune activation patterns, and the potential of IGRA conversion as a biomarker for early infection. Recruitment is complete, and follow-up is ongoing.

P105 FIVE-MINUTE EXPOSURE TO TRAFFIC-RELATED AIR POLLUTION AND ADHERENCE OF PARTICULATE MATTER ON CIRCULATING RED BLOOD CELLS

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10.1136/thorax-2026-BTSabstracts.266

Introduction and Objectives In a recent study, we showed that 60 min exposure to traffic-related air pollution particulate matter (PM) resulted in the appearance of PM adherent to circulating red blood cells (RBCs).[1] In the present study we sought to determine the effect of a shorter exposure to traffic-related air pollution, and the effect of wearing a FFP2 mask.

Methods After informed consent (Ethical reference QME23.0041), healthy adults were positioned next to a main road in Whitechapel (London) for 5 min. Personal exposure to ambient black carbon (BC) PM was measured using an aethalometer, in ng/m^3/min. Capillary blood was collected before exposure to traffic emissions and immediately after 5 min exposure. Some participants provided blood samples hourly for 8 hr post exposure, and in some, exposure was conducted while wearing a FFP2 mask. Unstained blood smears were analysed using light microscopy and the mean area (μm^2) of BC PM adherent to 3000 RBCs (BC-RBC)determinedby ImageJsoftware.Paired data were analysed using Wilcoxon test.

Results Traffic exposure significantly increased BC exposure in all participants (n=19; median (IQR) BC exposure 100.20 (76.67-211.20) ng/m^3/min pre-exposure, vs 4556 (2218-9576) ng/m^3/min during exposure; p<0.0001). In participants exposed to traffic without a FFP2 mask, 5 min of exposure significantly increased BC-RBC (n=11, median (IQR) BC-RBC $7.60x10^{-5}$ ($0.00-1.11x10^{-4}$) μm^2 vs $1.26x10^{-4}$($3.53x10^{-5}$-$1.87x10^{-4}$) μm^2, p<0.05; figure 1a). BC-RBC returned to baseline between 1 and 6 hr post exposure (n=2, figure 1b). In the remaining participants, wearing a FFP2 mask during traffic exposure attenuated the increase in BC-RBC, with no significant difference in BC-RBC pre-and post-exposure (n=8, median (IQR) BC-RBC $8.58x10^{-5}$ ($5.03x10^{-5}$-$1.88x10^{-4}$) μm^2 vs $6.55x10^{-5}$($2.66x10^{-5}$-$1.66x10^{-4}$) μm^2, p=0.23; figure 1c).

Conclusion Exposure to traffic-related air pollution for 5 minutes results in translocation of inhaled BC and adhesion to circulating RBCs in healthy adults. BC remain adherent to RBC

for up to 6 hr before returning to baseline. Wearing FFP2 masks during short period of exposure to traffic reduces translocation of inhaled traffic-related PM.

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P106 CROSS-REGIONAL DISPARITIES IN ACCESS TO TUBERCULOSIS HOSPITAL CARE IN INDONESIA, 2020–2024

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10.1136/thorax-2026-BTSabstracts.267

Introduction Indonesia ranks second globally for tuberculosis (TB) incidence (WHO 2024). As a developing country with an archipelagic geography over 17,000 islands, health facilities are unevenly distributed, with advanced referral hospitals concentrated in Java while eastern regions have far fewer, forcing residents in remote areas to travel long distances for adequate care. This may drive patients to seek hospital care across regions. We aimed to map cross-region travel and identify regional disparities using national insurance claims data over five years.

Methods We conducted a serial cross-sectional analysis of BPJS Kesehatan Sample Data (2020-2024), including members with a TB diagnosis (ICD-10 A15-A19) attending referral (hospital) facilities. Province of residence was compared with province of the treatment facility, grouped into six regions. Patients were classified as cross-region if treated outside their region of residence, aggregated to one observation per patient-year. Analysis was weighted using BPJS sampling weights; regional differences were tested by chi-square.

Results Among 24,304 patient-year observations, 1.3% (95% CI 1.1-1.4) accessed referral care outside their region of residence, varying significantly across regions ($p<0.001$). Rates were highest in Maluku-Papua (4.3%, 95% CI 3.3-5.3), followed by Kalimantan (3.9%, 3.2-4.7), Sulawesi (1.7%, 1.2-2.2), Bali-Nusa Tenggara (1.5%, 0.9-2.1), Sumatra (1.3%, 1.1-1.6), and lowest in Java (0.8%, 0.6-1.0). Notably, Java had the highest cross-province travel (5.2%) but the lowest cross-region travel, likely reflecting easier access across provinces. Of all cross-region visits, 36.9% were directed to Java, the largest national referral hub. Destinations clustered around national hubs (Central Java 11.4%, Jakarta 10.4%), while South Sulawesi (12.1%) emerged as a regional hub for eastern Indonesia.

Conclusion TB patients in Maluku-Papua, followed by Kalimantan, had substantially higher cross-region travel than those in Java, where the national capital and major referral centers are located. These patterns indicate the need for further investigation into barriers that exist in these regions.

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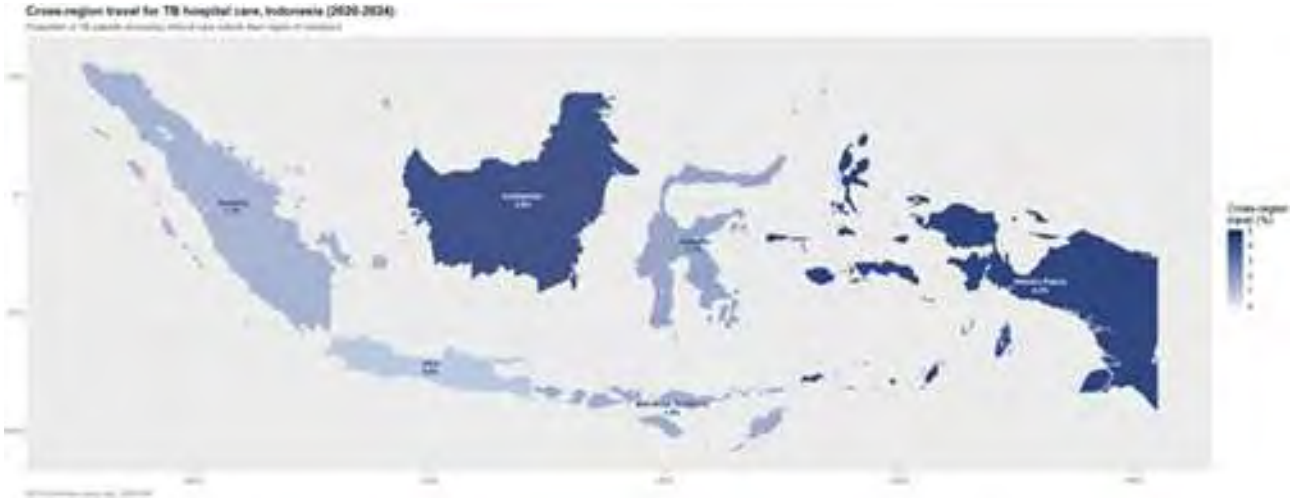
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P107 HIGH AMBIENT TEMPERATURE AND RESPIRATORY EMERGENCY ADMISSIONS DURING THE WARM SEASON: A DAILY TIME-SERIES ANALYSIS

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10.1136/thorax-2026-BTSabstracts.268

Background High ambient temperature is associated with respiratory morbidity, but evidence from temperate climatic regions remain limited. We conducted a daily time-series study in a temperate UK setting to investigate associations of emergency respiratory hospital admissions in relation to ambient



Abstract P106 Figure 1

temperature, during warm season (May–September) periods from 2008 to 2024.

Methods We analysed emergency respiratory admissions from a single NHS Trust in England during warm seasons from 2008 to 2024. Respiratory admissions were defined using primary ICD-10 codes J00–J99, with chronic lower respiratory disease (CLRD; J40–J47) examined as a secondary outcome. Temperature, air pollution and pollen data were obtained from fixed monitoring sources, with daily maximum temperature used as the main exposure metric. Distributed lag non-linear quasi-Poisson models estimated associations over lag 0–7 days, adjusting for temporal trends, relative humidity, ozone, pollen, day of the week, public holidays and year-specific effects. Relative risks were estimated at 24°C, 26°C and 29°C, corresponding to the 90th, 95th and 99th percentiles, using 19°C as the reference.

Results Across 2,601 warm-season days, there were 70,586 respiratory admissions, including 20,492 (29%) CLRD admissions. In the fully adjusted model, compared with 19°C, the cumulative risk over lag 0–7 days was 12.0% higher at 29°C (99th percentile; RR = 1.120, 95% CI: 1.038–1.208), whereas associations at 24°C and 26°C over lag 0–7 days were positive but not statistically significant. Cumulative risks at 24°C and 26°C peaked over lag 0–4 days, corresponding to 5.0% and 7.4% increases, respectively. Lag-specific increases were concentrated at lags 1–3. CLRD admissions showed a similar association at 29°C, with a 15.7% increase in cumulative risk (RR = 1.157, 95% CI: 1.005–1.331). Subgroup analyses showed increased risks among females, adults aged 16–69 years, older adults (>=70 years), and urban residents.

Conclusion Higher warm-season maximum temperature was associated with short-delayed increases in emergency respiratory hospital admissions, supporting the need to consider respiratory morbidity in local heat-health adaptation planning.

P108 REDUCING THE ENVIRONMENTAL IMPACT OF INHALER DISPOSAL IN SECONDARY CARE: A QUALITY IMPROVEMENT PROJECT

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10.1136/thorax-2026-BTSabstracts.269

Introduction and Objectives Inhalers account for approximately 3% of the NHS carbon footprint. A significant proportion of this impact arises from hydrofluoroalkane propellants used in pressurised metered-dose inhalers (pMDIs), which have high global warming potential. Even after use, residual propellant remains within these inhalers and can be released into the atmosphere if disposed of incorrectly.

Prior to this project, only a small number of inhalers were returned to the two inpatient pharmacies at our trust for correct disposal, and there was no inhaler recycling available. This quality improvement project (QIP) aimed to improve the sustainability of inhaler disposal by increasing inpatient inhaler returns and introducing inhaler recycling for pMDIs.

Methods Interventions included implementation of inhaler recycling bins within inpatient pharmacies, stakeholder engagement, review of existing disposal processes, and staff awareness initiatives.

Data were collected on inhalers returned from inpatient clinical areas over a 6-month period (September 2025-February 2026). Potential CO_2 equivalent (CO_2e) savings were estimated using values supplied by the recycling provider (Grundon), for average discarded inhalers. We also recorded the types of inhalers and used reference values[1] to calculate the maximum CO_2e savings if all recycled inhalers were full, for comparison.

Results A total of 521 inhalers were returned over the 6-month period, of which 382 were pMDIs suitable for recycling. Using the average value given (4.02kg of CO_2e per discarded inhaler), estimated CO_2e savings were 1543.28 kg CO_2e. Maximum savings, were all inhalers full, would have been 6682.8kg CO_2e.

The project identified a lack of staff awareness about correct inhaler disposal, variation in inhaler disposal practices, and enthusiasm for wider implementation of inhaler recycling.

Conclusions Implementation of inpatient inhaler recycling across two hospital sites was feasible and associated with reduced emissions. Improving inhaler return processes and staff awareness are essential for any recycling scheme to succeed, and we are continuing to work on this. As calculations were based on average discarded inhalers, actual carbon impact reduction may be higher, depending on residual inhaler content at time of disposal.

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P109 RESPIRATORY IMPLICATIONS OF RECENT WEIGHT LOSS PHARMACOTHERAPIES: A NARRATIVE REVIEW OF GLP-1 AND DUAL GIP/GLP-1 RECEPTOR AGONISTS

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10.1136/thorax-2026-BTSabstracts.270

Background Adverse effects due to glucagon-like peptide-1 receptor agonists (GLP-1RAs) and dual glucose-dependent insulinotropic polypeptide/glucagon-like peptide-1 receptor agonists (GIP/GLP-1RAs) have been reported in recent literature. The main reported effects have been on the gastrointestinal tract. The pulmonary and thoracic side effects are under-reported. We reviewed the current evidence on thoracic and respiratory outcomes associated with these injectable medications.

Methods A literature review was conducted in the OVID database with no year restrictions, using the keywords respiratory, pulmonary, thoracic and side effects, adverse effects, implications, complications, and GLP-1RAs or dual GIP/GLP-1RAs. Evidence from observational studies, randomised controlled trials, systematic reviews, meta-analyses and perioperative guidance documents was examined. A narrative synthesis of reported implications and complications was undertaken, focusing on obstructive sleep apnoea (OSA), asthma, chronic obstructive pulmonary disease (COPD), pulmonary hypertension, aspiration-related complications and perioperative respiratory outcomes.

Results Evidence suggests that GLP-1RAs and GIP/GLP-1RAs have notable effects on the respiratory system. In OSA, these medications were associated with significant reductions in apnoea-hypopnoea index and hypoxic burden. In asthma, studies reported fewer exacerbations and improved symptom

control in patients with obesity, potentially due to both weight reduction and anti-inflammatory mechanisms. In COPD, studies have shown reductions in exacerbation frequency and related hospitalisations. Data on respiratory infections were mixed, although several studies reported lower rates of bronchitis, pneumonia, and upper respiratory tract infections. In the post-operative period, the key respiratory concern is delayed gastric emptying, which may increase the risk of regurgitation, microaspiration, aspiration pneumonia, and pulmonary aspiration during anaesthesia or sedation.

Conclusion GLP-1RAs and dual GIP/GLP-1RAs appear to have positive effects on respiratory disease, particularly in OSA, asthma, and COPD, while evidence for perioperative aspiration risk remains inadequate. Current evidence supports a balanced, individualised approach to perioperative management. Further studies are required to clarify long-term pulmonary safety, aspiration risk and the extent of the pulmonary effects independent of weight loss.

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The breath of childhood: science, innovation and care

P110 IMPACT OF A CLINICIAN REFERRAL PATHWAY TO CITIZENS ADVICE SERVICE IN ADDRESSING HEALTH INEQUALITIES IN PAEDIATRIC RESPIRATORY MEDICINE

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10.1136/thorax-2026-BTSabstracts.271

Introduction Children and young people (CYP) living in disadvantaged socioeconomic circumstances experience poorer health outcomes, with the gap in health inequalities continuing to widen. This is particularly evident in respiratory disease, where children from deprived areas have higher rates of asthma resulting in activity limitation or emergency hospital admission. Families of CYP with chronic illness frequently face additional challenges, including financial, housing and transport barriers.[1] Paediatric respiratory teams often recognise these issues but lack the resources to address them directly. We evaluated a social prescribing programme delivered through Citizens Advice Service (CAS) designed to provide targeted support for the wider social determinants of child health.

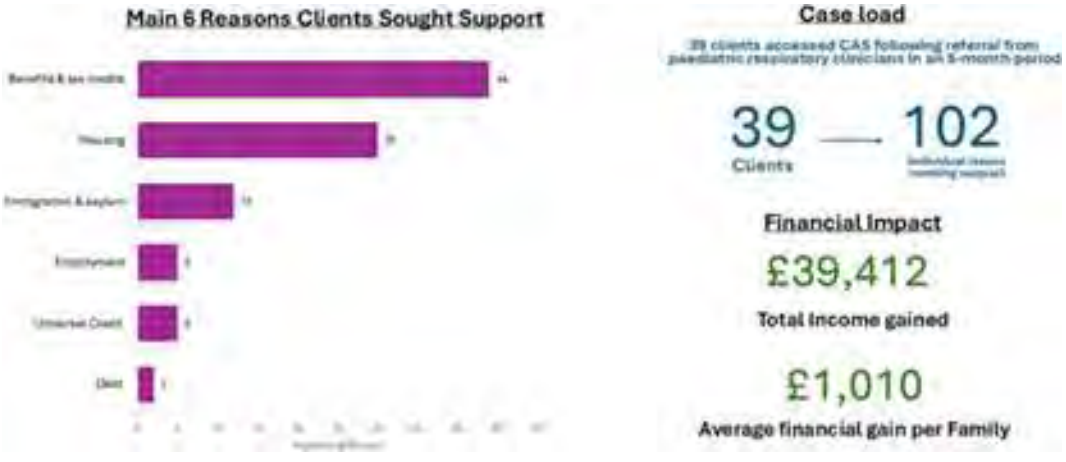
Methods Sheffield Children's Hospital's respiratory team established a social prescribing pathway, enabling clinicians to refer families directly to CAS. Specialist advisers provided support with issues such as benefits, housing, debt, employment, immigration, education and transport. Service activity and outcome data were retrospectively reviewed for families referred between August 2025 and March 2026.

Results 39 families engaged with CAS following respiratory clinician referral. Across these families, 102 individual issues were identified. The most common area of support was housing, accessed by 51% families, followed by benefits and tax credits (49%). Additional support included help with debt, immigration and asylum and employment with many families requiring assistance in multiple domains. The intervention generated a total income gain of £39,412 during the study period, equivalent to an average financial gain of £1011 per family.

Conclusions Families of CYP with chronic respiratory conditions experience significant social and financial challenges. The high number of issues identified in a relatively small cohort suggests the complexity of need within this population. This pathway successfully connected families with specialist support, resulting in meaningful financial gains and improved access to welfare and housing advice. Integrating social prescribing within paediatric respiratory services may represent a practical approach to addressing health inequalities and supporting family wellbeing. Research is needed to evaluate the impact of such interventions on long-term health outcomes.

REFERENCE

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Abstract P110 Figure 1 Support provided and financial impact of the CAS clinician referral pathway

P111 SHORT-TERM AIR POLLUTION EXPOSURE AND PEDIATRIC RESPIRATORY HOSPITAL ADMISSIONS IN ATHENS: A TIME-SERIES STUDY

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10.1136/thorax-2026-BTSabstracts.272

Children are particularly vulnerable to the respiratory effects of ambient air pollution, yet evidence on the short-term impact of regulated and emerging pollutants in Greece remains limited. We examined short-term associations between ambient air pollutants and daily respiratory hospital admissions in children aged 0–15 years in Athens. A population-based time-series analysis was conducted using daily pediatric respiratory hospital admissions during 2015–2019. Fixed-site measurements of regulated pollutants (PM2.5, PM10,NO2, O3) and emerging pollutants—black carbon (BC), its source-specific fractions (BCbb and BCff), and ultrafine particles (UFP)—were analyzed. Poisson regression models adjusted for overdispersion were applied, controlling for meteorological conditions, temporal trends, day of week, public holidays and seasonal population variation. Relative risks (RR) were estimated per interquartile range increase using lags up to six days and sensitivity analyses were performed for emerging pollutants in two-pollutant modelsadjusted for PM2.5 or NO2. Overall, 8,062 pediatric respiratory admissions were recorded (mean 4.42±3.78/day). Both PM2.5 and PM10 were significantly associated with admissions at lag0 (RR 1.047, 95% CI 1.003–1.093; RR 1.048, 95% CI 1.008–1.090). Increases in NO_2, BC, and UFP were associated with increased admissions on the same or previous day; however, these associations were not statistically significant. Following adjustment for PM2.5, only the association with UFP remained consistent, whereas adjustment for NO2 did not materially alter the findings. Short-term exposure to ambient particles was associated with increased same-day pediatric respiratory hospital admissions, supporting targeted air quality interventions.

P112 SHORT-TERM AIR POLLUTION EXPOSURE AND RISK OF AIRWAY INFLAMMATORY RESPONSE IN CHILDREN (CHERISH): A RANDOMISED MIXED FACTORIAL STUDY

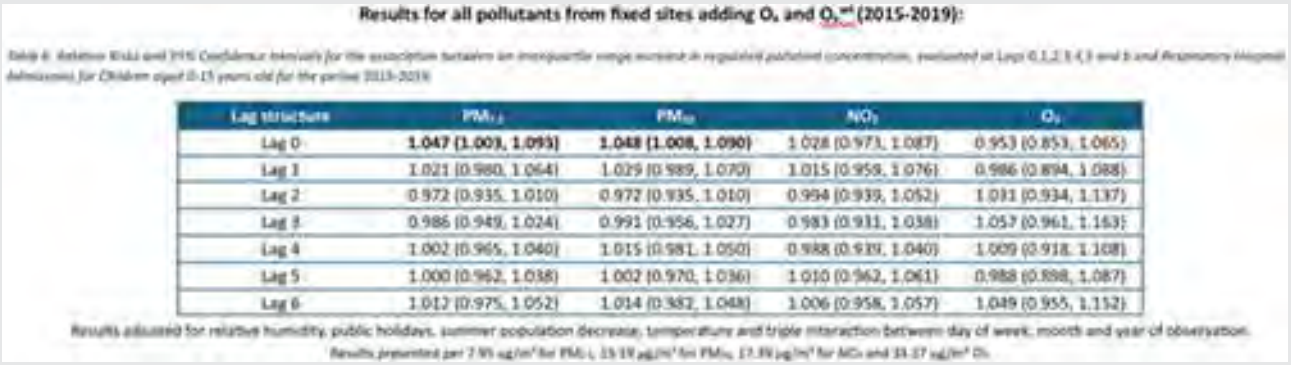
[1]S Moloney, [1]AC Thomson, [1]I Gonzalez Calvo, [1,2]H Hajmohammadi, [1,2]HE Wood, [3]MI Mead, [3]IS Mudway, [4]G Mosler, [1,2]J Scales, [4,2]A Whitehouse. [1]*Wolfson Institute of Population Health, Queen Mary University London, London, UK;* [2]*Centre for Applied Respiratory Research, Impact and Innovation, Queen Mary University of London, London, UK;* [3]*MRC Centre for Environment and Health, Environmental Research Group, Imperial College London, London, UK;* [4]*Centre for Genomics and Child Health, Blizard Institute, Faculty of Medicine and Dentistry, London, UK*

10.1136/thorax-2026-BTSabstracts.273

Introduction Due to their developing organs, children are particularly vulnerable to the adverse respiratory effects of air pollution. Physical activity may increase inhaled pollutant dose, yet the short-term effects of exercising in school playgrounds remains poorly characterised. CHERISH aims to investigate the impact of air pollution exposure during physical activity on airway function and inflammatory responses in primary school-aged children. We hypothesised that playground air pollution exposure is associated with acute reductions in lung function following physical activity, measured by oscillometric resistance at 5 Hz (R5).

Methods CHERISH is a randomised crossover mixed-factorial study with one within-subject factor (exposure condition: active vs. sedentary) involving children aged 8–11 years from London schools. Participants completed a 90-minute sedentary session and a 90-minute physical activity session in a randomised order, separated by at least two-week washout; all children wore activity monitors during sessions to characterise activity. Air pollution exposure was estimated using data from the Breathe London air quality network, with NO_2, PM_{10}, and $PM_{2.5}$ concentrations derived from sensors at participating school sites. Oscillometry was performed before, immediately after and 24-hours following exposure. Nasal lavage samples were collected before and at 24-hours after exposure for biomarker analysis, with interleukin-6 being the primary outcome variable. Participants with self-reported asthma performed baseline fractional exhaled nitric oxide (FeNO) measurement to classify asthma as T2-high or T2-low.

Abstract P111 Table 1 Relative risks and 95% confidence intervals for the association between an interquartile range increase in regulated pollutant concentration, evaluated at Lags 0,1,2,3,4,5 and 6 and respiratory hospital admissions for children aged 0-15 years old for the period 2015-2019



Results for all pollutants from fixed sites adding O_3 and $O_{3,ref}$ (2015-2019):

Lag structure	$PM_{2.5}$	PM_{10}	NO_2	O_3
Lag 0	**1.047 (1.003, 1.093)**	**1.048 (1.008, 1.090)**	1.028 (0.973, 1.087)	0.953 (0.853, 1.065)
Lag 1	1.021 (0.980, 1.064)	1.029 (0.989, 1.070)	1.015 (0.959, 1.076)	0.986 (0.894, 1.088)
Lag 2	0.972 (0.935, 1.010)	0.972 (0.935, 1.010)	0.994 (0.939, 1.052)	1.031 (0.934, 1.137)
Lag 3	0.986 (0.949, 1.024)	0.991 (0.956, 1.027)	0.983 (0.931, 1.038)	1.057 (0.961, 1.163)
Lag 4	1.002 (0.965, 1.040)	1.015 (0.981, 1.050)	0.988 (0.939, 1.040)	1.009 (0.918, 1.108)
Lag 5	1.000 (0.962, 1.038)	1.002 (0.970, 1.036)	1.010 (0.962, 1.061)	0.988 (0.898, 1.087)
Lag 6	1.012 (0.975, 1.052)	1.014 (0.982, 1.048)	1.006 (0.958, 1.057)	1.049 (0.955, 1.152)

Results adjusted for relative humidity, public holidays, summer population decrease, temperature and triple interaction between day of week, month and year of observation.

Abstract P112 Table 1 Baseline characteristics of participants enrolled in the CHERISH study

Characteristic	Value
Study population	
Participants recruited, n	330
Schools recruited, n	10
Age, years, mean (sd)	9.7 (0.24)
Female n (%)	155 (47.0)
Respiratory characteristics	
Self-reported asthma n (%)	32 (9.7)
Baseline FeNO*, ppb, mean (sd)	29.3 (20.2)
Ethnicity %	
Asian/Asian British, n (%)	126 (38.2)
Black/Black British, n (%)	67 (20.3)
Mixed ethnicity, n (%)	43 (13.1)
White/White British, n (%)	72 (21.9)
Other ethnic group, n (%)	20 (6.2)

Results A total of 330 children were recruited from 10 schools between November 2025 and May 2026 (table 1). Environmental exposure, oscillometry and nasal lavage data were collected across all study visits. Data cleaning and quality assurance are ongoing, with preliminary review indicating successful acquisition of exposure and physiological measurements. Analyses examining associations between air pollutant exposure, lung function and inflammatory biomarkers are underway and expected to be completed by November 2026.

Conclusions CHERISH will provide novel evidence on the acute respiratory and inflammatory effects of air pollution exposure during physical activity in an ethnically diverse group of children representative of the East London population. The findings may help inform public health guidance, school environmental policies, and strategies to reduce exposure in educational settings, particularly for children with asthma.

P113 WHAT IS THE ASSOCIATION BETWEEN HOUSEHOLD ENVIRONMENTAL EXPOSURES AND RESPIRATORY FUNCTION AMONG CHILDREN AGED 4-16 ATTENDING THE AIR POLLUTION CLINIC? A CROSS-SECTIONAL, OBSERVATIONAL STUDY

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Introduction and Objectives Childhood respiratory disease is heavily influenced by the social determinants of health. Children of lower socioeconomic status suffer higher rates of asthma, wheeze, hospital admissions, and poorer respiratory outcomes. The home environment is an important determinant of respiratory health, as children can be exposed to detrimental factors including damp, mould, secondhand smoke, combustion pollutants, and traffic-related air pollution. These environmental risks are particularly pronounced in deprived urban areas such as East London. Existing research on these associations has mainly focused on community-based populations. However, this study specifically examines the association between household environmental exposures and respiratory function among children aged 4–16 attending the Air Pollution Clinic at the Royal London Hospital, Whitechapel.

The objectives were to conduct a literature review, describe the characteristics of the cohort, compare the characteristics with national data where available, analyse univariable associations between household exposures and lung function as determined by the forced expiratory volume in one second to forced vital capacity ratio (FEV1/FVC), and identify consequences for clinical practice and future research.

Methods A cross-sectional observational study was conducted using routinely collected clinic data. Demographic, clinical, and environmental exposure variables were cleaned and coded in Stata. The primary outcome measure was lung function (FEV1/FVC). Descriptive statistics were used to summarise the cohort and household characteristics. Univariable linear regression analyses explored associations between household environmental exposures and FEV1/FVC.

Results 78 children were included, and complete FEV1/FVC data were available for 37. Household environmental exposures were more prevalent than national data indicate. No statistically significant associations were identified, but several exposures, including smoking and mould concerns, showed directional associations consistent with the existing literature. Confidence intervals were relatively wide throughout, indicating limited precision.

Conclusion This study adds to the limited evidence base in secondary care. Even though definitive associations could not be established, the findings support the continued relevance of considering housing and environmental factors within paediatric respiratory care. Larger, better-powered prospective studies using objective exposure measures are needed. Improving home environments may represent an important complement to conventional medical treatment for childhood respiratory disease.

P114 ENDOTRACHEAL ASPIRATES DEMONSTRATE HIGHER BACTERIAL BIOMASS AND SIMILAR MICROBIOTA COMPOSITION COMPARED WITH BRONCHOALVEOLAR LAVAGE IN CHILDREN WITH CHRONIC WET COUGH

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10.1136/thorax-2026-BTSabstracts.275

Paediatric lung microbiome studies are challenged by low-biomass samples, contamination risk and reliance on bronchoalveolar lavage (BAL), which is invasive and rarely justified in healthy children. Endotracheal aspirates (ETA) may provide a practical alternative, but whether ETA microbiota accurately reflect BAL microbiota in children with chronic wet cough (CWC) remains unclear. We hypothesised that ETA would more consistently pass quality-control thresholds while retaining microbiota profiles comparable to BAL.

Children with CWC (n=40, median age 4 years [IQR 2–5]) undergoing clinically indicated bronchoscopy and age-matched healthy controls (n=4, median age 3.5 years [IQR 2–4]) undergoing elective surgery were recruited. Paired BAL, ETA, nasopharyngeal (NP) and oropharyngeal (OP) samples were collected from children with CWC; ETA, NP and OP samples were collected from controls. BAL infection was identified in 18/40 children with CWC (45%). Bacterial load was quantified by 16S rRNA gene qPCR and microbiota composition assessed by

V4 16S rRNA gene sequencing. Contaminants were identified using decontam and a limit-of-detection (LoD) threshold was established using a serially diluted mock community.

Prior to microbiota filtering, bacterial load increased significantly from BAL to ETA, NP and OP (all $p<0.001$). Following contamination removal and LoD filtering, 24/40 BAL (60%) and 28/40 ETA (70%) CWC samples were retained. All healthy control ETA samples passed QC criteria (4/4, 100%). Comparative microbiota analyses were restricted to 20 CWC donors with paired BAL, ETA, NP and OP samples above the LoD. Alpha diversity did not differ between BAL and ETA, whereas NP showed reduced diversity and increased dominance, and OP greater richness. BAL–ETA beta-diversity dissimilarity was significantly lower than BAL–NP and BAL–OP dissimilarity using Bray–Curtis and Jaccard distances (all $p<0.001$). No genera differed significantly between BAL and ETA, whereas multiple genera differed between BAL and both NP and OP. ETA retained compositional similarity to BAL in children with culture-confirmed lower airway infection.

Following rigorous contamination control and biomass-based filtering, ETA generated higher-biomass microbiota profiles with greater sample retention than BAL while maintaining comparable microbial composition. ETA may provide a practical BAL surrogate and support accurate, LoD-informed microbiota quantification in healthy children.

P115 TREATMENT PATTERNS AND 12-MONTH OUTCOMES AFTER ACUTE PRESCHOOL WHEEZE PRESENTATIONS: THE PICTURE STUDY

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10.1136/thorax-2026-BTSabstracts.276

Introduction Recurrent preschool wheeze (PSW) results in frequent acute hospital attendances (Bloom; JACI:2021), but real-world treatment and outcomes are poorly described. In a prospective observational cohort study we describe treatment changes and disease outcomes over a 12-month period. No interventions were performed; treatment was directed by usual care clinicians.

Methods One hundred children aged 1–5 years presenting with an acute wheeze attack to a children's hospital were recruited and followed 4 monthly for 12 months. Demographics, index of multiple deprivation (IMD), prescribed treatments, wheeze attacks, symptom control (TRACK questionnaire) and caregiver quality of life (PACQLQ) were recorded. Changes across the four time points was tested with the Friedman test, Wilcoxon signed rank and McNemar tests.

Results 82/100 (82%) children median age 3.2 (IQR, 2-4.4) years, completed 12 months follow up. 86/100 had IMD scores of 1-4 (most deprived) and 55% from minority ethnic groups. The median attack rate in the previous 12 months was 3 (IQR 1.8-6), 80% were hospitalised in the preceding year, 52% were prescribed daily inhaled corticosteroids (ICS), 94% a short-acting bronchodilator and 17% montelukast. Over 12 months, treatment was changed in 35 children: escalation of ICS in 26 and step down or discontinuation in 15. Overall, during the 12 months, hospital admissions fell (median 2 (IQR 1-3) to 0.9 (IQR 0,1); ≥1 admission/child fell from 80% to 42%; McNemar $p<0.001$), unscheduled contacts fell (4.1 to 2.4; $p<0.001$) and OCS courses fell (2.0 to 1.1; $p=0.001$). Total wheeze attacks declined across the 4 monthly windows (Friedman $p<0.001$). Symptom control improved from 0–12 months (TRACK median 65 to 80; Friedman $p<0.001$), as did caregiver quality of life (PACQLQ 3.5 to 4.9; Friedman $p<0.001$).

Conclusions In an unselected group of children with PSW, healthcare use and attack severity fell, while symptom control and quality of life improved over 12 months. This was unrelated to any treatment intervention in the majority. A focus on phenotyping children to identify those who do not improve is needed to enable appropriate intervention to reduce wheeze attacks.

P116 A TOUGH PILL TO SWALLOW - ASSESSING THE IMPACT OF VIRTUAL PILL SCHOOL ON CHILDREN WITH CYSTIC FIBROSIS

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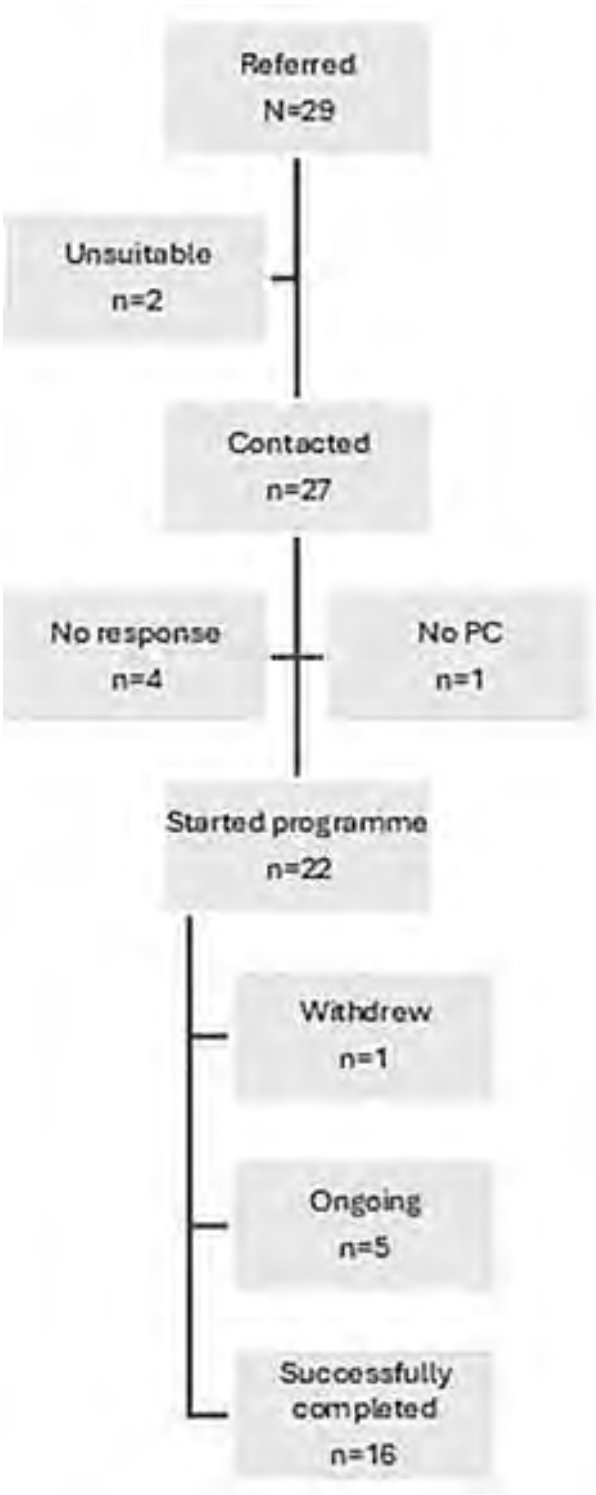
10.1136/thorax-2026-BTSabstracts.277

Introduction Difficulty swallowing tablets can adversely affect management of children with cystic fibrosis (CF), particularly pancreatic-insufficient patients requiring whole enzyme capsules. Also, highly effective modulator therapy requires children to swallow tablets when aged 6 years. A well-established in-patient Pill School, run by Health Play Specialists has proven effective; however, healthier children with CF have reduced inpatient admissions, leading to a demand for out-patient management of the issue. This project aimed to assess the effectiveness of Virtual Pill School, a remote programme using telephone and video appointments in children and young people.

Methods Clear referral guidance was in place to assess family suitability. Following consent, an introductory email offered telephone / video contact, leading to an assessment call evaluating child readiness, family commitment, programme details, and a start date. Support networks (parents/carers/siblings) were assessed. The child-led 10-stage programme employed graded 'sweet tablets' (from cake sprinkles to Creon, via decorations, moulded sweets, mini smarties, tic tacs) We required three successes per stage before progression (with a step back when necessary). Positive reinforcement included virtual stickers and completion certificates/gifts. A staff buddy system, dietary considerations, and visual aids (puppets) supported the scheme.

Results From May 2023 until currently, 29 referrals were received. Results are shown in the accompanying flow chart. The 2 children deemed unsuitable were due to an unsafe swallow, and a safeguarding issue. We have also had success in 5/7 children with cardiac conditions.

Conclusions Virtual Pill School offers a feasible and effective alternative to in-person support, allowing child-paced progress based at home. Success hinged on parental commitment (it involved 2–3 sessions/week), quiet spaces, clear boundaries, and visual modelling (especially for younger children and 2nd language families). Challenges included glitches with the remote video system, difficulties checking 'tablet' size, parental



Abstract P116 Figure 1

over-rewarding, reluctance in older children, and a need for high levels of energy and time from the staff. We learnt the importance of relaxed life-skill reinforcement and avoiding rushing the programme. Future tasks will include resource packs, formal feedback, cost-savings analysis, pharmacy verification for early modulators, and roll out in other specialties.

P117 PREDICTIVE ACCURACY OF STARWAVE, DIFFICULTY DRAWING BREATH, AND FEVERKIDSTOOL SCORES FOR HOSPITAL ADMISSION IN CHILDREN WITH SUSPECTED LOWER RESPIRATORY TRACT INFECTION (LRTI): A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.278

Introduction Identifying children in the community with suspected LRTI requiring hospital admission remains challenging. The 2025 NICE pneumonia guidelines (NG250) recommend further research to evaluate clinical prediction tools in this population, noting limited external validation data. STARWave, Difficulty Drawing Breath (DDB), and Feverkidstool (FKT) are three tools, yet their predictive performance in UK settings is unclear. We evaluated these metrics in GP-referred children presenting to a paediatric assessment unit (PAU).

Methods This was a retrospective review of electronic patient records at Gloucestershire Royal Hospital (WC260324). GP-referred patients assessed in PAU between 01/01/25–31/12/25 aged 28 days–18 years and discharged with an LRTI diagnosis were included. Exclusion criteria followed NICE NG250. Where available, GP data was obtained via integrated digital primary care records. The primary outcome was hospital admission. Univariable binary logistic regression assessed each prediction tools association with admission. Multivariable regression identified independent clinical predictors of admission.

Results 105 GP-referred children were included (median age 2.1 years, 58.1% male); 48 (45.7%) were admitted. Admitted patients had higher respiratory rates (p=0.006), higher heart rates (p=0.019), lower oxygen saturations (p=0.014), more chest retractions (p<0.001) and greater difficulty breathing (p=0.012) on initial presentation to the GP (table 1). Only 12/105 (11.4%) were eligible for FKT scores to be calculated; 11/12 (91.7%) had >10% risk of bacterial pneumonia and 10 of these were admitted. STARWave scores were higher in admitted patients (median: 3 vs 2, p=0.010). On univariable regression, normal-risk (OR 3.05, 95% CI 1.13–8.29, p=0.028) and high-risk (OR 3.57, 1.01–12.68, p=0.049) patients had higher odds of admission versus very low-risk patients. DDB scores were higher in admitted patients (median: 3 vs 2, p=0.019), though the regression model did not reach significance (p=0.120). On multivariable analysis, chest retractions (OR 3.57, 1.16–10.95, p=0.026) and moderate/severe vomiting (OR 4.64, 1.30–16.57, p=0.018) independently predicted admission.

Conclusions Only STARWave was significantly associated with admission. DDB scores were higher in admitted children but were not significant on regression. FKT was only calculable in 11% of patients. Chest retractions and vomiting were independent predictors of admission. These findings support the NICE recommendation for further validation.

Abstract P117 Table 1 Baseline characteristics by admission status (n=105)

Variable	Admitted (n=48)	Non-Admitted (n=57)	p-value
Demographics			
Age, median (IQR)	2.3 (1.6–3.6)	1.9 (1.4–3.4)	0.493
Age group, n (%)			0.092
Age <1 year	7 (14.6)	6 (10.5)	
Age 1–<2 years	10 (20.8)	24 (42.1)	
Age 2–<5 years	25 (52.1)	17 (29.8)	
Age 5–<12 years	5 (10.4)	7 (12.3)	
Age ≥12 years	1 (2.1)	3 (5.3)	
Male, n (%)	26 (54.2)	35 (61.4)	0.582
Female, n (%)	22 (45.8)	22 (38.6)	
Clinical Observations			
Temperature (°C), median (IQR)	38.0 (37.1–38.6)	37.9 (37.0–38.7)	0.621
Fever ≥37.8°C, n (%)	27 (56.2)	32 (56.1)	1.000
Fever ≥38.5°C, n (%)	15 (31.2)	16 (28.1)	0.888
Fever duration (days), median (IQR)	1.5 (0.0–4.2)	1.0 (0.0–3.0)	0.812
Respiratory rate (bpm), mean ± SD	43.0 ± 13.8	36.1 ± 11.5	**0.006**
Heart rate (bpm), median (IQR)	144.0 (130.0–158.5)	130.0 (120.0–145.0)	**0.019**
Oxygen saturation (%), median (IQR)	93.0 (90.5–96.0)	96.0 (93.0–98.0)	**0.014**
SpO_2 <94%	26 (54.2)	15 (26.3)	**0.007**
Clinical Signs			
Ill-appearance, n (%)	23 (47.9)	18 (31.6)	0.131
CRT prolonged (>3s), n (%)	3 (6.2)	3 (5.3)	1.000
Chest retractions, n (%)	31 (64.6)	17 (29.8)	**<0.001**
Difficulty breathing, n (%)	36 (75.0)	28 (49.1)	**0.012**
Grunting, n (%)	6 (12.5)	2 (3.5)	0.138
Wheeze, n (%)	16 (33.3)	12 (21.1)	0.232
Moderate/severe vomiting within 24 hours, n (%)	12 (25.0)	8 (14.0)	0.240
Investigations			
Blood test performed, n (%)	18 (37.5)	7 (12.3)	**0.005**
CRP (mg/L), median (IQR)	34.0 (19.5–58.7)	13.0 (7.0–33.0)	0.125
CRP >20 mg/L, n (%)	13 (27.1)	3 (5.3)	**0.005**
WCC ($\times 10^9$/L), mean ± SD	12.6 ± 4.9	5.8 ± 1.9	**0.002**
CXR performed, n (%)	30 (62.5)	16 (28.1)	**<0.001**
CXR abnormal finding, n (%)	22 (45.8)	10 (17.5)	**0.003**
Treatment			
Antibiotics given, n (%)	39 (81.2)	43 (75.4)	0.631
STARWave Score			
Total score, median (IQR)	3.0 (2.0–3.0)	2.0 (1.0–3.0)	**0.010**
Risk stratification, n (%)			0.054
Very low risk (0–1)	7 (14.6)	20 (35.1)	
Normal risk (2–3)	31 (64.6)	29 (50.9)	
High risk (≥4)	10 (20.8)	8 (14.0)	
DDB Score (ages 2–59 months, n=84)			
Total score, median (IQR)	3.0 (2.0–3.0)	2.0 (1.0–3.0)	**0.019**
Risk stratification, n (%)			0.109
Low risk (0–1)	9 (22.5)	18 (40.9)	
Intermediate risk (2–3)	24 (60.0)	23 (52.3)	
High risk (≥4)	7 (17.5)	3 (6.8)	
FKT Pneumonia Risk (n=12)			
Risk probability (%), mean ± SD	33.9 ± 22.1	85.5	(*)
FKT positive (>10% risk)	10 (90.9)	1 (100.0)	
FKT negative (≤10% risk)	1 (9.1)	0 (0.0)	

Normally distributed continuous variables reported as mean ± SD and compared using independent samples t-test. Non-normally distributed continuous variables reported as median (IQR) and compared using Mann–Whitney U test. Categorical variables reported as n (%) and compared using Chi-squared test or Fisher's exact test. (*) Insufficient sample size in non-admitted group (n=1) for statistical comparison.

P118 CURRENT PRACTICES AND BARRIERS IN PAEDIATRIC NON-INVASIVE VENTILATORY SUPPORT: A MULTI-CENTRE SURVEY

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10.1136/thorax-2026-BTSabstracts.279

Introduction The increased demand for non-invasive support in paediatric respiratory and sleep medicine and rising number of children with medical complexity are placing significant pressure on healthcare systems with limited resources. Hospital-at-home (HaH) presents a potential model to ease resource constraints. Non-invasive ventilation technologies, including Bilevel Positive Airway Pressure (BiPAP) and Continuous Positive Airway Pressure (CPAP), are widely used in hospitals and HaH. However, there remains limited understanding of ventilation practices across institutions. This study aimed to evaluate current practices and identify barriers to non-invasive support in paediatric respiratory and sleep medicine.

Methods Paediatric healthcare professionals including doctors, nurses and physiologists were invited to participate in an online survey to assess current practices around non-invasive support establishments, follow-up and potential barriers to care delivery.

Results 49 responses were received from 29 centres across 5 countries. BiPAP was the most commonly used modality, followed by CPAP, AutoCPAP, and Volume Assured Pressure Support (AVAPS or iVAPS). CPAP was most commonly used for obstructive sleep apnoea or obstruction (79.3%) and BiPAP for hypoventilation (100%). 62% of institutions initiate some non-invasive support at home.

The most common method of monitoring for compliance and gas exchange was within the hospital setting (69%), with home monitoring (59%) and remote monitoring via data clouds (28%). In stable patients, ventilatory compliance is routinely monitored by 76% of centres. Transcutaneous oxycapnography is used to monitor gas exchange in stable patients (72%), cardiorespiratory sleep study (59%) and oximetry (55%). Capillary blood CO2 and end-tidal CO2 were rarely measured (3% and 14% respectively). Initial monitoring commonly occurred within three months (59%). If remote monitoring was used, there was no clear consensus on frequency of data review.

Key healthcare barriers centred around resource limitations, including staffing (72%) and designated sleep beds (69%). Patient/family barriers were predominantly intolerance of equipment (97%) and mask issues (72%).

Conclusion The survey findings demonstrate wide variation in paediatric non-invasive support practices, alongside significant resource and patient-related barriers. These findings support the need to explore more sustainable and standardised care models, with greater integration of home-based and remote monitoring to ease demand for hospital-based care.

P119 OUTCOMES IN CHILDREN WITH TRISOMY 21 UNDERGOING DRUG-INDUCED SLEEP ENDOSCOPY DUE TO PERSISTENT OBSTRUCTIVE SLEEP APNOEA POST-ADENOTONSILLECTOMY

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10.1136/thorax-2026-BTSabstracts.280

Introduction Persistent obstructive sleep apnoea (OSA) post-adenotonsillectomy is common in children and young people (CYP) with Trisomy 21 (T21) due to multilevel airway obstruction. Drug-induced sleep endoscopy (DISE) is increasingly used to identify sites of upper airway obstruction and guide management, including DISE-directed surgery, non-invasive ventilation (NIV), and hypoglossal nerve stimulation (HNS). The impact of DISE on treatment pathways and clinical outcomes in CYP with T21 remains poorly characterised.

Aim To evaluate clinical outcomes following DISE in CYP with T21 and persistent OSA post-adenotonsillectomy.

Methods We performed a retrospective review of electronic patient records of CYP with T21 who underwent DISE at our institution between 2023 and 2025. Extracted data included demographic information, non-invasive ventilation (NIV) compliance (median usage [hrs/night]), and cardiorespiratory polygraphy parameters (obstructive apnoea hypopnoea index [OAHI]) before and after DISE. Patients who did not have a post-procedure sleep study were excluded. Data was analysed to assess intervention patterns and efficacy.

Results Seventeen children underwent DISE (median age 11.6 years [IQR 8.2 –13.9 years]). DISE identified the site of obstruction in all patients. Thirteen (76%) had DISE-directed surgery, frequently lingual tonsillectomy (n=12). Although 7 children (54%) had a reduction in OAHI following DISE-directed surgery, this was not statistically significant (mean OAHI change -1.2 evs/hr [sd 13.0], $P > 0.05$).

Twelve (71%) children had NIV pre-DISE; 100% had been non-adherent (median usage 1 hr/night). Following DISE, 8 children (47%) required NIV and had good adherence (median usage 7.25 hrs/night). Three children remained non-adherent to NIV and had HNS implanted with improvement in OSA seen (median OAHI: post-DISE 6.6 evs/hr [IQR 4.2 – 8.9], post-HNS 2.0 evs/hr [QR 1.8 – 2.2]).

Conclusions Non-adherence to NIV occurs frequently in children with T21. DISE is useful in the treatment of persistent OSA to identify the site of obstruction, which may be amenable to surgery. However, in several patients, DISE-directed surgery produced only partial improvements, reflecting multifactorial airway collapse in T21. Nonetheless, there were improved patient outcomes following DISE, including improved NIV adherence. In those who remain NIV non-adherent, HNS is an emerging procedure in children which appears effective in controlling persistent OSA.

P120 VARIATION IN IMMUNOLOGICAL INVESTIGATION FOLLOWING PAEDIATRIC EMPYEMA AND NECROTISING PNEUMONIA

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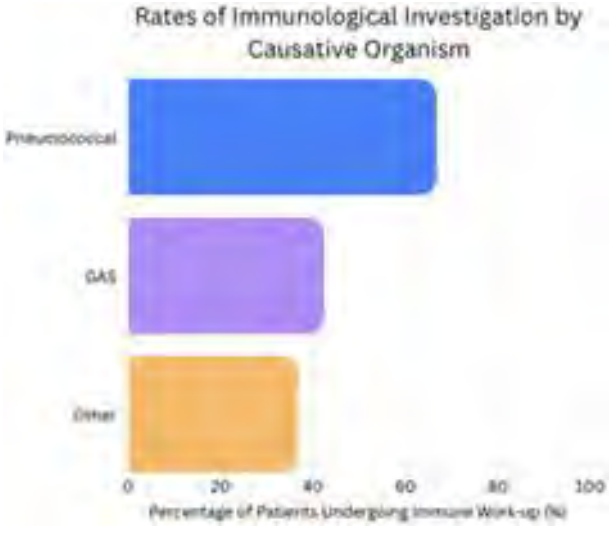
10.1136/thorax-2026-BTSabstracts.281

Introduction and Objectives Pleural empyema and necrotising pneumonia are significant causes of paediatric morbidity. Severe bacterial infection may occasionally indicate underlying immunodeficiency, although there is limited consensus regarding which children should undergo immunological investigation following empyema. This service evaluation assessed current investigation and follow-up practices within a tertiary paediatric respiratory service.

Methods A retrospective service evaluation was conducted of children diagnosed with pleural empyema or necrotising pneumonia over a three-year period to March 2025. Patients were identified from a departmental empyema database. Electronic patient records, microbiology results, laboratory systems and clinic documentation were reviewed. Data regarding causative organisms, immunological investigations, abnormal findings and follow-up were analysed.

Results Seventy-eight patients were included following exclusions. Group A Streptococcus was the most commonly identified organism (40%), followed by Streptococcus pneumoniae (18%). Overall, 45% (35/78) underwent at least one immunological investigation. Investigation rates varied between organism groups, with the highest rates observed in pneumococcal disease.

Abnormal immunological findings were identified in 15/78 (19%) patients in the entire cohort and in 15/35 (43%) of those who underwent investigation, the most common abnormality being suboptimal pneumococcal antibody responses. Thirteen patients required pneumococcal booster vaccination following investigation. Follow-up after abnormal findings was inconsistent, with only a minority undergoing documented repeat antibody testing following intervention. No inborn error of immunity was identified within the cohort.



Abstract P120 Figure 1 Proportion of children undergoing immunological investigation following pleural empyema or necrotising pneumonia by causative organism. Investigation rates varied between organism groups, with the highest rates observed in pneumococcal disease

Conclusion This service evaluation demonstrated substantial variation in immunological investigation and follow-up following paediatric empyema and necrotising pneumonia. Despite extensive investigation in some patients, no underlying inborn error of immunity was identified, suggesting low diagnostic yield from routine screening in unselected patients. These findings support development of more targeted and standardised investigation pathways following severe paediatric respiratory infection.

P121 OUTCOMES FOLLOWING PAEDIATRIC LUNG TRANSPLANTATION FOR CHILDHOOD DIFFUSE PARENCHYMAL LUNG DISEASE IN THE UK

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Background Childhood diffuse parenchymal lung disorders (DPLD) consist of a heterogeneous group of rare disorders linked to respiratory failure and premature mortality. Current guidelines recommend consideration of lung transplantation for selected children with severe life-threatening disease; detailed outcome data are sparse. We aimed to describe clinical characteristics, pre-transplant disease severity and post-transplant outcomes in children following lung transplantation for end-stage DPLD.

Methods Retrospective cohort study of children in the UK post lung transplantation between 2015 and 2026 at Great Ormond Street Hospital and Newcastle upon Tyne Hospitals for DPLD. Demographic characteristics, underlying diagnoses, pre-transplant disease severity, lung function and post-transplant outcomes were extracted from clinical records. Continuous variables are presented as median (IQR), categorical variables as n (%).

Results Eighteen children underwent lung transplantation at a median age of 11.0 years (IQR 7.0–15.0); 10/18 (56%) were male. Underlying diagnoses were childhood interstitial lung disease (6/18, 33%), lung graft-versus-host disease (4/18, 22%), bronchiolitis obliterans (3/18, 17%), pleuroparenchymal fibroelastosis (2/18, 11%), bronchiolitis obliterans with bronchiectasis (2/18, 11%), and alveolar capillary dysplasia with misalignment of pulmonary veins (1/18, 6%). A genetic diagnosis was identified in 6/18 (33%). Pre-transplant, 14/18 (78%) required home oxygen therapy and 12/18 (67%) regular systemic corticosteroids. Median follow-up was 2.4 years (IQR 0.8-4). At the last follow-up, 13/18 (72%) were alive. CLAD was present or suspected in 4/18 (22%). No cases of post-transplant lymphoproliferative disorder were identified. Among patients with available lung function data, median FEV_1 improved from 20% predicted pre-transplant to 72% predicted (IQR 58–89) at 1-year post-transplant, and median FVC improved from 32% predicted to 76% predicted (IQR 63–90).

Conclusion Children undergoing lung transplantation for end-stage DPLD may gain a significant post-transplant improvement in terms of survival and lung function; while survival in this cohort appears below that of the overall group of paediatric lung transplant candidates, limited numbers preclude formal statistical evaluation. Data on patients referred but not listed

Abstract P121 Table 1 Clinical characteristics, pre-transplant disease severity and post-transplant outcomes

Clinical characteristics	N=18
Age at transplantation, years	11.0 (7.0–15.0)
Sex	
Female	8 (44%)
Male	10 (56%)
Underlying diagnosis	
Alveolar capillary dysplasia with misalignment of pulmonary veins	1 (5.6%)
Bronchiolitis obliterans	3 (17%)
Bronchiolitis obliterans/bronchiectasis	2 (11%)
chILD	6 (33%)
Lung GVHD	4 (22%)
PPFE	2 (11%)
Genetic diagnosis identified	
No/Not recorded	12 (67%)
Yes	6 (33%)
Pre-transplant disease severity	**N=18**
Regular systemic steroids	12 (67%)
Steroid-sparing immunosuppression	8 (44%)
Home oxygen therapy	14 (78%)
Home non-invasive ventilation	7 (39%)
Home invasive ventilation	2 (11%)
Pre-transplant FEV1, % predicted	20 (15–28)
Pre-transplant FVC, % predicted	32 (22–52)
Post-transplant outcomes	**N=18**
Years until last follow-up or death	2.4 (0.8-4)
Survival status at last follow-up	
Alive	13 (72%)
Deceased	5 (28%)
Cause of death	
CLAD	1 (20%)
Graft failure	1 (20%)
Haemophagocytic lymphohistiocytosis	1 (20%)
Multiorgan failure	1 (20%)
Sepsis with multiorgan failure	1 (20%)
Chronic lung allograft dysfunction	
No	12 (67%)
Yes/possible	4 (22%)
Unknown	2 (17%)
CLAD phenotype	
Likely restrictive fibrotic	1 (5.6%)
No	11 (61%)
Possible	1 (5.6%)
Unknown	5 (27.8%)
Post-transplant lymphoproliferative disorder	
No	15 (100%)
FEV1 at 1 year, % predicted	72 (58–89)
FVC at 1 year, % predicted	76 (63–90)

Values are presented as median (IQR) for continuous variables and n (%) for categorical variables. Missing/unknown values are shown where applicable.

were unavailable, and we have not assessed wait-list mortality in this specific group. These findings support referral to a paediatric lung transplant service as an appropriate option for children with end-stage DPLD.

P122 OPPORTUNITY FOR EX-VIVO LUNG PERFUSION TO INCREASE PAEDIATRIC LUNG TRANSPLANTATION

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10.1136/thorax-2026-BTSabstracts.283

Abstract P122 Table 1

Paediatric donors	n=152	
Paediatric donors, lungs not offered (organs did not meet standard criteria)	42 (27.6%)	
Paediatric donors, lungs offered	110 (72.4%)	
Age of lung donor offers (years, mean (sd))	10.5 (4.6)	
Cause of death (all offers)	n=119	%
– trauma including RTA	33	27.7
– infection	12	10.1
– CNS tumour	5	4.2
– drowning	7	5.9
– ICH	7	5.9
– asphyxiation	26	21.8
– other	29	24.4
Donor blood group	n=102	%
– O	38	37.3
– A	48	47.1
– B	12	11.8
– AB	4	3.9
Donor weight (kg, n, mean(sd))	n=103, 32.3 (21.2)	
Donor height (cm, n, mean(sd))	n=102, 127 (39)	
Donor arterial pO2 at time of offer§ (kPa, n, mean (sd))	n=23, 48.0 (21.6)	
Transplanted n (%)	27/110 (24.5)	
Declined n (%)	83/110 (75.5)	
– Function / history	25 (22.7)	
– No suitable recipient	49 (44.5)	
– Logistics	3 (2.7)	
– Accepted, declined at retrieval	6 (5.4)	
Declined on function/history, subsequently deemed acceptable for ex-vivo lung perfusion n (%)	Relative: 9/25 (36%) Absolute: 9/110 (8.2%)	
Waitlist patients	n=20	
Age (years, mean (sd))	7.9 (4.7)*	
Weight (kg, mean (sd))	26.7 (16.4)	
Height (cm, mean (sd))	119 (29)	
Blood group	n	%
– O	15	75
– A	3	15
– B	2	10
– AB	0	0
Diagnosis	n	%
– Cystic fibrosis	3	15
– Pulmonary hypertension	9	45
– Childhood interstitial lung disease	4	20
– Bronchiolitis obliterans	3	15
– Other	1	5
Listing status	n	%
– Super-urgent	0	0
– Urgent	5	25
– Routine	15	75
Outcome from waitlist	n	%
– Transplanted	10	50
– Died on waitlist	6	30
– Too well, suspended/removed	2	10
– Remains on waitlist	2	10

RTA, road traffic accident; ICH, intracranial haemorrhage; §invasive ventilation pressures 20/5 cmH2O in FiO2 1.0; *p<0.05, Student's t-test, donor age vs recipient age

Introduction and Objectives Ex-vivo lung perfusion (EVLP) has the potential to increase rates of lung transplantation in adults as lungs deemed 'marginal' or 'extended criteria' can be perfused and ventilated to clear secretions and inflate areas of atelectasis, making more organs suitable for transplantation.[1] To date, a single case report describes the use of this technology in children.[2] We sought to assess the unmet need and potential benefits of EVLP for children in the UK waiting for a lung transplant.

Methods Using a retrospective study design we compared a 5-year period of paediatric (age <16 years) solid organ donor offers (January 2018 to December 2022) to data from the paediatric lung transplant waitlist at Great Ormond Street Hospital for Children for the contemporaneous period. Demographic data were compared (Student's t-test). Three experts (Senior transplant co-ordinator, 2 lung transplant paediatricians) retrieved data from records of offers deemed marginal and reviewed them for suitability for EVLP.

Results Lungs were offered from 110 paediatric donors across the UK and Europe in the 5-year period, of which 27 (24.5%) were transplanted (table 1). From offers declined on history or function, 9/25 were deemed suitable for consideration of EVLP. Wait list mortality during the time period was 6/20 candidates (30%). Waitlist patients are significantly younger than donors ($p<0.05$). There was no significant difference in donor: recipient height or weight (p=NS).

Conclusions There is a disparity in access to organs suitable for transplantation for children waiting for a lung transplant. In the UK children have a longer wait time to transplant ad higher wait list mortality. Currently, paediatric donors are older than the group waiting for organs which limits availability. Approximately 2 additional transplants per year may be facilitated if EVLP use can be extended into the paediatric patient group, this represents a relative increase of national transplants by approximately 25-33%.

Thank you to the statistics team at NHSBT who provided data for this project.

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Asthma or something else?

P123 OPTIMISING DIAGNOSTIC TEST THRESHOLDS AND PATHWAYS FOR ADULT ASTHMA

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Background Accurate diagnosis of asthma is challenging. There is variation between guidelines in test-combinations and thresholds used. 2024 BTS/NICE/SIGN cost-utility analysis showed specificity of 98% with sensitivity of 68% for blood eosinophil count (BEC)/ fractional exhaled nitric oxide (FeNO)/ two-week peak flow diurnal variability (PEFv) /bronchodilator reversibility (BDR)/ bronchial challenge testing (BCT).[1] We aimed to establish the optimal test sequence and thresholds for asthma in adults based on data from the Rapid Access Diagnostics for Asthma (RADicA) study.

Methods Symptomatic adults (age 16+) with GP-suspected asthma were assessed with FeNO, spirometry, BDR, BCT and BEC performed over two visits, with PEFv between visits. All then received 4-8 weeks of inhaled corticosteroids before repeat testing. An expert panel confirmed or excluded asthma (reference standard). Individual tests with area under receiver operator curve (AUROC) above 0.75 were included into an optimal threshold pathway analysis. These individual tests then had optimal thresholds set for a specificity of at least 95%. All clinically applicable potential pathways were tested.

Results Of 270 adults, 216 had a definitive diagnostic outcome (asthma n=133, 62%; not asthma n=83, 38%). In agreement with the BTS/NICE/SIGN cost-utility analysis, BEC/BDR/BCT performed best (specificity 94%, sensitivity 82%). The best-performing pathway without BCT (FeNO/PEFv/BEC/BDR) had specificity 76%, sensitivity 64%. For analysis of optimal thresholds, PEFv had an AUROC of <0.75 and was excluded. Optimal thresholds were FeNO >60ppb, BEC >0.3, BDR >9% absolute or 10% predicted, and BCTmeth PD20<0.65mg or an intra-BCT change in FEV1 of >22%. BCT as a stand-alone test

Abstract P123 Table 1 Comparing clinically applicable asthma diagnostic pathways using individual test thresholds optimised for specificity above 95%, n=172

Pathways	Description	Test 1	Test 2	Test 3	Test4	Sensitivity	Specificity	PPV	NPV	Requiring BCT
Cutoff used: BEC: >0.3, **FeNO:** >60ppb, **Spirometry:** AFO FEV_1/FVC< lower limit of normal; **BDR:** >9% and >200ml change in FEV_1 or >10% FEV_1 % Predicted; **BCT:** PD_{20} methacholine <0.65mg or intra BCT FEV1 22% change (or BCTmann PD15<440 or intra-BCT FEV1 change 25%)										
1	All tests available (2 visits)	FeNO	BEC	BDR	BCT	**0.971**	**0.884**	0.926	0.953	54%
2	No FeNO available (2 visits)	BEC	BDR	BCT	-	**0.971**	**0.899**	0.935	0.954	56%
3	No BEC available (2 visits)	FeNO	BDR	BCT	-	**0.951**	**0.899**	0.933	0.925	60%
4	No BCT available (1 visit)	FeNO	BEC	BDR	-	**0.650**	**0.899**	0.905	0.633	NA
5	AFO before BDR (1 or 2 visits)	FeNO	BEC	AFO+ so BDR, AFO- so BCT	BCT	**0.932**	**0.928**	0.950	0.901	62%

with this positive threshold performed best (specificity 99%, sensitivity 88%). No other pathways using clinically applicable combinations of these optimised thresholds were able to match the specificity of the current BTS/NICE/SIGN pathway (see table 1).

Conclusions Our analysis supports application of currently used diagnostic thresholds for asthma tests, and of the 2024 BTS/NICE/SIGN pathway, to confirm a diagnosis of asthma in a symptomatic adult. BCT is an essential component for accurate asthma diagnosis, yet access is limited to hospital-specialists. Wider access to BCT is key to improving accuracy of asthma diagnosis.

REFERENCE

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P124 FRACTIONAL EXHALED NITRIC OXIDE AND AIRFLOW VARIATION IN ADULTS WITHOUT RESPIRATORY DISEASE: CROSS-SECTIONAL ANALYSES IN SINGAPORE AND U.S. NHANES

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Background Fractional exhaled nitric oxide (FeNO) reflects type 2 airway inflammation and is widely used in asthma. However, the significance of elevated FeNO in individuals without respiratory disease is uncertain.

Objectives To determine whether FeNO is associated with biomarkers of type 2 inflammation and airflow variation among adults without respiratory disease.

Methods We conducted cross-sectional analyses in two study populations. The exploratory study population comprised respiratory-healthy adults recruited in Singapore in the SubTypes of Airways Diseases Research Study (STARS). To evaluate whether observed associations were present at the population level, we analyzed respiratory-healthy adults from the U.S. National Health and Nutrition Examination Survey (NHANES) 2007–2012. FeNO was categorized as <25, 25–49, and ≥50 ppb. The primary outcome was below-average airflow ratio (FEV_1/FVC z-score <0). Associations between FeNO and airflow were examined using Poisson regression with robust variance, adjusting for demographic factors and blood eosinophil counts.

Measurements and Main Results STARS included 57 respiratory-healthy adults (age 30±11 years), and the NHANES sample included 3,410 respiratory-healthy adults representing approximately 60.5 million U.S. adults (age 54±10 years). In STARS, FeNO correlated moderately with blood eosinophils, serum IgE, and sputum eosinophils. Higher FeNO categories were associated with left-shift of the FEV_1/FVC z-score distributions. FeNO ≥50 ppb was associated with increased prevalence of below-average airflow ratio in both STARS (adjusted prevalence ratio [PR] 3.4, 95% CI 1.5–7.7) and NHANES (adjusted PR 1.20, 95% CI 1.06–1.37), independently of blood eosinophil count.

Conclusions Among adults without respiratory disease, elevated FeNO is associated with other markers of type 2 inflammation and with subtle reductions in airflow ratio.

P125 SYMPTOM PROFILES AND DIAGNOSTIC PATTERNS IN A MULTI-MORBID RESPIRATORY COHORT: FINDINGS FROM THE SMART COHORT

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Introduction Respiratory patients frequently present with multiple concurrent diagnoses, and the relationship between presenting symptoms and final diagnoses in this context is poorly characterised. This study describes symptom and diagnosis associations and diagnostic co-occurrence patterns in a prospectively recruited respiratory cohort.

Methods 220 patients from the SMART cohort were included. Ten binary symptoms were recorded at presentation and analysed against eight respiratory diagnoses. Multimorbidity was characterised by describing the distribution of concurrent diagnoses per patient. Pairwise symptom-diagnosis associations were quantified using phi coefficients and Fisher's exact test with Benjamini-Hochberg FDR correction. Logistic regression and a supplementary multilabel analysis using LASSO regularised regression and multilabel Random Forest were performed to describe independent symptom-diagnosis relationships.

Results Multimorbidity was common, with 85% of patients carrying two or more concurrent diagnoses and 25.9% carrying three or more. Asthma (60.5%) and Allergic Rhinitis (49.5%) were the most prevalent diagnoses. Wheeze was the most strongly associated symptom with Asthma (OR 6.07, 95% CI 2.60–15.2, FDR-corrected p=0.002). Upper airway symptoms including blocked nose, runny nose, and post-nasal drip clustered together and were associated with rhinitis diagnoses. Cough and chest pain were associated with GORD. Recurrent infections and runny nose were more strongly associated with Non-Allergic than Allergic Rhinitis. Multilabel analyses identified distinct symptom signatures per diagnosis that were consistent across methods. Unadjusted and adjusted analyses differed for several symptom-diagnosis pairs, reflecting the influence of diagnostic co-occurrence on pairwise associations.

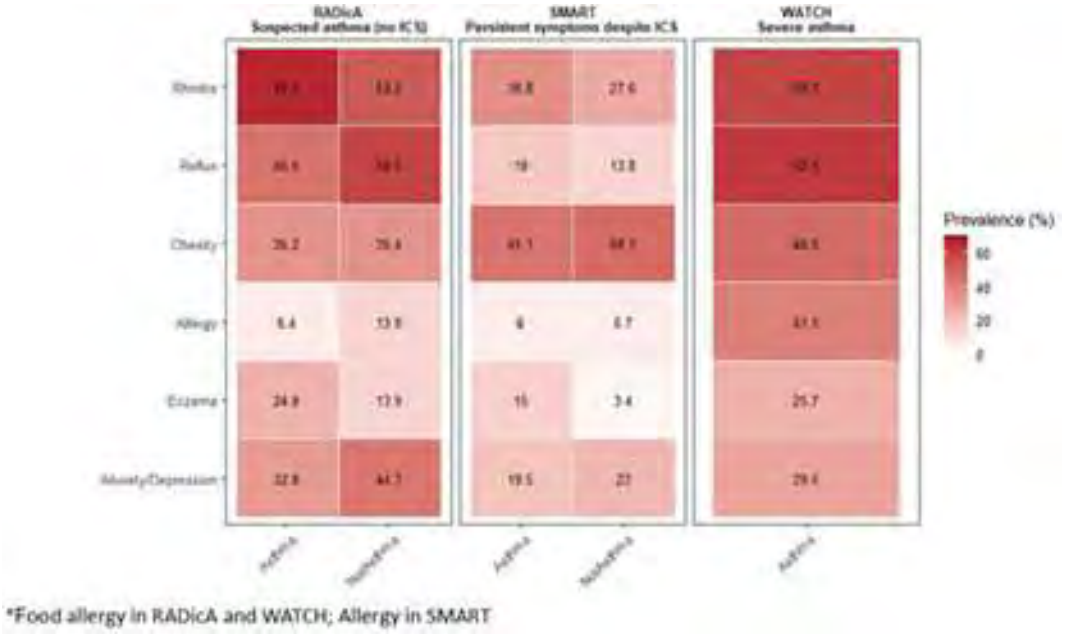
Conclusions Multimorbidity is the norm rather than the exception in this respiratory cohort. Distinct symptom profiles are nonetheless identifiable for common respiratory diagnoses, with consistent findings across multiple analytical approaches. These descriptive findings provide a foundation for future work developing symptom-guided investigation pathways.

P126 MULTIMORBIDITY CLUSTERS AND ASSOCIATED CHARACTERISTICS IN MILD, MODERATE AND SEVERE ASTHMA COHORTS

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10.1136/thorax-2026-BTSabstracts.287

Background The phenotypic nature of multimorbidity in asthma across all disease severities is poorly understood. This study



Abstract P126 Figure 1 Prevalence of comorbidities across three cohorts

aimed to describe multimorbidity phenotype clusters, characterise their features, and assess their impact on asthma control in mild, moderate and severe asthma cohorts.

Methods We combined data from three international observational cohorts: (1) the Rapid Access Diagnostics for Asthma (RADicA) study (Manchester, UK), recruiting symptomatic steroid-naïve adults with GP-suspected asthma, (2) the SMART study (Dublin, Ireland), including patients with clinician-diagnosed asthma who remained uncontrolled despite inhaled corticosteroids; and (3) the Wessex Asthma Cohort of Difficult Asthma (WATCH; Southampton, UK), comprising severe asthma. Comorbidities were identified from self-report, medical records or medication history. Cluster analyses were performed to identify multimorbidity phenotypes. Associations between cluster membership and asthma control (Asthma Control Questionnaire [ACQ]) were evaluated.

Results Data from 925 adults were included in the analysis: 204 from the RADicA study (61% had asthma), 220 from SMART (60% subsequently confirmed as asthma), and 501 from WATCH. Comorbidities were common even in mild treatment-naïve mild asthmatic patients (66% had at least one comorbidity), and were more prevalent in the uncontrolled (83%) and severe asthma cohorts (98%). Reflux, obesity, rhinitis and anxiety were consistently prevalent across all three cohorts (figure 1). Four multimorbidity clusters were identified in each cohort, with broadly similar patterns. The presence of these comorbidity clusters was associated with asthma control, although the magnitude of these effects varied between cohorts. In mild asthma, membership of the obesity/reflux cluster was associated with lower rates of good asthma control (ACQ<0.75) after inhaled corticosteroid treatment (42.2% vs 64.8%; p=0.020). In severe asthma, associations between the obesity/reflux (p=0.076) clusters and asthma control were less pronounced, with 89% of patients reporting sub-optimal control.

Conclusion Multimorbidity is prevalent across all asthma severities and is associated with worse subjective asthma control. The weaker associations observed in severe asthma may reflect the high baseline prevalence of poor control in this population. Addressing multi-morbidities across all severities may improve outcomes beyond a sole focus on severe asthma. Further research is needed to better elucidate the underlying mechanisms of these associations.

P127 BEYOND THE WHEEZE: A PHENOTYPE-DRIVEN APPROACH TO ASTHMA EXACERBATIONS

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10.1136/thorax-2026-BTSabstracts.288

Background Asthma exacerbations are associated with significant morbidity and mortality. Current management is usually corticosteroids regardless of the underlying inflammatory phenotype. We aimed to characterise asthma exacerbations using physiological and inflammatory measures and to determine whether exacerbation characteristics relate to baseline disease phenotype.

Methods In this single-centre, prospective longitudinal study, participants with asthma were followed for up to three years. Detailed phenotyping was performed at baseline and repeated when participants experienced worsening symptoms sufficient to seek medical attention. Events were classified as asthma exacerbations (AE), possible exacerbations (PE), or non-exacerbation events (NE). Assessments included but were not limited to: demographic data, including an asthma history, spirometry, airway oscillometry, blood eosinophil count, induced sputum, fractional exhaled nitric oxide (FeNO), viral PCR, and nasal brush sampling for transcriptomic analysis.

Abstract P127 Table 1 Key Physiological and inflammatory data collected in the APEX study at baseline and exacerbation

	Baseline	T2-high Asthma Exacerbation	p-value	Baseline	T2-low Asthma Exacerbation	p-value
N%	23(52)			21(48)		
R5 ($cmH_20/L/s$)	4.2(3.1-5.8)	5.7(4.6-6.9)	0.03	3.7(3.0-5.4)	5.7(3.4-4.6)	0.04
R20 ($cmH_20/L/s$)	2.9(2.5-3.8)	3.8(2.8-4.2)	0.21	3.2(2.9-4.2)	3.8(2.7-3.0)	0.6
R5-20 ($cmH_20/L/s$)	1.0(0.4-2.1)	2.4(1.4-2.9)	0.02	0.6(0.2-1.2)	1.2(0.3-2.7)	0.04
Ax ($cmH_20/L/s$)	12.8(5.0-33.3)	39.8(4.8-72.4)	0.006	9.8(2.5-21.7)	27.8(2.6-53.8)	0.03
FEV_1% predicted	73.5(21.8)	64.1(23.0)	<0.001	84.1(18.3)	75.2(18.0)	<0.001
FeNO (ppb)	55.1(35.6)	78.5(68.7)	0.03	25.1(18.8)	21.5(10.5)	0.46
Eosinophil count ($x10^9/L$)	0.4 (0.21)	0.6 (0.31)	0.04	0.17(0.1)	0.11(0.82)	0.07
ACQ7	1.4 (0.8)	3.5 (0.8)	<0.001	1.5(0.7)	3.1(0.8)	<0.001

Data stratified according to Type 2 inflammatory status at exacerbation compared with paired baseline data. R5 = Resistance at 5Hz, R20 = Resistance at 20 Hz, R5-20= Peripheral resistance Ax= Area of Reactance, FEV_1= Forced expiratory volume in 1 second, FeNO = Exhaled Nitric Oxide, ACQ = Asthma Control Questionnaire score.

Results A total of 305 participants were recruited. At baseline, over 65% were classified as type 2 (T2)-low. T2-high participants had worse lung function (FEV_1 % predicted 75.8% vs 87.7%; FEV_1/FVC 69.0% vs 76.0%; both $p<0.001$), and greater peripheral airway resistance (R5–R20 1.16 vs 0.84 cmH_20/L/s, $p=0.01$).

Baseline T2 biomarkers were significantly higher in participants who subsequently experienced an AE compared with those who did not (FeNO 29.5 vs 17.0 ppb, $p<0.001$; blood eosinophils 0.23 vs 0.14 $\times10^9$/L, $p=0.04$). Furthermore, 43.2% of the AE group were T2-high at baseline compared with 23.0% of the NE group.

44 unique AEs and 38 unique PEs were observed. Respiratory viruses were detected in 63% of AEs. Although both AE and PE events were associated with similar deterioration in asthma control, only AEs demonstrated significant increases in R5–R20 compared with the respective baseline data (0.55 vs 2.15 cmH_20/L/s $p<0.001$). Most AEs (72%) were T2-high. Both T2 high and low exacerbations had similar loss in FEV1, but T2-high exacerbations were associated with greater peripheral resistance. T2 status remained stable between baseline and exacerbation.

Conclusions Asthma exacerbations demonstrate substantial inflammatory and physiological heterogeneity. Biomarker-driven assessments are important to develop a personalised approach towards asthma exacerbations. Airway oscillometry may provide additional value in distinguishing asthma exacerbations from loss of asthma control.

P128 LONGER ASTHMA DURATION IS ASSOCIATED WITH ELEVATED NON-T2 SPUTUM BIOMARKERS AND REDUCED T2 INFLAMMATION IN SEVERE ASTHMA

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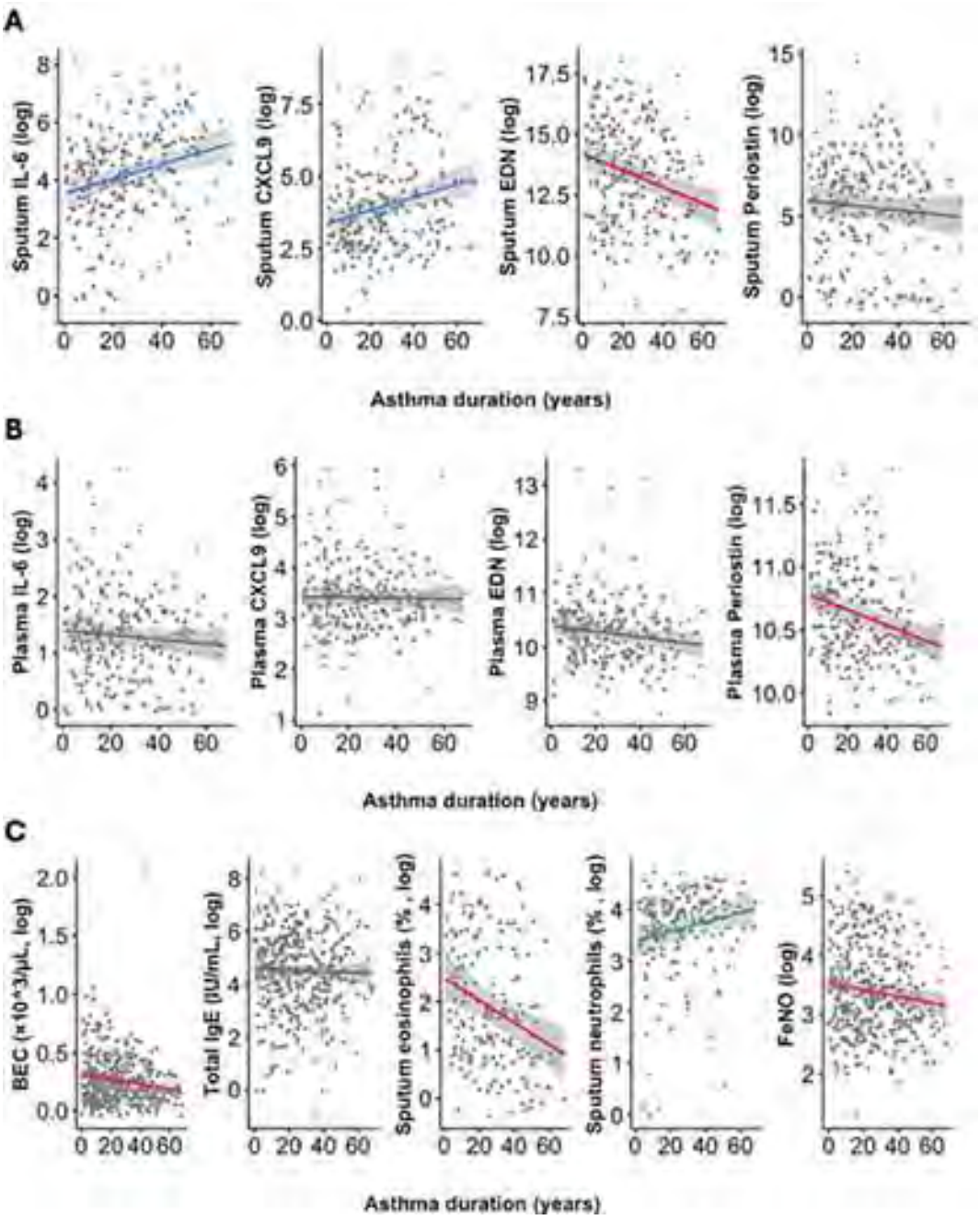
Background Longer asthma duration predicts non-remission in Type-2 (T2) biologic-treated severe asthma, but underlying mechanisms remain unclear. We investigated associations between asthma duration and inflammatory biomarkers that may explain differential biologic response.

Methods We analysed cross-sectional data from adults with severe asthma in U-BIOPRED. Asthma duration was defined as years from diagnosis to study entry. T2 and non-T2 biomarkers were measured in blood and sputum. Identical assays were performed in PRISM, a validation cohort of biologic-initiators. Multivariable regression models adjusted for age, sex, ethnicity, BMI, smoking, and oral corticosteroid dose. Associations between duration-related biomarkers and 12-month biologic remission were explored in PRISM.

Results In U-BIOPRED (n=411), median asthma duration was 23 years (IQR 12-38). Longer duration was associated with higher non-T2 biomarkers including sputum CXCL9 (β=0.024, 95%CI 0.011-0.038), sputum IL-6 (β=0.024, 95%CI 0.011-0.037), and sputum neutrophils (β=0.009, 95%CI 0.001-0.017), but lower T2 biomarkers including plasma periostin (β=-0.006, 95%CI -0.009 to -0.003), eosinophils (blood: β=-0.002, 95%CI -0.003 to -0.001; sputum: β=-0.023, 95%CI -0.035 to -0.011), sputum EDN (β=-0.032, 95%CI -0.049 to -0.014) and FeNO (β=-0.006, 95%CI -0.010 to -0.001).

PRISM (n=474) confirmed these associations. Higher sputum CXCL9 was associated with reduced remission in anti-IL-4Rα-treated patients with asthma duration ≥20 years (β=-1.73, 95% CI -3.180 to -0.276).

Conclusion Longer asthma duration was associated with elevated airway non-T2 inflammation and reduced systemic and airway T2 inflammation. This highlights the importance of airway biomarker sampling and suggest combined T2 and non-T2 therapeutic strategies may be needed to achieve remission in long-standing asthma.



Abstract P128 Figure 1 Multivariable linear regression of asthma duration against biomarker levels, adjusted for age, sex, race, smoking status, BMI and daily prednisolone dose. Regression lines are colour-coded by phenotype and significance: red = significant T2 biomarkers; blue = significant non-T2 biomarkers; grey= non-significant. **(A) Induced sputum**: longer duration was associated with lower T2 biomarkers (EDN) and higher non-T2 biomarkers (IL-6, CXCL9). **(B) Plasma**: longer duration was associated with lower T2 biomarker (periostin). **(C) Current clinical biomarkers**: longer duration was associated with lower T2 biomarkers (blood and sputum eosinophils, FeNO) and higher non-T2 biomarker (sputum neutrophils)

P129 EARLY IDENTIFICATION OF 'HIGH-RISK' ASTHMA AND PROGRESSION TO BIO-ELIGIBILITY: A RETROSPECTIVE COHORT STUDY USING UK DATA

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10.1136/thorax-2026-BTSabstracts.290

Background Biologic therapies have transformed severe asthma management, prompting debate around earlier intervention in disease trajectory. The ability to identify patients likely to progress to bio-eligibility access criteria is required.

Methods Retrospective cohort study using UK primary care data. Adults with asthma at varying baseline exacerbation frequencies (first ever exacerbation, second exacerbation, two exacerbations/two years and three exacerbations/three years) followed for five years. Primary outcome was bio-eligibility,

defined as ≥3 exacerbations within 12 months; supplementary analysis defined bio-eligibility as ≥2 exacerbations in 12 months. Prognostic models using LASSO logistic regression identified 'high-risk' patients (highest 10% of predicted risk). For each cohort selection scenario, minimum cumulative oral corticosteroid (OCS) dose was estimated.

Results Bio-eligibility attainment varied across cohort selection scenarios with proportions ranging from 13.9% (2996/21490) for the first exacerbation cohort, to 37.7% (3210/8519) for the three exacerbations in three years cohort ('high-risk cohort' 26.3% (565/2149) - 61.4% (523/852)). Sensitivity analysis using lower exacerbation thresholds increased bio-eligibility proportions across scenarios. Cumulative OCS exposure was substantial irrespective of bio-eligibility status (e.g. 16.3% (731/4480) of the two exacerbations/two years cohort who had ≥4 exacerbations in five years, did not reach bio-eligibility, with minimum 1200mg cumulative prednisolone exposure).

Discussion Progression to bio-eligibility based on exacerbation frequency is heterogenous, even amongst 'high-risk' patients. However, patients had substantial ongoing OCS exposure regardless of bio-eligibility attainment. Cumulative OCS dose may be a more effective bio-eligibility criterion in severe asthma and further investigation of the economic impact and effect on OCS-associated morbidity using this access criterion are required.

P130 DIGITALLY ASSESSED HOME FEV1 AND INHALER ADHERENCE TO IDENTIFY SEVERE ASTHMA AS A CAUSE OF POORLY CONTROLLED ASTHMA

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10.1136/thorax-2026-BTSabstracts.291

Introduction Identifying severe asthma and distinguishing it from patients with other causes of uncontrolled asthma can be challenging. Home monitoring of variance in lung function alongside presence of exacerbations despite good adherence may be a better an efficient way to identify severe asthma.

Hypothesis A statistical model derived from digital monitoring data (lung function and inhaler adherence), incorporating T2 inflammation, may better identify severe asthma than traditional tools.

Methods The Astec score was developed in two cohorts, INCASun study,[1] and a healthy control population. The model was tested in an independent validation cohort of patients recruited to the Traits study (NCT05357274). Full inclusion and exclusion criteria and study protocol have been published prior.[2]

Results Of the 220 patients who completed the Traits study, 133 had asthma. 112 had asthma as the primary cause of their symptoms (A), 21 had asthma but with prominent treatable traits causing their symptoms (TT), and 87 did not have asthma (NA). Those in A had a median Astec of 3.8, while TT had a median score of 1.55 and NA had a score of 1.67 (both $p<0.01$).

Fifteensubjectsultimately requireda biologic.Astec scores were 3.2 points higher in those whowere prescribedbiologic therapy (95% CI 1.9 to 4.3, $p<0.001$). A one unit increase in Astec score was associated with a 32% increased chance of beingcommencedon a biologic (OR 1.32, 95%CI 1.13-1.54, $p<0.001$).53remained on high dose inhaled corticosteroid therapy.A one unit increase in Astec score was associated with a 55% chance of finishing the study on a high dose inhaled corticosteroids (ICS) (OR 1.55, 95% CI 1.35-1.82, $p<0.001$).

Discussion/Conclusion The Astec score distinguishes those with asthma with treatable traits from those who require high dose ICS or biologic therapy. This represents an efficient and scalable alternative to traditional methods of classifying asthma.

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P131 SARCOPENIA IN ASTHMA: A CROSS-SECTIONAL ASSESSMENT OF MUSCLE STRENGTH, MASS AND PHYSICAL PERFORMANCE

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Background Sarcopenia is a potentially important extrapulmonary manifestation of asthma, yet evidence regarding skeletal muscle health remains limited. We evaluated the three key domains of sarcopenia – muscle strength, muscle mass, and physical performance – in adults with asthma and explored the influence of sex and disease severity.

Methods Adults with physician-diagnosed asthma (n=70) and healthy age and sex-matched controls (n=39) participated in this cross-sectional study. Handgrip strength, quadriceps muscle strength, appendicular skeletal muscle mass adjusted for body mass (ASM/BMI), and 4-m gait speed were assessed. Between-group comparisons were performed using independent t-tests, with stratified analyses according to sex and asthma severity.

Results Compared with controls, adults with asthma showed a trend for lower muscle strength: handgrip strength (40 vs 36 kg; $p=0.08$) and quadriceps strength (47 vs 41 kg; $p=0.05$). Sex-stratified and severity analyses showed significant reductions in handgrip strength (27 vs 34 kg; $p=0.02$) and quadriceps strength (32 vs 39 kg; $p=0.008$) among females but not males with severe disease. Muscle mass was lower in adults with asthma by 13% (ASM/BMI: 0.81 vs 0.93; $p=0.007$), both in females (0.67 vs 0.78, $p=0.0001$) and males (1.01 vs 1.13, $p=0.02$), and exacerbated by severe disease (female $p<0.0001$; male $p=0.006$). Physical performance was impaired in asthma (1.48 vs 1.73 $m \cdot s^{-1}$, $p<0.0001$), in females but not males with severe disease. Collectively the prevalence of sarcopenia was indicated in 24% of middle-aged adults with asthma.

Conclusion Around one in four middle-aged adults with asthma exhibit impairments across key domains of sarcopenia, including muscle strength, muscle mass, and physical performance, with differences influenced by sex and disease severity. These findings suggest that sarcopenia may represent a clini-

cally relevant extrapulmonary treatable trait in people with asthma that warrants further investigation.

P132 BLOOD EOSINOPHIL COUNT IS ASSOCIATED WITH TREATMENT RESPONSE TO ORAL CORTICOSTEROIDS FOR ASTHMA ATTACKS

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10.1136/thorax-2026-BTSabstracts.293

Background Asthma attacks are commonly treated with oral corticosteroids (OCS), antibiotics, or both, despite underlying inflammatory heterogeneity. In COPD, blood eosinophil-guided OCS treatment results in better outcomes. We examined whether higher pre-attack blood eosinophil counts (BEC) are associated with lower risk of treatment failure after OCS-treated asthma attacks.

Methods We used two independent datasets: a longitudinal UK primary-care cohort (OPCRD, n=61,395) and 22 randomised controlled trials (ORACLE2, n=793). The primary outcome was treatment failure within 14, 28, and 90 days following the index attack. Multivariable logistic regression models were used to evaluate associations between BEC and treatment failure.

Results In OPCRD-patients treated with acute OCS, higher BEC was associated with lower odds of treatment failure at 14 days (OR 0.87, 95% CI 0.85–0.89) and 28 days (OR 0.91, 95% CI 0.89–0.93), but not 90 days (OR 0.98, 95% CI 0.96–1.00) (figure 1). Similar associations were observed in ORACLE2 at 14 days (OR 0.65, 95% CI 0.44–0.95), but not at 28 (OR 0.95, 95% CI 0.79–1.16) or 90 days (OR 1.11, 95% CI 0.98–1.25). The association was observed irrespective of additional antibiotic treatment, but not when treated with antibiotics alone.

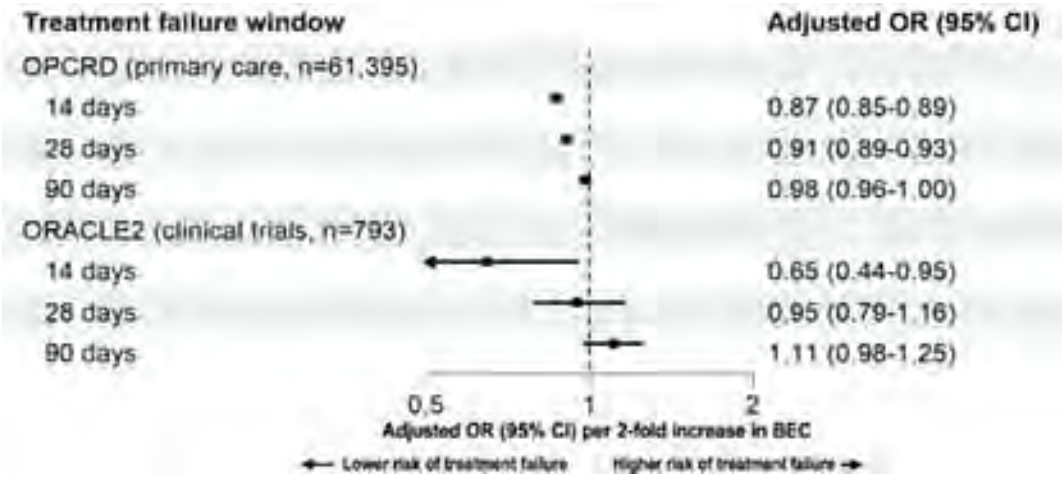
Conclusion In both real-world and randomised trial cohorts, higher pre-attack BEC were associated with lower risk of early treatment failure after OCS-treated asthma attacks. These findings support trialling acute biomarker-directed OCS treatment.

P133 WHEN TO SEND IN THE TROOPS - OPTIMAL TIMING FOR BREATHING PATTERN DISORDER ASSESSMENT IN SEVERE ASTHMA

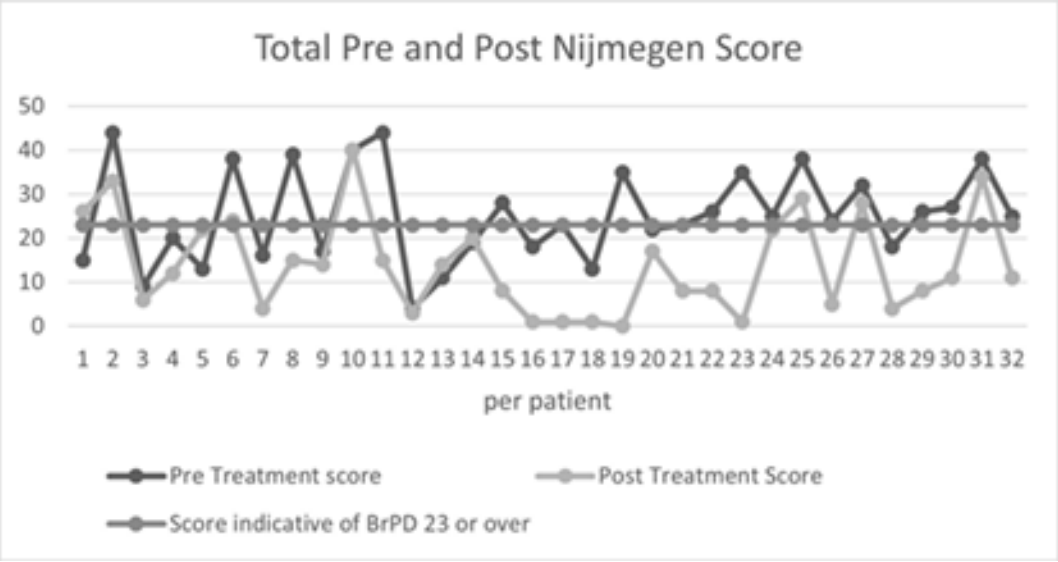
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10.1136/thorax-2026-BTSabstracts.294

Introduction In our regional severe asthma service, we observed a high number of symptoms suggestive of Breathing Pattern Disorder (BrPD) among patients commencing biologic therapy. This correlated with previously reported rates of up to 47% (Kurukulaaratchy, 2025). Given limited respiratory physiotherapy input we aimed to identify the optimal point within the patient pathway to identify BrPD.



Abstract P132 Figure 1 Association between continuous blood eosinophil counts and treatment failure following oral corticosteroid-treated asthma attacks in OPCRD and ORACLE2 cohorts. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for treatment failure at 14, 28, and 90 days following initial OCS prescription in the OPCRD and ORACLE2 cohorts. ORs represent the change in odds of treatment failure per 2-fold increase in blood eosinophil count. Multivariable logistic regression models were applied, adjusted for age, sex, BMI, and number of prior-year attacks. Values <1 indicate lower odds of treatment failure



Abstract P133 Figure 1

Methods From January 2026 all patients commencing biologic therapy for severe asthma were asked to complete a Nijmegen Questionnaire (NQ). This is the only validated symptom score for BrPD. The questionnaire was completed at biologic treatment commencement. Scores were repeated after 3 months of treatment, via paper, online, or telephone, enabling screening without altering treatment pathways. 32 patients were screened over 8 weeks.

Results A significant proportion of the patients with a raised baseline NQ score showed a clinically significant decrease. The number of patients scoring over 23 (threshold for BrPD) dropped from 59% to 21% (figure 1).

The NQ considers multiple domains for BrPD. Comparisons were carried out for clinically relevant domains for BrPD. There was a significant improvement in shortness of breath, chest tightness, the need to deep breath, and frequency of sigh ($p<0.001$) between pre and post treatment in the patients who had initial scores indicating BrPD.

Conclusions The significant change in NQ total score and scoring domains indicates uncontrolled asthma that is not fully optimised may increase the score on the NQ and be interpreted as BrPD.

This evidence suggests that the assessment of BrPD, should preferably be carried post optimisation of severe asthma. Especially where screening for BrPD is carried out using a questionnaire-based approach. Further research is needed in this area to understand the impact of asthma stabilisation on BrPD, and the optimal time for physiotherapy assessment and treatment. This would reduce the impact on stretched physiotherapy resources.

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P134 LARYNGEAL DYSFUNCTION IN A DISTRICT GENERAL HOSPITAL ASTHMA CLINIC: A SERVICE NEEDS ASSESSMENT IN AN URBAN POPULATION

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Introduction Laryngeal dysfunction (LD), including inducible laryngeal obstruction (ILO), is increasingly recognised as a treatable trait contributing to difficult-to-control asthma symptoms and healthcare utilisation. However, its prevalence remains poorly defined and access to diagnostic confirmation varies considerably by region. This study aimed to quantify the prevalence of suspected LD, symptom burden, and referral patterns for laryngeal assessment in patients attending a DGH asthma clinic serving a socioeconomically deprived urban population.

Methods We conducted a retrospective cross-sectional review of 119 consecutive patients attending a general asthma clinic at a DGH between June 2025 and June 2026. Clinician suspicion of LD and Pittsburgh Vocal Cord Dysfunction Index (PVCD) scores were extracted. Additional data included referral for provocation laryngoscopy, emergency department (ED) attendance, hospital admissions, oral corticosteroid (OCS) use, and history of intensive care unit (ICU) admission and intubation.

Results LD was suspected clinically in 64/119 patients (54%). PVCD scores were available for 114 patients; 60% scored ≥4 and 24% scored ≥9, indicating a substantial symptom burden consistent with possible LD. The cohort demonstrated high healthcare utilisation: 50% had at least one ED attendance and 25% required hospital admission in the preceding year. The median number of oral corticosteroid courses was 1.0 (IQR 0.0–3.0) per patient over 12 months, with 41/117 (35%) requiring 3 or more courses. Seven patients had previous ICU admissions, including four who had been intubated. Despite this burden, only 30% of patients were referred for provocation laryngoscopy.

Conclusion In a DGH asthma clinic serving a deprived, ethnically diverse urban population, LD is commonly suspected and supported by high screening scores, yet formal diagnostic laryngoscopy is underutilised. The discrepancy between clinical suspicion and diagnostic confirmation highlights a significant unmet service need. These findings support the development of integrated ILO diagnostic pathways within non-specialist asthma services to improve diagnostic accuracy and reduce misattribution of symptoms to asthma in patients whose symptoms may be driven primarily by laryngeal dysfunction.

P135 SOUND AND VISION: DO VIDEO-RECORDINGS WITH SOUND IMPACT DIAGNOSTIC ACCURACY AND INTER-RATER AGREEMENT OF A DIAGNOSIS OF INDUCIBLE LARYNGEAL OBSTRUCTION?

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Introduction Inducible laryngeal obstruction (ILO) is an inappropriate narrowing of the larynx during inspiration, with an accepted visual benchmark being ≥50% inspiratory laryngeal closure during provocation laryngoscopy. Previous research found that inter-rater agreement of ILO diagnosis from review of video recordings of provocation laryngoscopies varied from 'slight-to-moderate' (Walsted *et al.*, 2017) to 'fair' for silent, longer video durations. Further studies that include sound were recommended (Wong *et al.*, 2026).

Objectives To compare rates of accuracy and agreement for diagnosing ILO between expert speech & language therapy (SLT) raters with and without sound data, investigating the hypothesis that sound will improve both the accuracy of diagnosis and inter-rater reliability.

Methods Electronic records were retrospectively reviewed for 100 patients between February 2025-January 2026 referred to our Tertiary-level upper airway service for assessment. Video-recordings of the patients' larynx during provocation laryngoscopy were subsequently blindly rated by three SLTs. Recordings were rated for presence or absence of ILO and compared with the diagnosis given at assessment. Videos were viewed in a randomised order, initially without sound and later, with sound.

Results Of the 100 patients reviewed 80% were female, 20% male, median age was 53 (IQR 29.5), and asthma diagnosis was confirmed in 71%. Eighty-eight were referred for ILO assessment, of whom 27 (31%) received a confirmed ILO diagnosis following provocation laryngoscopy.

Inter-rater reliability (Fleiss' Kappa) among the three SLTs was 'slight' when evaluating laryngoscopy videos without sound ($\kappa = 0.187$). Reliability improved to 'moderate' agreement when sound was included ($\kappa = 0.508$).

When ratings were compared to the confirmed ILO diagnosis, average sensitivity (true positive) with and without sound was 88.6% and 86.3% respectively. Average specificity (true negative) was 80.2% with sound, compared to 58.7% without sound.

Conclusions Sound capture can enhance the diagnostic assessment of ILO. The addition of sound increased inter-rater reliability from slight to moderate agreement. Whilst ratings demonstrated high sensitivity without sound, the addition of sound substantially improved specificity, helping to identify non-ILO presentations.

Standardising diagnostic laryngoscopy protocols, and including sound, may improve diagnostic accuracy, reduce clinical misclassification, and support clinical decision-making.

P136 IMPLEMENTATION OF A PROVOCATION LARYNGOSCOPY SERVICE WITHIN A DIFFICULT ASTHMA CLINIC: A SERVICE EVALUATION AT ONE YEAR

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Introduction Inducible laryngeal obstruction (ILO) is a treatable trait identified in up to 50% of patients with severe asthma,

Abstract P136 Table 1 Baseline characteristics and laryngoscopy findings for patients undergoing provocation laryngoscopy within a severe asthma service (N = 81)

Characteristic	N (%) or Median (IQR)
Baseline Characteristics (N = 81)	
Age, years	47 (38–59)
Female sex	64 (79%)
Smoking status*	
Never	49 (72%)
Ex-smoker	14 (21%)
Current smoker	5 (7%)
Comorbidities	
Gastro-oesophageal reflux	69 (85%)
Dysfunctional breathing	47 (58%)
Allergic rhinitis	55 (68%)
Nasal polyps	3 (4%)
Prior intubation	11 (14%)
Biologic therapy at baseline	31 (38%)
Benralizumab	12
Tezepelumab	11
Mepolizumab	4
Dupilumab	4
Omalizumab	2
Maintenance OCS at baseline†	20 (25%)
Median dose, mg/day‡	10 (5–20)
Rescue prednisolone courses, prior 12 months	5.0 (3.0–7.0)
ED presentations, prior 12 months	1.0 (0.0–2.0)
Hospital admissions, prior 12 months	1.0 (0.0–2.0)
Laryngoscopy Findings (N = 75 with a diagnostic result)	
ILO identified (any)	65 (87%)
Diagnosis	
ILO	60 (80%)
ILO + central hypersensitisation	4 (5%)
ILO + EDAC	1 (1%)
Central hypersensitisation only	8 (11%)
EDAC only	1 (1%)
?BPD, no ILO	1 (1%)
Procedure incomplete§	6 (7%)

Table legend
Data are presented as median (IQR) for continuous variables and N (%) for categorical variables.
*Smoking status data available for 68/81 patients; percentages are of those with data recorded.
†Includes one patient receiving physiological hydrocortisone replacement for adrenal insufficiency (dose 10/5/5 mg). Dose analysis excludes this patient (n = 19).
‡Numeric therapeutic doses available for 19/20 mOCS patients.
§Procedure incomplete due to inability to tolerate laryngoscopy (n = 3) or inability to replicate breathlessness at the time of procedure (n = 3).
Two additional patients had a documented history of nasal polypectomy but no active polyps at the time of laryngoscopy; these are not included in the nasal polyps count.
Abbreviations: IQR, interquartile range; ILO, inducible laryngeal obstruction; OCS, oral corticosteroids; ED, emergency department; EDAC, excessive dynamic airway collapse; BPD, breathing pattern disorder.

yet diagnostic laryngoscopy access remains limited. We report the implementation and first-year outcomes of a provocation laryngoscopy service embedded within our tertiary referral difficult asthma clinic at an inner city hospital serving a diverse urban population.

Methods retrospective service evaluation of all patients referred from the difficult asthma service who underwent provocation laryngoscopy (January 2025–January 2026). Data were collected from electronic clinical records. Baseline characteristics were recorded at the time of laryngoscopy. Follow-up data were obtained at routine clinical review. Only patients with follow up data ≥9 months post laryngoscopy were included in outcome analysis. Statistical comparisons were performed using the Wilcoxon signed-rank test.

Results 81 patients underwent provocation laryngoscopy; 75 received a diagnostic result. ILO was identified in 65 (87%). The cohort was predominantly female (79%), median age 47 years (IQR 38–59). There was a high prevalence of comorbidities: gastro-oesophageal reflux 85%, allergic rhinitis 68%, dysfunctional breathing 58%; 14% had a history of prior intubation. 38% were on biologic therapy and 25% on mOCS at baseline (table 1).

22 patients were identified with follow-up at least 9 months post laryngoscopy (median 12.0 months, IQR 10.4–12.0); 7/22 (32%) had biologic changes during follow-up, these were retained as a cohort characteristic. Prednisolone courses fell from a median of 5.0 (IQR 4.0–8.0) to 3.0 (IQR 1.5–4.0) per year (n=19, p=0.006). The proportion of patients with at least one hospital admission fell from 48% to 29% (10/21 vs 6/21).

Conclusion Embedding provocation laryngoscopy in a difficult asthma service identified ILO in 87% of a pre-selected cohort with high comorbidity burden, prior intubation rates and mOCS use. At follow up, significant reductions in rescue prednisolone courses and hospital admissions were observed. These preliminary findings support ILO identification as a clinically meaningful treatable trait in difficult asthma and supports the case for prospective evaluation of ILO-directed treatment within difficult asthma services.

Abstract P137 Table 1 The demographic characteristics, comorbidities, symptoms, investigation findings and site/extent of large airway collapse in a cohort of EDAC patients

	N=20	
Demographics		
Age (years, mean, SD)	47 (13.9)	
Female (%)	19 (95%)	
BMI (median, IQR)	33.5 (9.25)	
Smoking status (current/ex/never)	0 / 2 (10%) / 18 (90%)	
Comorbidities		
Asthma	17 (85%)	
OSA	5 (25%)	
ILO	5 (25%)	
GORD	12 (60%)	
COVID preceding symptoms Yes Hospitalised	 10 (50%) 4 (20%)	
Symptoms		
Breathlessness	17 (85%)	
Cough: Productive With 'barking' character	14 (70%) 5 (25%) 3 (15%)	
Wheeze	8 (40%)	
Multiple exacerbations	8 (40%)	
Dysphagia	4 (20%)	
Investigations		
FEV1 % predicted (median, IQR)	91 (13.8)	
VCmax % predicted (median, IQR)	100 (24.0)	
FEV1/FVC (%) (median, IQR)	82 (11.2)	
Airflow instability on flow volume loop	14 (70%)	
Airflow instability on CPET (n=18)	13 (72%)	
Site and extent of airway collapse	**Extent of collapse**	
Site	**50-79%**	**80%+**
Trachea or main bronchi	20	14
Trachea only	9	9
Main bronchi only	4	11

P137 EXCESSIVE DYNAMIC AIRWAYS COLLAPSE (EDAC): AN UNDER-RECOGNISED CAUSE OF SYMPTOMS IN THE SEVERE ASTHMA CLINIC?

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10.1136/thorax-2026-BTSabstracts.298

Background EDAC is a condition characterized by pathological narrowing or collapse of the large central airways (trachea and main bronchi) during expiration, usually by 50% or more.[1] Here we describe the clinical features, investigation findings and response to treatment in the first 20 patients in our rapidly expanding EDAC cohort originally referred to our service with suspected worsening asthma.

Methods Data on patients identified with EDAC from asthma clinics is being collected as part of an ongoing Clinical Service Evaluation (Trust audit ID number 25-327C). The diagnosis of EDAC was confirmed by direct endoscopic visualisation of >50% trachea/main bronchi collapse on expiration. These data come from the first 20 patients on the database with completed data fields who presented to asthma clinics with worsening symptoms between Nov' 23 and March '25 (16 months).

Results Our cohort were almost exclusively female (19/20) with a mean age of 47 and most were overweight/obese (n=19/20, median BMI 33.5). 17/20 had asthma diagnoses though 19/20 had normal spirometry (median FEV1 predicted 91%; VCmax predicted 100%). The most common symptoms were exertional breathlessness and cough though some patients had wheeze or multiple exacerbations. 14/20 had evidence of airflow instability on flow volume loops, which was more prominent on cardio-pulmonary exercise tests (CPETs). All 20 patients had >50% large airway collapse, of which 14/20 had >80% collapse. 15 patients had trials of CPAP, of whom 13 reported symptomatic improvement (6 in dyspnoea, 2 in cough, 5 improved sleep quality).

Conclusion In obese patients with asthma diagnoses, symptoms of breathlessness and/or cough with preserved spirometry there should be a high index of suspicion for EDAC. Airflow instability, commonly seen on flow volume loops but more evident on CPET may support the diagnosis which should be confirmed on endoscopy. CPAP appeared to be an effective treatment where tolerated. Further work is underway at our centre to better define the extent of airway collapse which is physiologically and clinically significant and the optimal treatment pathway for EDAC.

REFERENCE

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Novel approaches to improving COPD care: imaging, AI and acute intervention

P138 CARDIOPULMONARY EVENT OUTCOMES AND RISKS IN COPD ACCORDING TO GOLD STAGE - AN OBSERVATIONAL STUDY IN UK PRIMARY AND SECONDARY CARE

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Introduction and Objectives Chronic obstructive pulmonary disease (COPD) and cardiovascular disease (CVD) are among the leading causes of death globally. COPD confers an elevated risk of CVD and pulmonary events, yet contemporary nationwide information about the magnitude of this risk is limited. We aimed to describe the incidence and timing of cardiopulmonary (CP) events in people with COPD.

Methods This retrospective cohort study included patients with an incident diagnosis of COPD in the Clinical Practice Research Datalink (CPRD), Hospital Episode Statistics (HES) and Office of National Statistics (ONS) linked dataset. Patients aged ≥40 years with a first diagnosis of COPD between 01 January 2014 and 31 December 2019 were included. The primary outcome was CP events defined as any moderate or severe acute exacerbation of COPD (AECOPD), or hospitalisation for heart failure, myocardial infarction, cardiac arrest, stroke or arrythmia (including atrial fibrillation, atrial flutter, and other tachyarrhythmias).

Results Of 79,567 patients with COPD (mean age 68 years, 54.0% male) had a median follow up of 3.2 years. 27.0%, 44.5%, and 5.9% respectively were on an antiplatelet, statin or open triple. 42.7% experienced a CP event within 1 year and 64.1% within 5 years of COPD diagnosis. CP event rate increased according to baseline GOLD stage; 56.9% GOLD stage 1 compared to 68.1% in GOLD stage 4 (figure 1). Up to 5 years follow up, 59.8% had a moderate AECOPD, 12.1% severe AECOPD and 21.1% a cardiovascular event. Rates of CP events were highest in the year post COPD diagnosis (0.85 per patient year (PPY); 95%CI 0.85 - 0.86) but remained high after 5 years of follow up (0.63 PPY; 95%CI 0.62 - 0.63).

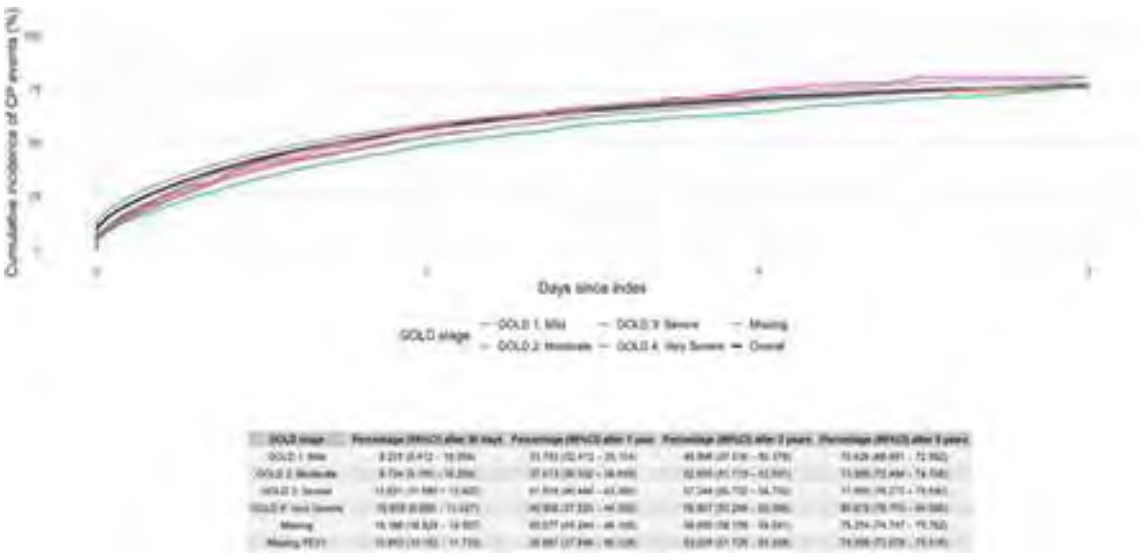
Conclusions Following a diagnosis of COPD, ~2 in 5 people will have experienced a CP event within one year and ~2 in 3 people within 5 years, with higher rates in those with worse lung function. The cardiovascular event rate in COPD, emphasises the need for proactive, early optimisation of guideline-directed management to address CP risk and reduce comorbid disease burden in these patients.

P139 OPPORTUNISTIC THORACIC CT-AI BODY COMPOSITION AS A SARCOPAENIA CASE-FINDING FLAG IN RESPIRATORY DISEASE: DXA VALIDATION

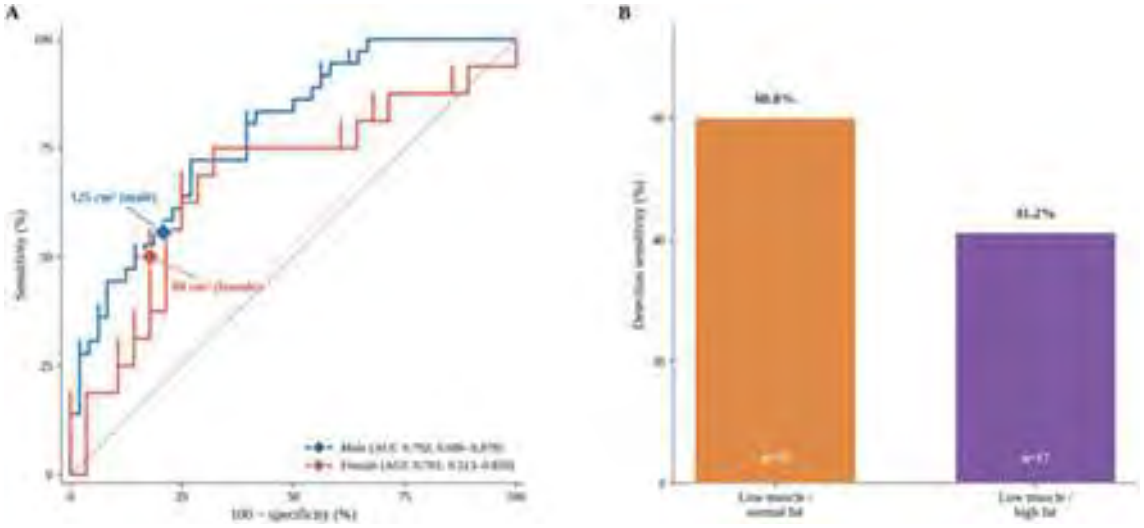
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10.1136/thorax-2026-BTSabstracts.300

Introduction Sarcopaenia is prevalent in respiratory disease and associated with poor outcomes. Formal assessment requires referral for dual-energy X-ray absorptiometry (DXA), rarely



Abstract P138 Figure 1 Cumulative incidence of CP events overall and stratified by GOLD stage



Abstract P139 Figure 1 (A) Receiver operating characteristic curves for CT-AI thoracic muscle area classifying DXA-defined low muscle mass, stratified by sex (male n=84, female n=44). Diamonds mark the EWGSOP2-anchored operating points (125 cm2 male, 80 cm2 female); AUC by bootstrap (2000 iterations). (B) Detection sensitivity for low muscle mass within the two low-muscle Prado phenotypes, showing reduced detection in the low muscle/high fat group. Performance is in-sample; external validation is required. DXA reference: ALMI <7.0 kg/m2 (male), <5.5 kg/m2 (female)

embedded in routine care. Artificial intelligence (AI) pipelines can extract muscle and fat measures from routine thoracic computed tomography (CT). We evaluated a pretrained CT-AI pipeline against DXA as an opportunistic low muscle mass flag.

Methods Two cohorts were pooled: a stable COPD cohort (n=79) and a post-hospitalisation COVID-19 cohort (n=49); n=128, each with CT and DXA. The pretrained pipeline (Xu *et al*., doi:10.1148/radiol.222937 extracted thoracic muscle and subcutaneous adipose tissue (SAT) areas at multiple vertebral levels. Agreement against DXA used Deming regression, Bland-Altman analysis and Lin's concordance correlation coefficient (CCC). T8 muscle area against appendicular lean mass (ALM) was carried forward for classification, ALM providing the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) threshold anchor; T5 SAT against trunk fat was the best fat pair. CT-AI muscle thresholds were back-calculated from the Deming equation at sex-specific EWGSOP2 cut-points. Discrimination used sex-stratified AUROC. Structured agreement failure was characterised using a four-quadrant phenotype framework, cross-classifying low or normal muscle (EWGSOP2 ALMI thresholds) with high or normal fat (within-sample sex-specific trunk-fat median).

Results CT-AI agreed well with DXA across domains: muscle CCC 0.789 (95% CI 0.725-0.837) and fat CCC 0.791 (0.727-0.839). Discrimination for low muscle mass was moderate (AUC 0.792 male, 0.701 female; figure 1A). Using EWGSOP2-anchored thresholds, sensitivity was 53.8% (95% CI 40.5-66.7), specificity 80.3% (70.0-87.7), positive predictive value 65.1% and negative predictive value 71.8% (52 low-mass; 76 normal). False negatives were confined to the two low-muscle phenotypes: miss rates were 40.0% in the low muscle/normal fat group and 58.8% in the low muscle/high fat group (n=17; mean age 72.7 years; figure 1B).

Conclusions CT-AI body composition showed good DXA agreement and moderate discrimination for low muscle mass. Sensitivity is insufficient for conventional screening but defensible as an opportunistic case-finding flag against no assessment. Positive flags warrant confirmatory DXA or clinical evaluation; negative flags should not be treated as reassurance. Failure appeared greatest in the low muscle/high fat phenotype. Prospective validation and operating-point optimisation are planned.

P140 IS LUNG VOLUME REDUCTION CANDIDATE CASE FINDING USING QUANTITATIVE COMPUTED TOMOGRAPHY FEASIBLE IN LUNG CANCER SCREENING?

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10.1136/thorax-2026-BTSabstracts.301

Background COPD is often underdiagnosed and undertreated. Lung volume reduction (LVR) benefits selected individuals with heterogenous emphysema, but patient identification is challenging. Lung cancer screening (LCS) with low-dose CT (LDCT) represents a novel case-finding strategy for potential LVR candidates, but the epidemiology and distribution of emphysema within LCS populations are poorly characterised.

Methods We conducted a retrospective, observational study of individuals with radiologist-reported severe emphysema within the Chesire & Mersey LCS programme (June 2021 – October 2024). Cohort A comprised individuals also known to our respiratory service with clinical metadata including spirometry; Cohort B comprised individuals from other regional sites, without clinical metadata. Consecutive LDCT were analysed using StratX® quantitative CT (qCT) to define the prevalence and distribution of LVR targets.

Results Amongst 61,914 individuals who underwent LCS, 606 had severe emphysema reported. We selected 110 (Cohort A) and 60 (Cohort B); 5 were excluded for missing data. qCT was possible in 159/165 (96%). In Cohort A (n=106, 43 [40%] female, median age 65 [IQR 60-69] years, BMI 23.5

[20.8-27.3] kg/m^2, ppFEV1 57% [38-72]), ppFVC 91% [78-107], 90% had ≥1 target lobe (60% bilateral), 36 (34%) had right-sided heterogenous disease (34 upper lobe-predominant) and 36 (34%) had left-sided heterogenous disease (33 upper lobe-predominant). 26 (25%) met additional LVR referral criteria; 1 (<1%) had been referred previously. In Cohort B (n=53, 22 (42%) female, median age 67.5 [66-73] years), 98% had ≥1 target lobe; 15 (28%) had right-sided heterogenous disease (all upper lobe-predominant) and 9 (17%) had left-sided heterogenous disease (8 upper lobe-predominant); 4 (7.5%) had been referred previously.

Conclusions qCT of LDCT from a regional NHS LCS programme is feasible and identifies a high prevalence of potential LVR targets among individuals with severe emphysema. Future work should explore the implementation and outcomes of targeted LVR case-finding pathways aligned to LCS.

P141 REGIONAL MUCUS PLUG BURDEN PREDICTS ADVERSE OUTCOMES IN COPD PATIENTS IDENTIFIED THROUGH LUNG CANCER SCREENING: AN AUTOMATED QUANTITATIVE CT ANALYSIS

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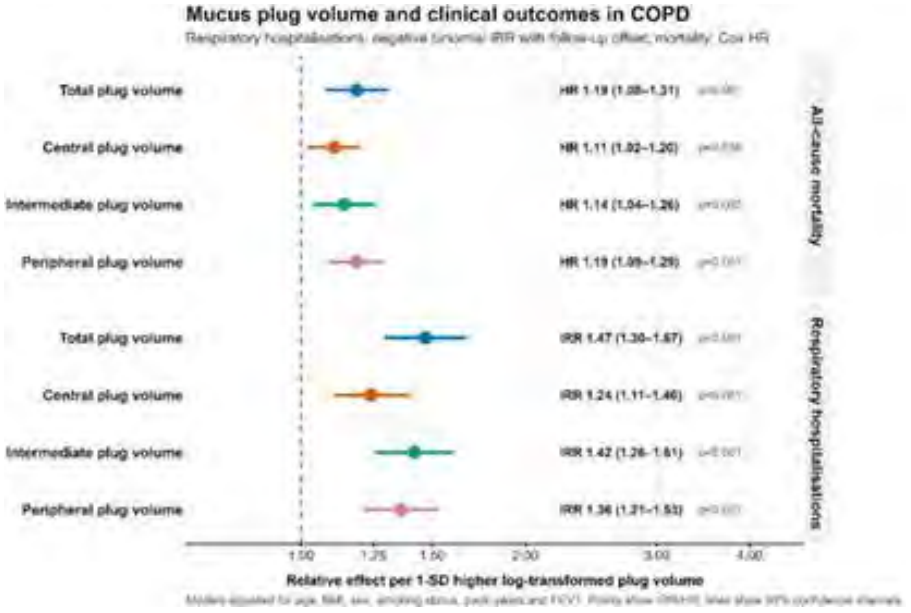
10.1136/thorax-2026-BTSabstracts.302

Introduction and Objectives Mucus plugging is increasingly recognised as an important feature of chronic obstructive pulmonary disease (COPD), contributing to airflow obstruction, exacerbations and disease progression. Automated CT analysis enables objective quantification of mucus plug burden and location, but its prevalence and prognostic significance in screening populations remains poorly characterised. We aimed to determine the prevalence of CT-derived mucus plugging and evaluate the association of total and regional mucus plug burden with respiratory hospitalisations and all-cause mortality.

Methods Participants with COPD (FEV1/FVC<0.70) from the SUMMIT lung cancer screening study who underwent baseline CT and automated mucus plug quantification were included. Total, central, intermediate and peripheral mucus plug volumes were quantified. Respiratory hospitalisations were identified through Hospital Episode Statistics linkage and mortality through registry linkage. Associations with respiratory hospitalisations and mortality were evaluated using multivariable negative binomial and Cox regression models, respectively, adjusted for age, sex, BMI, smoking status, pack-years and FEV1.

Results Among 2,866 participants with COPD, mean age was 66.6±6.1 years, 42.1% were female, mean FEV1 was 66.4±18.7%, and median smoking exposure was 42 pack-years (IQR 33–53.8). Mucus plugging was present in 1,821 participants (63.5%). Intermediate airway plugging was the most common pattern (53.9%), followed by peripheral (35.1%) and central plugging (25.4%), highlighting heterogeneity in airway plug distribution. Over a median follow-up of 6.0 years there were 721 respiratory hospitalisations and 381 deaths. Higher total mucus plug volume was independently associated with increased respiratory hospitalisation rates (IRR 1.47, 95%CI 1.30–1.67, p<0.001) and all-cause mortality (HR 1.19, 95%CI 1.08–1.31, p<0.001) per standard deviation increase in log-transformed volume. Regional plug burden was also prognostic, with intermediate airway plugging demonstrating particularly strong associations with respiratory hospitalisations (IRR 1.42, 95%CI 1.26–1.61, p<0.001), while peripheral airway plugging showed the strongest association with mortality (HR 1.19, 95%CI 1.09–1.29, p<0.001). Associations remained significant across all airway compartments (figure 1).

Conclusions CT-derived mucus plug burden is highly prevalent in COPD identified through lung cancer screening and independently predicts respiratory hospitalisations and mortality. The prognostic significance of plug location suggests that regional mucus plugging may represent a novel imaging biomarker for risk stratification.



Abstract P141 Figure 1

P142 SYSTEMIC CARDIOVASCULAR BURDEN IN COPD: CT-DETECTED AORTIC AND CORONARY ATHEROSCLEROSIS AS A PREDICTOR OF FREQUENT EXACERBATIONS

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10.1136/thorax-2026-BTSabstracts.303

Introduction COPD is a major cause of morbidity and mortality. The frequent exacerbator phenotype (FEP) is associated with worse outcomes and increased healthcare use. Aortic and/or coronary atherosclerosis, often detected incidentally on chest CT, may reflect systemic inflammation and vascular vulnerability, yet its independent predictive value remains uncertain.
Objective To determine whether chest CT–detected aortic and/or coronary atherosclerosis is independently associated with frequent exacerbations in COPD and whether its inclusion enhances prognostic model performance.
Methods Retrospective study of 307 COPD patients evaluated between 2023–2025. FEP was defined as ≥2 moderate exacerbations/year or ≥1 severe episode. Atherosclerosis was extracted from routine chest CT reports. Demographic, clinical, functional and cardiovascular variables were collected. Multivariate logistic regression and ROC analyses were performed ($p < 0.05$).
Results FEP prevalence was 54.2%. Atherosclerosis was more frequent in exacerbators (72.1% vs 43.5%; $p < 0.001$). FEV1 was lower in this group (47.2% ± 12.5 vs 58.6% ± 13.1; $p < 0.01$). In the adjusted model, atherosclerosis was associated with a four-fold higher risk of frequent exacerbation (OR 4.00; $p = 0.036$), while FEV1 remained the strongest predictor ($p < 0.001$). Adding atherosclerosis improved discrimination (AUC 0.746; sensitivity 77.2%, specificity 63.2%) with good calibration (Hosmer–Lemeshow $p = 0.41$).
Conclusion Incidental aortic and/or coronary atherosclerosis on chest CT is an independent biomarker of the frequent exacerbator phenotype. Its inclusion strengthens prognostic accuracy beyond clinical and functional variables, reinforcing its value in risk stratification and preventive strategies.

P143 A STUDY OF USE OF MRI TO MEASURE MODULATION OF EOSINOPHIL RELATED INFLAMMATION IN COPD

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10.1136/thorax-2026-BTSabstracts.304

Introduction Biologics targeting IL5 have transformed asthma care, leading to interest in their use in COPD with an eosinophilic phenotype. Clinical trials have shown some efficacy in reducing exacerbation frequency but with less efficacy than in asthma. Hyperpolarised xenon ventilation MRI (XeMRI) provides a tool to study their effect in vivo, and XeMRI ventilation defect percent (VDP) has been shown to decrease after biologics in asthma.
Methods 25 participants with eosinophilic COPD (FEV1 20-80% predicted, at least one eosinophil count > 0.3x109cells/L plus at least two moderate or one severe exacerbation in the last year) and no history of childhood or active asthma, had XeMRI to measure VDP before and after 12 weeks of treatment with mepolizumab (100mg s/c 4 weekly). Treatment in accord with GOLD guidelines was continued throughout. Participants were stable for 4 weeks before measurement. Funding: GSK supported study (NCT05138250).
Results Participants were aged 67.6(9.8) years. Eosinophil count fell from baseline to 12 weeks (0.31x109 to 0.08x109 cells/L, $p<0.001$) but FEV1 (52.9 to 53.8%, $p=0.27$) and mean VDP (31.5 to 31.4%, $p=0.91$) did not change. Further analysis with XeMRI Treatment Response Mapping (TRM), a method that also assesses regional change in partially ventilated regions, showed improved ventilation in 20/25 participants (80%), significantly more than expected by chance ($p=0.0009$).
Discussion Previous studies with bronchodilators in COPD have shown that VDP decreases by around 4% (Singh et al, 2022, Respir Res.23(1):26). Anti-eosinophil biologics in asthma produce similar results (McIntosh et al, 2023, Chest164(1):27-38) and yet we found no group change in VDP produced by mepolizumab in eosinophilic COPD, with TRM showing more subtle effects. As VDP can vary significantly in response to bronchodilators in COPD, the lack of response to mepolizumab in COPD compared to asthma may be due to differing underlying pathophysiology and not simply greater baseline obstruction.

P144 50 SHADES OF GREEN: PROOF-OF-CONCEPT FOR DIGITAL SPUTUM COLOUR QUANTIFICATION

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10.1136/thorax-2026-BTSabstracts.305

Introduction and Objectives Sputum colour is an established marker of airway inflammation and infection. Objective measurement using the Murray colour chart has been shown to correlate with disease severity and predict exacerbations, hospitalisations, and mortality. Changes in sputum colour can also drive treatment decisions, including antibiotic initiation. We aimed to determine whether sputum colour can be objectively quantified using digital image analysis and whether these measurements are associated with clinically relevant outcomes.
Methods We performed digital colour quantification on sputum samples from patients enrolled in the POSTED study, a prospective longitudinal study of weekly sputum sampling including individuals with bronchiectasis and COPD. Standardised triplicate photographs were obtained per sample, with mean values used for analysis. Nine digital colour metrics were quantified using ImageJ (imageJ.net): Red, Green, Blue, Hue, Saturation, Brightness, a*, l*, and b*. Colour metrics were compared against the Murray reference chart (Mucoid, Mucopurulent, Purulent, and Purulent Severe) and patient-reported sputum colour via the BEST-Colour scale.
Results In the POSTED cohort, 524 sputum samples from 57 patients were included. Median [IQR] age was 70 [65-79]. 42% had a diagnosis of COPD, with the remainder bronchiectasis. All nine metrics correlated significantly with Murray Chart except a* ($\rho = 0.02$, $p = 0.72$). Saturation was the strongest correlate: $\rho = 0.53$ ($p < 0.001$). Correlation with patient-re-

ported BEST-Colour scoring was present but less consistent. Only Saturation ($\rho = 0.29$, $p < 0.001$) and b* ($\rho = 0.21$, $p < 0.001$) reached strong significance. Hue was marginally significant ($\rho = -0.14$, $p = 0.02$). The remaining six metrics were non-significant. A one standard deviation change in saturation was significantly associated with initiation of antibiotics within the next 30 days.

Conclusion This study provides proof-of-concept that sputum colour can be quantified through digital image analysis, and changes in digital colour metrics are relevant to clinical outcomes.

P145 ABSTRACT WITHDRAWN

P146 IMPACT OF PROPHYLACTIC AZITHROMYCIN ON HOSPITAL ADMISSIONS, INPATIENT BED DAYS AND COMMUNITY STEROID USE IN CHRONIC RESPIRATORY DISEASE: A RETROSPECTIVE SERVICE EVALUATION

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10.1136/thorax-2026-BTSabstracts.306

Background Long-term azithromycin is used in selected patients with chronic respiratory disease to reduce exacerbation frequency, although real-world evidence across mixed phenotypes remains limited. We evaluated its impact on healthcare utilisation and corticosteroid use in a specialist respiratory service.

Methods We conducted a retrospective service evaluation of consecutive patients commenced on prophylactic azithromycin for COPD, bronchiectasis, asthma and overlap syndromes at a single-centre district general hospital from January 2023 to April 2026. Outcomes were assessed over 12 months pre- and post-initiation. Primary endpoints included hospital admissions, inpatient bed days and community oral corticosteroid courses. Paired comparisons were performed using Wilcoxon signed-rank testing due to non-normal data distribution. Statistical significance was defined as $p<0.05$.

Results Forty patients were included (mean age 69.9±10.3 years; 52.5% female). Diagnoses included COPD (42.5%), bronchiectasis (35.0%), asthma (17.5%) and overlap syndromes (5.0%).

Hospital admissions decreased from 44 to 31 events (−29.5%; 1.10 vs 0.78 per patient-year; p=0.136). Inpatient bed days reduced from 285 to 130 (−54.4%; 7.12 vs 3.25 per patient-year; p=0.140). Community corticosteroid courses decreased from 120 to 76 (−36.7%; 3.00 vs 1.90 per patient-year; ***p=0.036***), demonstrating statistical significance as shown in table and figure 1. Consistent directional improvement was observed across all healthcare utilisation outcomes, with the greatest absolute reduction in inpatient bed days.

Conclusion In this real-world cohort of chronic respiratory disease patients, prophylactic azithromycin was associated with clinically meaningful reductions in hospital admissions, inpatient bed days and community corticosteroid use. A statistically significant reduction in corticosteroid prescribing was demonstrated, with strong consistent non-significant trends towards reduced acute healthcare utilisation. These findings support the role of macrolide prophylaxis in selected patients and highlight the need for larger prospective evaluations.

Abstract P146 Table1 Clinical outcomes before and after prophylactic azithromycin

Outcome	Before	After	% Change	p value
Hospital admissions	44	31	−29.5%	0.136
Inpatient bed days	285	130	**−54.4%**	0.140
Community steroid courses	120	76	−36.7%	**0.036**

P147 AMINOPHYLLINE DURING AN ACUTE EXACERBATION OF OBSTRUCTIVE AIRWAYS DISEASE: IS IT WORTH IT?

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10.1136/thorax-2026-BTSabstracts.307

Introduction Intravenous aminophylline is often used as an adjunct to conventional treatments in the management of acute exacerbations of asthma and COPD. Its use is limited due to the narrow therapeutic index and high risk of adverse effects such as cardiac arrhythmias.[1] GOLD does not recommend use of methylxanthines during an acute COPD exacerbation.[2] We elected to review the real-time of value of aminophylline during acute exacerbations of asthma and COPD.

Methods We retrospectively reviewed the case notes of patients that were treated for an acute exacerbation of asthma or COPD over 1 year. Baseline data including age, sex, diagnoses and treatments were collected. Outcomes included ICU admission, length of hospital stay, re-admission within 28 days and death.

Results Patients with asthma exacerbation who received aminophylline had a longer stay in hospital (9.4 days vs 4.1 days). They were more likely to require ICU admission (29% vs 0%) and had a higher mortality (12.9% vs 3.3%). They were less likely to be re-admitted within 28 days (6.5% vs 40%).

Patients with COPD exacerbation who received aminophylline had a shorter length of stay (5.7 days vs 6.8 days). They were more likely to require ICU admission (21.4% vs 0%) and had a higher mortality (28.6% vs 0%). They were less likely to be re-admitted within 28 days (7.1% vs 30%).

Overall, in patients that received aminophylline 2 (4.4%) developed 'new' AF due to an acute asthma exacerbation.

In patients that did not receive aminophylline, 3 (6%) developed 'new' AF due to an acute exacerbation of COPD.

Conclusion Patients receiving aminophylline during an acute exacerbation are more likely to require ICU admission and have a high mortality risk. Aminophylline may reduce the length of stay in COPD patients. However, these patients are at risk of developing 'new' AF. Therefore, aminophylline should be used with caution and only when first-line treatments have failed to achieve a good clinical response.

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P148 A PILOT STUDY OF THE EFFECT OF METFORMIN ON AIRWAY GLUCOSE IN COPD: THE METFORMIN AND AIRWAY GLUCOSE IN COPD (MAGIC) TRIAL

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Background and Objective Patients with COPD have elevated levels of airway glucose. This may increase the risk of bacterial infection leading to exacerbation and disease progression. We aimed to test whether treating COPD patients with metformin, an oral hypoglycaemic used in diabetes, reduced airway glucose.
Methods In this randomized, double-blind, placebo-controlled crossover pilot study, we assigned patients with COPD to two 3-month periods of treatment with metformin 1g or placebo twice daily, followed by the alternate treatment after a 2-week washout period. Patients were required to be free of infection, antibiotic or oral steroid treatment in the 8 weeks prior to enrolment. The following were collected at baseline and monthly thereafter: sputum (lower airways sample), nasal/upper airway lining fluid samples using synthetic absorptive matrix (SAM), spirometry, and St Georges Respiratory Questionnaire (SGRQ) and COPD Assessment Test (CAT) scores. The primary outcome was a change in sputum glucose following 3 months treatment with metformin compared to 3 months of placebo.
Results 14 patients were randomised of whom 3 completed the study, mainly due to withdrawals for exacerbations (8/11 withdrawals). In n=4 patients who completed 3 months metformin treatment, there were no significant changes in sputum or nasal glucose with compared to baseline. Metformin did not affect COPD symptom scores or spirometry.
Conclusions Treatment with metformin in this underpowered pilot study did not significantly reduce airway glucose in COPD. Larger studies are required to definitively evaluate this.

P149 EQUITY OF ACCESS TO VIRTUAL WARD (VW) CARE FOR EXACERBATIONS OF COPD: A RETROSPECTIVE COHORT STUDY

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Background Virtual Ward (VW) services are increasingly used to support patients with exacerbations of chronic obstructive pulmonary disease (ECOPD). However, little is known about whether access to VW care is equitable across patient groups and healthcare settings.
Methods A retrospective cohort study utilized routinely collected healthcare data from patients admitted with ECOPD between February 2020 and December 2023 across four hospitals within University Hospitals Birmingham (Queen Elizabeth Hospital Birmingham, Heartlands Hospital, Good Hope Hospital, and Solihull Hospital) in the West Midlands, UK. Virtual Ward (VW) services were primarily delivered through Birmingham Community Healthcare (BCHC) and Solihull Community services. Patients aged ≥35 years with a confirmed diagnosis of ECOPD were included. Admissions resulting in death within 24 hours were excluded. The primary outcome measured was access to VW care. Univariable and multivariable logistic regression analyses were conducted to identify factors associated with VW access. The adjusted model included age, sex, ethnicity, deprivation, and hospital site. Sensitivity analyses excluded admissions involving intensive care unit (ICU) admission or invasive ventilation.
Results The cohort comprised 27,116 admissions, of which 1,040 (3.8%) received VW care. Significant variation in VW access was observed across hospital sites. Increasing age was independently associated with reduced odds of VW access (aOR 0.987, 95% CI 0.981–0.992). Compared with White patients, South Asian patients had substantially lower odds of VW access (aOR 0.323, 95% CI 0.198–0.526). Patients from less deprived groups also had lower access, with reduced odds observed in deprivation quintiles 4 (aOR 0.546, 95% CI 0.377–0.790) and 5 (aOR 0.537, 95% CI 0.356–0.808) compared with the most deprived group. Hospital site demonstrated the strongest association with VW access. Findings remained consistent following exclusion of admissions involving ICU admission or invasive ventilation.
Conclusion Access to VW care for ECOPD was not uniformly distributed across healthcare settings or patient groups. Persistent differences by ethnicity, deprivation, and hospital site remained after adjustment for measured confounders and sensitivity analyses. These findings suggest potential inequalities in access to VW care and highlight the need for further evaluation of referral pathways and service delivery models.

P150 ACUTE EXACERBATION OF COPD: STANDARD INPATIENT CARE VERSUS VIRTUAL WARD STEPDOWN CARE IN A DISTRICT GENERAL HOSPITAL

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10.1136/thorax-2026-BTSabstracts.310

Background Virtual Wards (VW) have been increasingly used in the United Kingdom tofacilitatecommunity care for patients who would otherwise haverequiredlonger periods of inpatient treatment. At Kingston and Richmond NHS Foundation Trust (KFRT), the VW has been managing patients with exacerbations of COPD(ECOPD) with nebulisers,oxygenand monitoring in their own homes since 2022.
Aims To compare outcomes of patients admitted to VW versus those who remained in hospital until discharge. Outcomes assessed included length of stay (LOS), readmission and mortality at 30days, 90 days, and 12 months.
Methods All patients admitted to KRFT with ECOPD in 2023 were retrospectively analysed.
Results 336 patients were included for analysis. 114 were onboarded to VW and 222 remained as inpatients. Key differences in baseline characteristics between the two groupsincluded moreinpatients were current smokers (34.7% versus 15.8%), had unknown COPD GOLD classification (43.2% versus 9.6%) and more acute NIV use (p=0.015) (table 1). Inpatient LOS was 3.0 days shorter in the VW group (p<0.001),howeverthe total LOS was 4.5 days longer in the VW cohort (p<0.001).30 dayCOPD-related readmission ratewassimilar between the two groups (inpatient 21% versus VW 18%)and comparable with national NRAP average of 21.4%1.90 dayreadmission ratetrended towardslower in the VW group (p=0.084). The mortality rate was significantly higher in inpatients compared to VW group at 30 days, 90 days and 1 year (p=0.023, 0.03 and 0.014 respectively).

Conclusions Overall, the results show that ECOPD can be safely managed at home under the monitoring of a VW. Readmission rates trended lower in the VW cohort. Mortality was significantly higher in the inpatient cohort at all time points measured, most likely a consequence of increased comorbidity, type 2 respiratory failure and frailty in this cohort. We hope to compare matched comorbidity groups in the future. We have demonstrated that hospital inpatient LOS can be safely reduced by delivering hospital level care in a VW ward setting at home.

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P151 VIRTUAL OXYGEN WARDS FOR INDIVIDUALS RECOVERING FROM ACUTE RESPIRATORY ILLNESS: A REAL-WORLD OBSERVATIONAL STUDY

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Background The increased burden on secondary care during the COVID-19 pandemic provided a catalyst for considering alternative models to inpatient care. NHS England has issued guidance for the provision of virtual wards for patients with acute respiratory infections who do not require oxygen. However, many patients with COPD and other respiratory conditions require oxygen for a prolonged period during recovery, and there is limited data regarding the safety of providing oxygen at home for these patients.

Methods A retrospective analysis was conducted of patients discharged to an oxygen virtual ward between December 2022 and July 2023 in a UK district general hospital. Patients with improving clinical trajectories and oxygen requirements of up to 4 $L.min^{-1}$ were eligible. The primary outcomes were 30-day mortality and readmission rates.

Results From December 2022 to July 2023 inclusive, 104 patients discharged to the oxygen virtual ward were included in the analysis: 81 were discharged with continuous oxygen and 23 with ambulatory oxygen. Fourteen patients were readmitted within 30 days, and five patients died. There was no significant difference in readmission or mortality rates between those discharged with ambulatory oxygen and those discharged with continuous oxygen (p=0.63). The average per-patient cost of the virtual ward was £786, equivalent to 1.75 acute inpatient medical bed days.

Conclusion Providing oxygen at home for selected patients recovering from acute respiratory illness appears to be safe and low cost, with a low risk of readmission and death. This approach has the potential to significantly alleviate the burden on acute hospitals while allowing better targeting of healthcare resources.

P152 INFLUENCE OF ADMISSION TROPONIN DURING COPD EXACERBATION AND SURVIVAL

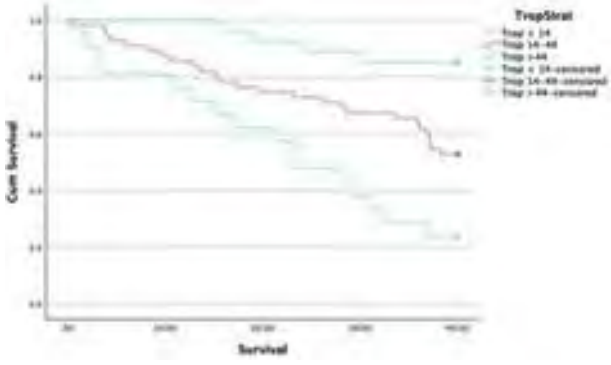
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10.1136/thorax-2026-BTSabstracts.312

Introduction and Aims Increased attention is being focused on the shared vulnerabilities between COPD Exacerbation and Cardiovascular disease. We aimed to evaluate the prognostic value of cardiac troponin measured during admission with COPD on subsequent survival.

Methods We identified all COPD admissions in the Northern HSC Trust in 2023, sampling 40% across 2 sites and recorded baseline demographics; admission investigations including peripheral eosinophil count, Neutrophil Lymphocyte ratio, CRP, Troponin, and BNP; Statin and Antiplatelets prescription; and outcome measures including length of stay (LOS) and survival. We calculated Q-risk and assessed the proportions already on preventative therapy.

Results 1045 admissions had a primary diagnosis of COPD exacerbation, with 761 admitted to Site A. Inpatient mortality rate was 4.4%, with a readmission rate of 23% within 12 months. We sampled 396 patients. Baseline Characteristics: Age 71.7 years (SD 9.6), 31.5% were male, and the mean LOS was 6.90 days (SD 6.7). Troponin was measured 33% on admission, and 22% had a BNP result. On admission, 49% were prescribed statins, and 37% were prescribed antiplatelet agents. LOS correlated ($p < 0.001$) with age [$r = 0.199$], Q risk [$r = 0.209$], and admission Troponin [$r = 0.345$]. There was no influence on LOS when stratifying patients by prevention therapy, peripheral eosinophilia > 0.3 (10.3 % of the cohort), or BNP. Q Risk scoring (n 122) revealed that 20% were moderate risk and 70% were high risk of MACE in the next 10 yrs. Combining these groups, more than 50% were on antiplatelets, and 65% received statins at the time of admission. Survival was 85% at 12 months, 66% at 24 months, and 55% at 36 months*. Stratifying by admission troponin revealed a clear separation in long-term survival (figure 1). A similar pattern was observed with readmission data.



Abstract P152 Figure 1 Survival stratified by troponin on admission for exacerbation of COPD survival functions ILD: cohort studies and service improvement

Conclusion COPD remains a huge burden to healthcare utilisation. Our survival analysis was blinded to the burden of disease, comorbidity and frailty. Admission troponin is useful in predicting long-term survival. The majority of COPD patients are not readmitted within 12 months, indicating hospitalisation may be a critical opportunity to risk-stratify or to initiate cardiovascular prevention therapy.

ILD: cohort studies and service improvement

P153 PULMONARY HYPERTENSION IN NINTEDANIB-TREATED FIBROTIC ILD: REAL-WORLD PREVALENCE AND SAFETY IN A THEORETICALLY AT-RISK COHORT FROM A SINGLE CENTRE EXPERIENCE

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10.1136/thorax-2026-BTSabstracts.313

Background and Aims Nintedanib, a tyrosine kinase inhibitor, has licensed uses in fibrotic interstitial lung disease (F-ILD). Its safety in pulmonary hypertension (PH) is unclear; VEGF inhibition carries a theoretical risk of pulmonary vascular remodelling and precipitating PH. Consequently, patients with significant PH were excluded from pivotal trials. Data on safety is lacking. Aim is to assess safety of nintedanib use in patients with fibrotic ILD and PH with a real-world F-ILD cohort treated with Nintedanib to assess safety and outcomes.
Methods We conducted a retrospective, single-centre analysis of F-ILD treated with Nintedanib who underwent PH evaluation. Data extracted included demographics, echocardiographic parameters, Right Heart Catheterisation (RHC) hemodynamic, and use of PH-targeted therapies.
Results The cohort consisted of 25 patients and mean age: 67; 56% Male), indications for use were Idiopathic Pulmonary Fibrosis (IPF) (36%), and progressive fibrotic ILD. Echocardiography showed high PH probability in 44 %, with Right ventricular (RV) dilation present in 40 %. RHC confirmed significant PH (mPAP range: 32-52 mmHg) in a subset. Notably, 32% (n=8) received concomitant targeted PH therapy. Despite theoretical contraindications regarding VEGF inhibition in PH, Nintedanib was maintained alongside vasodilators. No acute right heart failure explicitly attributable to Nintedanib was observed. Mortality (24%) was driven by ILD progression rather than PH-specific events.
Conclusion Contrary to trial exclusions, real-world practice involves continuing antifibrotics in confirmed PH. While the mechanism of VEGF inhibition poses a theoretical vascular risk, co-administration appeared feasible in this cohort. Vigilant monitoring of RV function is warranted to distinguish potential drug-induced vascular toxicity from hypoxic disease progression. However, large scale studies are needed for a better understanding on this topic.

P154 PPFE: PULMONARY FUNCTION, NUTRITIONAL STATUS AND OUTCOMES FROM A SINGLE CENTER

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Introduction Pleuroparenchymal fibroelastosis (PPFE) is a rare interstitial lung disease characterised by upper lobe fibrosis. UK single-centre outcome data remain limited. We describe clinical features, pulmonary function trajectories, nutritional status and mortality correlates in a defined cohort.
Methods Retrospective review of patients with radiologically confirmed PPFE at a single UK centre (2004–2024). Demographics, comorbidities, CT pattern, BMI, serial pulmonary function tests (PFTs) and vital status were extracted. Group comparisons used Mann-Whitney U and chi-square tests; BMI–PFT correlation used Spearman's ρ.
Results 90 patients were identified (53% male; median age 76 years, IQR 69–83). 40% had died at data censure. Bi-apical CT distribution was present in 76%; 18% demonstrated concurrent lower lobe UIP/honeycombing. Key comorbidities included cardiovascular disease (42%), bronchiectasis (31%), COPD (28%) and GORD (23%); CTD-related PPFE was identified in 12%. Median BMI was low at 21.0 kg/m^2 (IQR 18.9–24.0), with 24% classified as underweight (BMI <18.5). BMI showed a significant positive correlation with FVC %predicted (Spearman ρ=0.247, p=0.049), suggesting nutritional status may reflect disease severity. Baseline FVC was 84% predicted (IQR 64–100) and DLCO 60% (IQR 50–71). Serial PFTs demonstrated significant decline in FVC (median −1%, p=0.040) and FEV1 (median −2%, p=0.030), with 60% showing FVC decline over follow-up. Deceased patients had significantly lower baseline FVC than survivors (59.5% vs 84% predicted, p=0.001). Patients with FVC <70% predicted had a 94% mortality rate. DLCO <60% predicted was associated with 90% mortality, and concurrent UIP pattern trended towards higher mortality (29% vs 6%, p=0.062).
Conclusion In this single-centre PPFE cohort, low BMI is prevalent and correlates with reduced FVC, supporting routine nutritional assessment. Baseline FVC and DLCO are the strongest predictors of mortality, with FVC <70% predicted identifying a very high-risk group. These findings support early identification, serial PFT monitoring and multidisciplinary management in UK PPFE practice.

P155 SURVIVAL AFTER SURGICAL LUNG BIOPSY IN INTERSTITIAL LUNG DISEASE: INFLUENCE OF HISTOPATHOLOGICAL DIAGNOSIS. A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.315

Background The impact of diagnostic lung biopsy on survival in interstitial lung disease (ILD)is poorly defined. In this study, we evaluate overall and early survival in ILD patients undergoing lung biopsy.

Methods We conducted a retrospective study of 99 ILD patients who had lung biopsies from 2008 to 2025. Histopathology groups were categorised as usual interstitial pneumonia (UIP), non-specific interstitial pneumonia (NSIP), hypersensitivity pneumonitis (HSP), non-specific fibrosis, and other ILD patterns (organising pneumonia and respiratory bronchiolitis associated interstitial lung disease). Overall and median survival of each category from the date of biopsy to death or census was analysed using Kaplan–Meier analysis and log-rank test. Overall, post-biopsy 6-month mortality was also assessed.

Results Overall median survival was 68 months. Survival differed significantly across histopathological groups (p=0.0389). Median survival was shortest in UIP, 61.5 months (95% CI 21.1–96.8). In comparison, median survival was 122.6 months (95% CI 57.9-∞) in NSIP, 132.1 months (95% CI 19.9-144.1) in non-specific fibrosis, 140.8 months (95% CI 111.8-∞) in HSP, and 88.8 months (95% CI 63.7-∞) in other ILD patterns. 6-month survival for the overall cohort was approximately 92–93%, showing a 6–8% mortality, with the highest early mortality observed in the UIP subgroup.

Conclusion There are significant differences in survival among different ILD histopathological groups following surgical lung biopsy. Overall, poorer outcomes are demonstrated in the UIP cohort, suggesting underlying disease phenotype to be the driving factor rather than the biopsy procedure itself.

P156 RITUXIMAB IN CONNECTIVE TISSUE DISEASE–ASSOCIATED INTERSTITIAL LUNG DISEASE: A REAL-WORLD COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.316

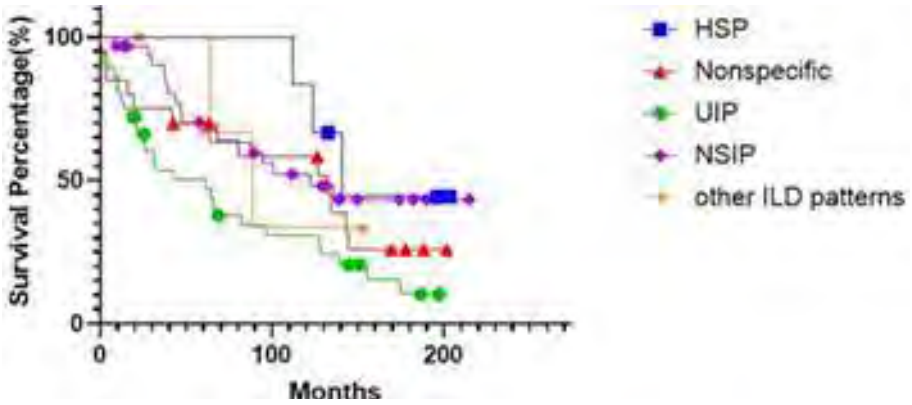
Background Interstitial lung disease associated with connective tissue disorders (CTD-ILD) is a leading cause of morbidity and mortality. Despite progress, there is a significant unmet need for effective management.

Objectives To evaluate the impact of Rituximab (RTX) in CTD-ILD and explore outcomes according to concomitant treatment and underlying CTD.

Methods We retrospectively and prospectively identify CTD-ILD patients treated with RTX. Changes in FVC and DLCO from baseline to follow-up are assessed. Patients are also stratified according to treatment strategy [RTX monotherapy, RTX+ mycophenolate mofetil (MMF), RTX+ Conventional Disease-Modifying Anti-Rheumatic Drugs (cDMARDs)] and underlying CTD.

Results So far, eligible are 40 patients;23 (57,5%) are female with a median age of 59,6 years (range: 21 to 80). Median follow-up is 12.4 months (IQR 7.2–19.8). Main underlying diagnoses are rheumatoid arthritis (22,5%),systemic sclerosis (40%) and inflammatory myositis (35 %). At baseline, median FVC pred is 68 % (range: 39 to115), and median DLCO is 48% (range: 20 to 117).

Following RTX treatment, the median absolute FVC% pred change is +5,4% (range: -16 to 33,p=0,009). while median relative FVC% pred change is +6,44% (range:-23,3 to +50,9,p=0,010). Median and mean FVC change is +90ml and +123ml respectively (range, -720 ml to +1980 ml). No significant changes are observed in DLCO.



Abstract P155 Figure 1

Comparison between RTX monotherapy (n=10), RTX+MMF (n=17), and RTX+ cDMARDs (n=11) show no significant differences. However, RTX+cDMARDs demonstrate the greatest numerical improvement in FVC, with median FVC change +100ml,+40ml and +220ml respectively.

Among RTX+MMF-treated patients, those receiving concomitantly nintedanib (n=6) show better FVC preservation than those treated with RTX+MMF alone (median relative FVC% pred change: 6.6% vs −1.1%). Nevertheless, no statistically significant difference was found.

Patients with IIM- ILD demonstrate the most favorable response, with median FVC gain +260ml (range: −650ml to +890 ml), although differences between CTD subgroups are not statistically significant.

SARS-CoV-2 infection occurred in 8 patients (20%).

Conclusions RTX is associated with significant improvement in FVC or stabilization of pulmonary function. Although no statistically significant, the greatest benefit is observed in patients with inflammatory myositis and in those receiving RTX+ cDMARDs. Ongoing prospective follow-up will further evaluate subgroup differences.

P157 THE INTERDISCIPLINARY INTERSTITIAL LUNG DISEASE (ILD IN) GROUP HIGHLIGHTS THE NEED TO OPTIMISE THE ILD SPECIALIST PRACTITIONER'S ROLE ACROSS THE ILD CLINICAL PATHWAY

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10.1136/thorax-2026-BTSabstracts.317

Background Specialist interstitial lung disease (ILD) clinical pathways aim to standardise the delivery of care and management of patients with ILD across the UK. However, variation in service provision and workforce capability persists, highlighting the need for a clearly defined specialist ILD healthcare practitioner role to meet the needs of modern ILD services.

Aim We developed a process protocol to identify workforce gaps and define the competencies, skills, and professional requirements necessary for ILD healthcare practitioners to function effectively as core members of contemporary specialist ILD multidisciplinary services.

Methods A national Taskforce comprising by ILD specialists from diverse professional backgrounds, steering committee of the ILD Interdisciplinary Network (ILD-IN), developed a strategic workforce-mapping protocol with a plan to include the following actions:

- Mapping the current workforce needs and gaps across specialist ILD services.
- Undertaking stakeholder engagement to capture perspectives on the role and its future requirements.
- Identify key stakeholders involved in ILD service delivery and workforce development.
- To reach consensus on the core competencies, knowledge, skills, leadership attributes, and service improvement capabilities required for the specialist ILD healthcare practitioner role.

Results The Survey findings and stakeholder feedback will undergo thematic analysis, with quantitative data summarised using descriptive statistics. Key themes will identify a range of clinical, leadership, operational, educational, and service development competencies required to meet the evolving demands of highly specialised ILD services. These findings are expected to inform the development of a competency-based framework to support workforce development and standardisation of practice.

Conclusion The effectiveness and sustainability of specialist ILD services depend on a skilled and competent workforce capable of meeting the increasing complexity of contemporary ILD care pathways. This work will provides an evidence-informed foundation for the development of a specialist ILD competency framework, supporting workforce transformation and enhancing the quality and consistency of care delivered across ILD services.

P158 EXPERT ILD REVIEW IMPROVES DIAGNOSTIC CONFIDENCE AND REDUCES UNCLASSIFIABLE DISEASE IN FIBROTIC ILDS

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10.1136/thorax-2026-BTSabstracts.318

Background Diagnostic uncertainty remains common in fibrotic interstitial lung diseases (ILDs), particularly outside idiopathic pulmonary fibrosis (IPF), despite multidisciplinary team (MDT) discussion.

Aim To evaluate the impact of structured expert ILD clinician review on diagnostic confidence and classification within an MDT setting.

Methods Consecutive patients referred to a tertiary ILD service between July 2019 and May 2020 with suspected fibrotic ILD were prospectively assessed. Cases were discussed at MDT using referrer information, then underwent structured expert ILD clinician review, followed by blinded re-discussion at MDT. Diagnoses were assigned with likelihood thresholds and diagnostic confidence categorised using established Ryerson criteria. Diagnostic changes and certainty were assessed pre- and post-review.

Results Sixty-nine patients were included. Agreement between referrer and final MDT diagnosis was fair ($\kappa=0.28$). Overall agreement between pre- and post-review MDT first-choice diagnosis was good ($\kappa=0.69$) and was highest for IPF ($\kappa=0.91$). Expert review led to revision of first-choice diagnosis in 33% of non-IPF cases, most commonly due to newly identified exposures, drug history, or connective tissue disease features. The proportion of unclassifiable ILD decreased from 11.6% to 2.9% ($p=0.041$). High diagnostic certainty increased from 59.4% to 72.5%, driven by changes within the non-IPF ILD group.

Conclusion Structured expert ILD clinician review prior to MDT discussion significantly reduces diagnostic uncertainty and improves confidence, particularly in non-IPF fibrotic ILD, supporting its routine incorporation into diagnostic pathways.

P159 REGIONAL INTERSTITIAL LUNG DISEASE MDT COLLABORATION BETWEEN A TERTIARY CENTRE AND DISTRICT GENERAL HOSPITALS: ACCEPTABILITY, FEASIBILITY, AND EDUCATIONAL IMPACT

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10.1136/thorax-2026-BTSabstracts.319

Aims Regional MDT models have been proposed to extend tertiary ILD expertise into secondary care, yet evidence on its acceptability across diverse professional groups remains limited. This study evaluates case outcomes and staff experience of a virtual regional ILD MDT, to inform a national model of integrated ILD care delivery.

Methods MDT forums incorporated tertiary ILD specialist, local respiratory physicians, radiologists, rheumatologists, and specialist nurses across eight district hospitals. Cases were prospectively classified: no ILD (C1); ILD for secondary care management (C2); routine tertiary referral (C3); urgent tertiary referral (C4). Following one year of operation, a survey was distributed to all participating staff. Respondents rated impact on practice, diagnostic confidence, and referral behaviour using five-point Likert scales. Open-ended questions explored valued aspects, areas for improvement, and referral practice changes.

Results Across 89 meetings (January–December 2025), 1,000 cases were reviewed. The majority (76%) were managed locally (C1 21%, C2 55%, C3 23%, C4 1%). Routine waiting times to ILD services fell from 12 to 4 weeks; urgent cases maintained consistent two-week access.

Survey responses were received from 50 participants across all eight trusts, representing consultants and specialist physicians (n=29), radiologists (n=7), specialist nurses (n=5), rheumatologists (n=3), and allied health professionals (n=6). 96% reported the MDT changed how they manage ILD patients and 90% reported a change in referral behaviour. Confidence improved across all domains (see figure 1). All (100%) would recommend this model to other centres; 98% agreed the MDT improves local patient care quality and 96% agreed it reduces unnecessary patient journeys. Open-ended responses identified access to tertiary expertise, streamlined referral pathways, and peer learning as most valued; meeting duration, job planning, and documentation were most cited areas for improvement.

Conclusions Regional virtual ILD MDT collaboration enabled local management of the majority of cases and reduced tertiary waiting times. Survey data demonstrate strong acceptability across all professional groups with meaningful gains in diagnostic confidence and referral behaviour. This model warrants national implementation with appropriate funding, delivering equitable ILD care closer to home and meeting NHS England priorities for integrated specialist services.

P160 INCIDENCE, PREVALENCE AND THE DIAGNOSTIC ODYSSEY IN RARE CYSTIC LUNG DISEASE

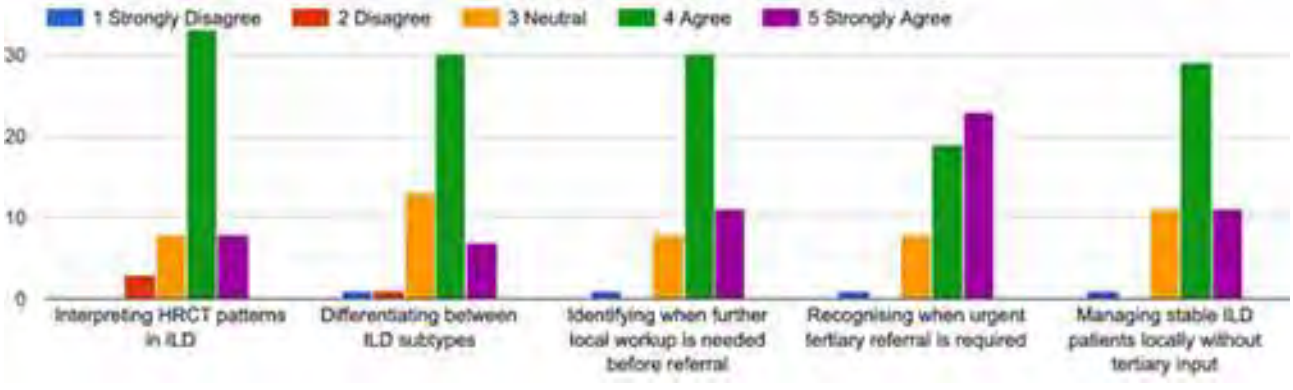
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10.1136/thorax-2026-BTSabstracts.320

Introduction Rare cystic lung diseases (RCLD), including lymphangioleiomyomatosis (LAM), Birt–Hogg–Dubé syndrome (BHD), pulmonary Langerhans cell histiocytosis (PLCH), lymphocytic interstitial pneumonia (LIP), and light chain deposition disease (LCDD), are conditions in which healthy lung parenchyma is replaced by non-functional cysts. Like many rare diseases there is little understanding of RCLDs and it is difficult to ascertain the true burden of these diseases due to insufficient epidemiological data. Our aim is to estimate the incidence and prevalence of RCLD in the UK by using real-world data and examine the diagnostic journey of patients with RCLD.

Methods We conducted a retrospective cohort-study using UK primary care electronic health records from Clinical Practice Research Datalink (CPRD). Individuals aged ≥ 18 years with a diagnosis of RCLD between 1990-2023 were identified using pre-defined diagnostic code lists (SNOMED-CT). Hospital Episode Statistic (HES) were used alongside CPRD data to analyse diagnostic journey and temporal analyses.

Result From CPRD record, 140 eligible incident cases with RCLD were identified between 1990 to 2023. LAM was the most diagnosed RCLD with an incidence of 0.3 per million people, followed by LIP with an incidence of 0.19 per million people, PLCH (0.047 per million people), BHD (0.0079 per million people) and LCDD (0.0039 per million people). Based



Abstract P159 Figure 1 Self-reported improvement in clinical confidence across five clinical domains following regional ILD MDT participation (n=50)

on CPRD data, 14.2% of cases had no presenting symptoms identified pre-index whilst 64.7% had presented to their primary care with respiratory symptoms prior to diagnosis. 51% of cases had a history of tobacco use and only 14.6% had pneumothorax prior to diagnosis.

Conclusion RCLDs are rare within UK primary care. The incidences differ across diseases. This study has provided the first contemporary population-based estimates of RCLD incidence and can be used to inform future epidemiological research.

P161 REAL-WORLD NINTEDANIB OUTCOMES FOLLOWING ANTIFIBROTIC ELIGIBILITY EXPANSION AND ILD SERVICE REDESIGN AT A UK SPECIALIST CENTRE

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10.1136/thorax-2026-BTSabstracts.321

Background Expansion of antifibrotic prescribing criteria to include patients with progressive pulmonary fibrosis (PPF) and those with preserved lung function in idiopathic pulmonary fibrosis (IPF) substantially increased demand on UK interstitial lung disease (ILD) services. In response, the Southampton ILD service underwent redesign including pathway optimisation, expansion of the multidisciplinary team, enhanced antifibrotic support, and streamlined referral processes. We evaluated nintedanib treatment outcomes before and after implementation of these changes.

Methods Retrospective evaluation of patients commencing nintedanib before (January 2021-December 2022) and after (January 2023-September 2024) ILD service redesign. Patients with at least 12 months follow-up were included. Demographics, physiology, treatment and outcome data were collected from electronic patient records and antifibrotic service databases.

Results A total of 248 patients were included (95 pre-2023 and 153 post-2023). Service redesign was associated with a reduction in mean referral-to-first-appointment time from 18.5 to 5.2 weeks and an increase in pre-treatment pharmacist counselling from 38.9% to 100%. Patients commencing treatment post-2023 had higher baseline lung function, with mean FVC 77.5% (SD 17.9) versus 70.4% (SD 13.5) and mean TLCO 49.8% (SD 16.6) versus 46.5% (SD 14.6). Full-dose treatment persistence at 12 months increased from 20.0% to 43.1%, while dose reduction rates decreased from 39.0% to 31.4%. All cause discontinuation at 12 months was lower in the post-2023 cohort (25.5% vs 41.1%; $p<0.001$), with a corresponding reduction in 12-month mortality (11.1% vs 27.4%; $p=0.001$).

Conclusions Following expansion of antifibrotic eligibility and implementation of a redesigned multidisciplinary ILD service, patients commencing nintedanib demonstrated improved treatment persistence and lower 12-month mortality. These improvements occurred alongside earlier specialist assessment, higher baseline lung function at treatment initiation, and enhanced antifibrotic support. Further studies are required to determine the relative contribution of these factors to improved clinical outcomes.

P162 PREVALENCE AND CLINICAL MANAGEMENT OF PATIENTS WITH ILD ASSOCIATED WITH PH – A UK REGIONAL ILD CENTRE PERSPECTIVE

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Rationale Pulmonary hypertension (PH) is a common, serious comorbidity of interstitial lung disease (ILD), associated with increased mortality. Early recognition can support more timely, targeted care.

Methods Retrospective data from a UK regional centre (Nov 2024–Nov 2025) were screened; cases with intermediate or high echocardiographic likelihood of pulmonary hypertension were analysed for key clinical, radiological, and echocardiographic features (figure 1).

Results Of 777 patients screened, 54 (7%) had intermediate or high echocardiographic probability of PH. Mean maximum tricuspid regurgitation velocity (TRVmax) was 3.25 m/s and mean systolic pulmonary artery pressure (sPAP) 43.4 mmHg. Pro-BNP was measured in 70%, median of 515.5 pg/mL. Mean FVC/DLCO ratio was 1.9, above the >1.6 PHrisk threshold in ILD. Oxygen therapy was used by 31.5% (17/54): 22%

Abstract P162 Table 1 Clinical, Radiological, Functional and Echocardiographic Characteristics of ILD Patients with Pulmonary Hypertension: Regional ILD Service from November 2024 to November 2025



ambulatory oxygen and 9% ambulatory and long-term oxygen. Cardiovascular comorbidities were present in 87% (38/54), most commonly arterial hypertension (38.9%), atrial fibrillation (38.9%), and ischemic heart disease (9.3%). Emphysema was identified on CT scan in 35.2%. By mid-January 2026, 22.2% (12/54) had died during follow-up. Only 7% (4/54) were referred to a PH centre, with one patient receiving phosphodiesterase-5 inhibitor therapy.

Conclusion Our data show frequent echocardiographic likelihood of PH in the ILD cohort, underscoring the need for a carefully defined PHILD care pathway to ensure accurate diagnosis and timely access to targeted treatment.

P163 NAVIGATING TREATMENT INTOLERANCE: REAL-WORLD OUTCOMES OF ANTIFIBROTIC SWITCHING IN IPF AND PPF

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Introduction Antifibrotic therapies slow disease progression in idiopathic pulmonary fibrosis (IPF) and progressive pulmonary fibrosis (PPF), but adverse events (AEs) frequently limit tolerance. Observational studies suggest that switching therapy may improve outcomes in patients intolerant of initial treatment. However, prospective and randomised data are lacking. This study evaluated real-world outcomes following antifibrotic switching due to treatment intolerance in IPF and PPF.

Methods We conducted a retrospective review of medical records of patients attending the ILD clinic at Cork University Hospital from 2014-2025. Patients with IPF or PPF receiving antifibrotic therapy were identified, and those requiring treatment interruption due to AEs were analysed. Data collected included initial antifibrotic agent, reason for treatment interruption, dose reduction, antifibrotic switching, and post-switch outcomes.

Results Among 518 patients on antifibrotic therapy (70% IPF, 30% PPF), treatment interruption due to AEs occurred in 112/312 (36%) patients initiated on nintedanib and 61/206 (30%) patients initiated on pirfenidone.

Following interruption of nintedanib, 22/112 (20%) patients tolerated nintedanib after dose reduction, 24/112 (21%) discontinued therapy, and 66/112 (59%) were switched to pirfenidone. Among patients switched from nintedanib to pirfenidone, 7/66 (11%) required further dose reduction and 17/66 (26%) discontinued treatment due to recurrent AEs.

Following interruption of pirfenidone, 10/61 (16%) tolerated pirfenidone after dose reduction, 11/61 (18%) discontinued therapy, and 40/61 (67%) patients were switched to nintedanib. Among patients switched from pirfenidone to nintedanib, 9/40 (23%) required subsequent dose reduction and 7/40 (18%) discontinued treatment due to AEs.

Overall, 82/106 (77%) patients who underwent antifibrotic switching remained on disease-modifying therapy without subsequent treatment discontinuation (table 1).

Gastrointestinal AEs were the commonest cause of treatment interruption in both the nintedanib (49%) and pirfenidone (31%) cohorts. The next most common AEs were liver enzyme derangement with nintedanib (24%) and rash with pirfenidone (21%).

Conclusions In real-world practice, antifibrotic switching is frequently employed following treatment intolerance and enables ongoing therapy in a substantial proportion of patients. However, post-switch dose reduction and treatment discontinuation remain common. Proactive AE monitoring and timely dose adjustment may help maximise long-term exposure to disease-modifying therapy in routine clinical practice.

Abstract P163 Table 1

	Nintedanib	Pirfenidone
Outcomes after antifibrotic switching	Switched from Pirfenidone to Nintedanib: n=40 • Dose reduction: 9 (23%) ○ Main adverse events: ▪ GI symptoms: 8 ▪ Fatigue: 1 • Discontinued: 7 (18%) ○ Main adverse events: ▪ GI symptoms: 5 ▪ LFT derangement: 2	Switched from Nintedanib to Pirfenidone: n=66 • Dose reduction: 7 (11%) ○ Main adverse events: ▪ GI symptoms: 4 ▪ Dizziness: 1 ▪ Rash: 1 ▪ Unclear: 1 • Discontinued: 17 (26%) ○ Main adverse events: ▪ Rash: 3 ▪ GI symptoms: 3 ▪ Fatigue: 2 ▪ GI symptoms and Fatigue: 1 ▪ LFT derangement: 1
Remained on antifibrotic after switching (n,%)	33/40 (83%)	49/66 (74%)
Overall remained on antifibrotic after switching (n,%)	82/106 (77%)	

P164 THE CLINICAL VALUE AND COST-EFFECTIVENESS OF ROUTINE BLOOD MONITORING IN PATIENTS ON PIRFENIDONE FOR IDIOPATHIC PULMONARY FIBROSIS: A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.324

Background and Aims Pirfenidone is a widely used antifibrotic therapy in the management of idiopathic pulmonary fibrosis (IPF).[1] Manufacturers recommend baseline renal function testing and monthly liver function (LFT) monitoring for the first 6 months, then three monthly thereafter.[2] Within the health-board, routine monitoring also includes full blood count (FBC) and renal function. We evaluated whether routine blood monitoring following Pirfenidone initiation identified clinically relevant abnormalities leading to treatment discontinuation. We assessed the cost per clinically relevant finding, evaluated drug tolerance and adherence to manufacturer-recommended monitoring.

Methods We conducted a retrospective study of 86 patients who initiated Pirfenidone for IPF in the Health Board between July 2021 and November 2025 and remained on treatment for at least 2 months; seven patients were excluded for shorter treatment duration. Routine outpatient monitoring of FBC, LFT and renal function in months 1,2,3,6,9 and 12 following treatment initiation were reviewed. Clinically relevant abnormalities resulting in treatment discontinuation were identified.

Results See figure 1. 88.6% of patients underwent all manufacturer-recommended blood tests. Three clinically relevant abnormalities were identified, all involving LFTs within months 1-3. The cost per clinically relevant blood test result was £1051.97, with 418 tests performed per clinically relevant finding. 78% of patients tolerated treatment and 22% discontinued. Among those who discontinued, 84% stopped because of side effects, and 16% due to abnormal LFTs.

Conclusion Routine blood monitoring following Pirfenidone initiation produced a low yield of clinically relevant abnormalities leading to treatment discontinuation. No clinically relevant abnormalities were identified in FBC or renal function monitoring, while three were detected through LFT monitoring. These findings are consistent with pivotal trial data (ASCEND/CAPACITY), which reported rare treatment discontinuation due to LFT derangement (2-4%). The substantial cost in time and resources per clinically relevant finding raises questions regarding the frequency, duration and necessity of current monitoring practices. As many UK regional ILD centres routinely monitor renal function and/or FBC alongside recommended LFT monitoring, these findings have implications for reducing unnecessary testing and associated costs across the UK.

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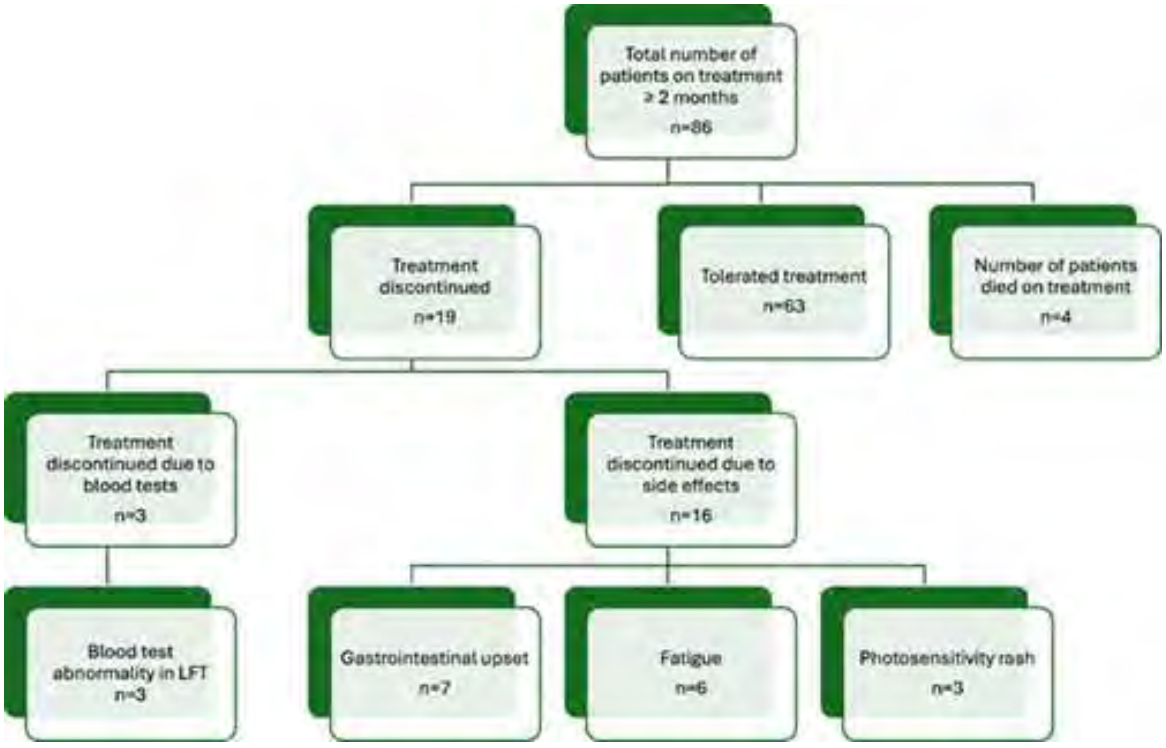
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P165 ANTI-CARBAMYLATED PROTEIN ANTIBODIES IN FIBROINFLAMMATORY LUNG DISEASE INCLUDING RHEUMATOID ARTHRITIS & SARCOIDOSIS

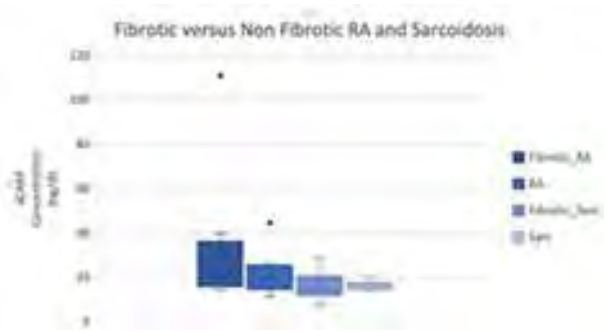
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10.1136/thorax-2026-BTSabstracts.325

Introduction Interstitial lung disease (ILD) can be a devastating consequence of Rheumatoid arthritis (RA). Serological markers are used in the diagnosis of RA, however 30% of RA patients remain seronegative. Anti-carbamylated protein antibodies (anti-CarP) and antibodies against peptidyl-arginine deiminase type four (anti-PAD4) may be useful in diagnosing RA ILD. We



Abstract P164 Figure 1



Abstract P165 Figure 1

aim to investigate this relationship and assess for any utility in Sarcoidosis.

Methods aCARP and PAD4 was quantified in 19 serum samples in duplicate using Human aCARP ELISA Kit 19 from patients with RA. Results were correlated to and stratified by clinical data, smoking status and qualitative CT descriptors of disease burden. A subgroup analysis (n 6) assessing fibrotic burden by quantitative CT Lung Texture Analysis (LTA) was performed. The RA cohort was compared to another fibroinflammatory lung condition Sarcoidosis (n = 16)

Results The mean age of the RA cohort was 68.5, 47% were male, 63% were Seropositive, 58% had fibrotic disease [table 1]. There were no significant differences for the presence or absence of ILD for all measures prior to stratification, excepting greater age. Correlation between aCARP and DASCRP 0.518 (p 0.023) and ESR 0.489 (p 0.034) was found. Using qualitative burden of disease [None/Mild/Moderate/Severe]: there were significant difference in aCARP between groups p 0.004. PAD4 demonstrated a similar pattern. Smoking status was also significant p 0.035. LTA found a strong relationship to Pulmonary Vascular Volume [PVV], an indirect marker of fibrotic disease. There did not appear to be a useful relationship in the Sarcoidosis comparison (figure 1).

Conclusions The results suggest a strong association with disease burden and PVV in RA patients but not in sarcoidosis. It is not clear if these antibodies could help identify high-risk RA patients who would benefit from intensive monitoring in early disease or help guide treatment choices.

P166 INTRAVENOUS METHYLPREDNISOLONE IN ACUTE EXACERBATIONS OF INTERSTITIAL LUNG DISEASE: A SINGLE-CENTRE EXPERIENCE

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10.1136/thorax-2026-BTSabstracts.326

Introduction and Objectives Acute exacerbations of interstitial lung diseases (AE-ILD) are associated with high morbidity and mortality. intravenous methylprednisolone pulse therapy (IV MP) is widely used as the first-line treatment despite limited high-quality evidence supporting its efficacy in different ILD subtypes. This study evaluated survival, radiological and physiological outcomes following IVMP therapy and explored potential prognostic markers.

Methods A retrospective audit was conducted in 58 patients treated with IVMP for AE-ILD between 2018 and 2024 at Luton and Danstable University Hospital. The patients received 500–1000 mg of intravenous methylprednisolone daily for three consecutive days followed by oral prednisolone. ILD subtypes were classified according to 2025 ATS/ERS recommendations and MDT outcomes. The primary outcome was 90day survival. Secondary outcomes included radiological response assessed by CT imaging at 8–12 weeks and physiological response measured by forced vital capacity (FVC) at 3–6 months. Autoimmune profiles were reviewed to identify connective tissue disease–associated ILD and assess prognostic implications.

Results Main ILD subtypes consisted of connective tissue disease–associated ILD (CTDILD, 34.5%), idiopathic pulmonary fibrosis (IPF, 27.6%), idiopathic nonspecific interstitial pneumonia (NSIP, 15.5%), and hypersensitivity pneumonitis (6.9%). Ninetyday survival was 68.8% in IPF and 60.5% in nonIPF ILD, with no significant difference (p = 0.763). 41 patients (70.7%) had follow up CT scans and 31.7% demonstrated radiological progression despite treatment. Nonfibrotic ILDs showed significantly greater radiological improvement compared with fibrotic patterns (90.9% vs 52.6%, p = 0.049). Patients demonstrating CT improvement had significantly better survival (85.7% vs 53.8%; OR 5.14, p = 0.049). Lung function data were available in 23 patients, with a greater mean FVC decline observed in those with IPF. Autoimmune markers were present in 60.3% of patients but were not associated with a significant survival difference.

Conclusions IV MP therapy demonstrates comparable short-term survival across ILD subtypes in AEILD. Nonfibrotic ILD shows superior radiological response, and early CT improvement is a strong prognostic indicator. These findings support continued use of IV MP in AEILD and highlight the value of subtypespecific and imagingguided treatment strategies. A local treatment protocol was developed based on these findings.

P167 SURVIVAL AFTER ANTIFIBROTIC INITIATION IN RHEUMATOID ARTHRITIS-ASSOCIATED USUAL INTERSTITIAL PNEUMONIA COMPARED WITH IDIOPATHIC PULMONARY FIBROSIS: A REAL-WORLD RETROSPECTIVE COHORT STUDY

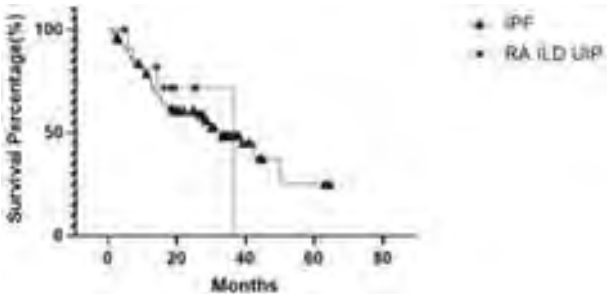
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10.1136/thorax-2026-BTSabstracts.327

Introduction Antifibrotic therapy slows disease progression in idiopathic pulmonary fibrosis (IPF) and rheumatoid arthritis-associated (RA-UIP). However, real-world survival outcomes following antifibrotic initiation in RA-ILD with a usual interstitial pneumonia pattern (RA-UIP) remain poorly characterised. We compared survival after antifibrotic initiation in patients with RA-UIP and IPF.

Methods We performed a retrospective cohort study of patients with fibrotic interstitial lung disease who were initiated on antifibrotic therapy at a regional interstitial lung disease service. Patients were classified as having IPF or RA-UIP. Survival was measured from the date of antifibrotic initiation. Kaplan-Meier analysis was used to estimate survival, and survival curves were compared using the log-rank test. An unadjusted hazard ratio (HR) for mortality was calculated.

Results Ninety-nine patients were included: 87 with IPF and 12 with RA-UIP. Median follow-up from antifibrotic initiation was 34.0 months in IPF and 19.3 months in RA-UIP. During follow-up, 43 deaths occurred in the IPF group, and 4 deaths



Abstract P167 Figure 1

occurred in the RA-UIP group. Median survival following antifibrotic initiation was 32.4 months in IPF and 36.6 months in RA-UIP. Survival did not differ significantly between groups by log-rank testing (p=0.77). The unadjusted HR for mortality in IPF compared with RA-UIP was 1.17 (95% CI 0.45–3.05).

Conclusion In this real-world cohort, observed survival after antifibrotic initiation was not significantly different between patients with IPF and RA-UIP. Although RA-UIP patients had numerically longer median survival and fewer deaths proportionally, the small RA-UIP sample size, low number of events, and wide confidence intervals preclude definitive conclusions. Larger multicentre studies are needed to better characterise outcomes following antifibrotic initiation in RA-UIP.

Bronchiectasis unconfined

P168 IMMUNE SIGNATURES AND INFLAMMATORY CELL PROFILES DIFFER BETWEEN DISEASED AND NON-DISEASED LUNG SEGMENTS OF ANATOMICALLY HETEROGENOUS BRONCHIECTASIS

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10.1136/thorax-2026-BTSabstracts.328

Introduction Immune dysregulation is a characteristic feature of bronchiectasis but, whether in heterogenous disease,[1] this occurs uniformly throughout the lungs or is concentrated in areas where bronchiectatic changes are localised remains unknown. We sought to investigate whether immune signatures observed in diseased lung segments are similar or distinct from those in non-diseased regions.

Methods We performed bronchoscopy in subjects with bronchiectasis and systematically took samples from diseased and non-diseased regions (defined radiologically on prior CT scan). Bronchoalveolar lavage (BAL) and bronchial brushings were taken and analysed by flow cytometry to enumerate granulocytes and RNA-sequencing to characterise immune signatures respectively.

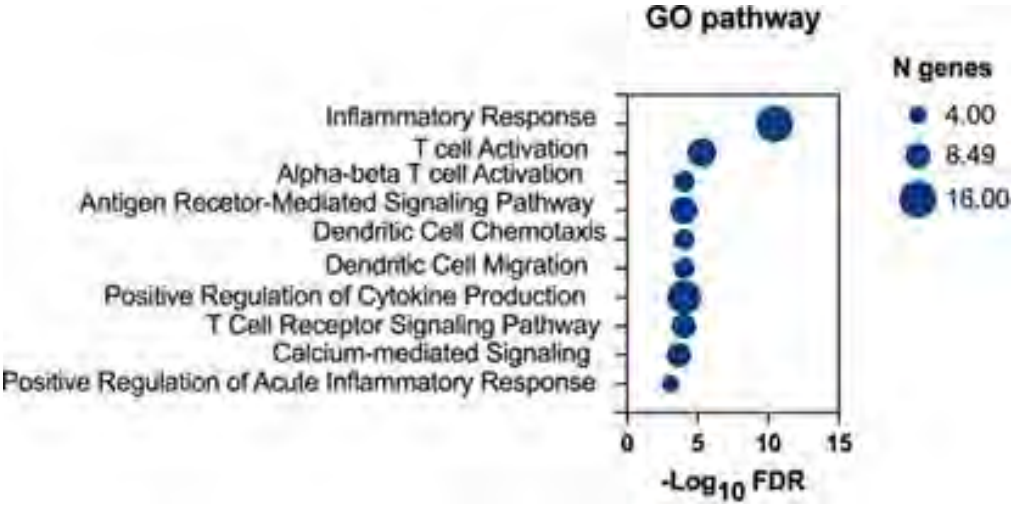
Results 12 subjects with bronchiectasis were included and all had sampling at diseased (right middle lobe (RML) 58.3%, right lower lobe (RLL) 16.7%, lingula 16.7%, left lower lobe (LLL) 8.3%) and non-diseased lung regions (lingula 41.7%, LLL 33.3%, RML 16.7% RLL 8.3%. There were no significant differences in % eosinophils and neutrophils between diseased and non-diseased lung segments (expressed as proportion of total CD45-positive cells (P=0.73 and 0.42 respectively). There was a trend towards increased CXCR4-expressiing neutrophils in diseased lobes (P=0.077), indicative of greater neutrophil activation and active NETosis. Conversely, CXCR2-expressing neutrophils were significantly reduced in diseased lobes (P=0.0425), indicating a profile of more immature neutrophils being present in these regions.

Transcriptomic evaluation of bronchial brushings, identified 191 differentially expressed genes that were upregulated (Log-2Fold change>1, P adjusted<0.1) in diseased segments. Gene ontology analysis identified enrichment of inflammatory processes and T-cell and dendritic cells related pathways, as seen in figure 1, suggesting that differences in non-granulocytic cells may distinguish immune profiles in diseased and non-diseased segments.

Conclusion Adaptive immune signatures and inflammatory cell profiles differ between diseased and non-diseased areas in bronchiectasis. Local microbiome profiles may be an important determinant of this and our future studies will integrate microbial and immune data in this context. The findings may also inform drug-delivery requirements for future lung-targeted therapies.

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Abstract P168 Figure 1 Gene ontology analysis of observed pathways in diseased vs non-diseased lobes

P169 CLINICAL, PHYSIOLOGICAL AND MICROBIOLOGICAL CHARACTERISTICS BY TYPE 2 INFLAMMATORY STATUS IN BRONCHIECTASIS

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Background Type 2 (T2) inflammation has emerged as a relevant inflammatory endotype in bronchiectasis; however, its association with disease characteristics remains elusive. We aimed to explore clinical, physiological and microbiological characteristics according to T2 inflammatory status in a prospective bronchiectasis cohort.

Methods This preliminary cross-sectional analysis included prospectively recruited patients with clinically and radiologically confirmed bronchiectasis attending a tertiary outpatient bronchiectasis clinic. Patients with smoking history ≥10 pack-years, asthma or COPD were excluded. Participants underwent lung function assessment (spirometry, static lung volumes, and diffusing lung capacity for CO [DLCO]), fractional exhaled nitic oxide (FeNO) measurements), blood biomarker assessment, bronchoscopy with bronchoalveolar lavage for microbiological evaluation, and disease characterization using exacerbation history, Bronchiectasis Severity Index (BSI), FACED score and St George's Respiratory Questionnaire (SGRQ). Patients were stratified as T2-high (blood eosinophil counts [BEC] ≥300 cells/μL and/or FeNO ≥25 ppb) or T2-low. Non-parametric statistics were used for group comparisons and Spearman coefficients for correlations.

Results Fourteen patients (mean age 60.5±17.7 years, 12[86%] female) were analysed. Mean BEC was 169±135 cells/μL and mean FeNO 15.9±10.5 ppb. Six patients were classified as T2-high and eight as T2-low. Median exacerbation frequency was 2/year (range 0–5); eight patients reported frequent (≥2/year) exacerbations, which were associated with lower FeNO and FVC values (both $p<0.05$). Two patients presented FEV1/FVC<0.7, mean FEV1 was 2.37±0.76L, mean FVC 3.19±1.02L and mean DLCO 73.3±16.6% predicted. Compared with T2-high patients, T2-low individuals demonstrated lower DLCO values (63.4±11.5 vs 85.8±13.8% predicted, $p=0.037$), whereas spirometric indices did not differ significantly. Although between-group differences were not statistically significant, T2-low patients showed numerically greater disease burden, with higher BSI (6.75±2.6 vs 4±2.1) and FACED (2.12±0.64 vs 1.17±1.33) scores, while lower BEC were associated with higher FACED scores ($p<0.005$). *P. aeruginosa* isolation was reported in four patients (29%) and occurred more frequently in T2-low compared with T2-high patients (38% vs 17%).

Conclusion In this exploratory prospectively recruited cohort, lower T2 biomarker expression was associated with signals of impaired gas transfer, greater disease burden and increased microbiological burden. Prospective recruitment is ongoing to further characterize inflammatory heterogeneity in bronchiectasis, and the final data will be presented at the congress.

P170 THE DIAGNOSIS DETECTIVES: IMPROVED RECOGNITION OF AETIOLOGY IN BRONCHIECTASIS THROUGH A REGIONAL DIAGNOSTIC CLINIC

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Overview ERS bronchiectasis guidelines emphasise the importance of aetiological diagnosis. Despite this, EMBARC data suggests around 40% of cases are classed as idiopathic. As a regional centre for Cystic Fibrosis (CF), Primary Ciliary Dyskinesia (PCD) and non-CF bronchiectasis, we developed a diagnostic service for younger patients or those with suspicion of multi-system disease.

Methods We reviewed referrals since service initiation in 2023. Diagnostic assessment includes clinical review, blood and respiratory sample testing, lung function, nasal and lower airway FeNO, radiology, sweat chloride testing (SCC), and genetic testing (CF50, extended CFTR genotyping and the R189 ciliopathy panel) where indicated, alongside same-day multiprofessional support. Virtual or in-person follow-up to review results and determine ongoing management is also scheduled.

Results Forty-nine individuals have attended. Most referrals came from secondary care respiratory services (33/49, 67%), with over half from our local health board (27/49, 55%). Bronchiectasis was confirmed on new imaging or re-review of previous in 37/49 (76%), with a likely aetiology identified in 30/37 (81%). Treatment changes followed review in 34/49 (78%).

Altered CFTR function, indicated by raised SCC and/or CFTR variants, was common. One patient received a late diagnosis of CF (now trialling CFTR modulator therapy), 8 were diagnosed with CFTR-related disorders, and 3 had persistently elevated SCC (≥2 abnormal results) without meeting criteria for diagnosis with CF or CFTR-related disorder.

Coexisting airways disease was frequent. Asthma was diagnosed or confirmed in 11/37 (30%) of those with bronchiectasis. Asthma therapy was escalated in 10/11 (91%), and 3/5 meeting NICE criteria for biologic therapy are trialling this following asthma MDT discussion.

Onward referral to a national centre resulted in two new PCD diagnoses.

Abstract P170 Table 1 Characteristics and results of clinic assessments

	(n)(%)
Total	49 (100%)
Female	28 (57%)
Mean age (range)	41.7 (18-90)
Mean FEV_1 (%pred)	82
Ps. Aeruginosa	8 (16%)
CFTR Variants	13 (27%)
SCC >30mmol/L	15 (31%)
Nasal FeNO <200ppb	5 (10%)

Conclusion This service reduced diagnostic uncertainty, identified clinically relevant comorbidity, and through taking a cross-respiratory approach, drove treatment change including access to specialist treatment such as biologics.

Specialist diagnostic services may reduce 'idiopathic' bronchiectasis labels and reveal CFTR dysfunction as a potential treatable trait.

Future priorities are improving equitable access for patients living further from our centre and strengthening links with newborn screening to identify referrals with CF-SPID as a further potential marker of CFTR dysfunction.

P171 REVIEW OF THE MANAGEMENT PLAN OF NEW PATIENTS SEEN IN THE BRONCHIECTASIS CLINIC: A RETROSPECTIVE OBSERVATIONAL STUDY

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10.1136/thorax-2026-BTSabstracts.331

Background Outpatient management of bronchiectasis varies, and data on adherence to guideline-based advice are limited.
Methods We performed a retrospective observational study of 35 new patients attending a bronchiectasis clinic. Patient demographics, clinical advice, and interventions were recorded and compared with european data.
Results Of 35 patients, 18 (51.4%) were females; median age was 75 years (range 18–97). One patient did not attend the clinic. Previous sputum microbiology was reviewed for all patients. 30 patients (85.7%) received antibiotic advice and prescriptions, 32 (91.4%) were reviewed by chest physiotherapists, 11 (31.4%) received smoking cessation advice, whereas 11 (31.4%) were non-smokers, five (14.3%) received lifestyle modification advice, eight (22.9%) received vaccination advice, and 15 (42.9%) had further sputum microbiology arranged. No patient received a written bronchiectasis management plan. Median exacerbations were one; seven patients (20%) required hospitalisation in the subsequent year. By comparison, european data (EMBARC) reported median exacerbations of two and hospitalisation in 26.4% of patients.
Conclusion In this cohort, guideline-based outpatient management was variably implemented. Exacerbation rates and hospitalisations were lower than reported in EMBARC. This study highlighted areas for optimisation, particularly written management plans. We plan to introduce written bronchiectasis management plan for new patients, which will encompass all aspects of bronchiectasis management. After implementation, we will perform another analysis, and aim to reduce the rate of exacerbation and hospitalisations by a further 20%.

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P172 UNDERSTANDING BARRIERS TO RECRUITMENT IN BRONCHIECTASIS HUMAN CHALLENGE STUDY: A QUALITATIVE ANALYSIS

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Introduction and Objectives Bronchiectasis is the third most common chronic respiratory disease, affecting an estimated 1 in 200 adults in the United Kingdom.[1] It is characterised by chronic airway inflammation, recurrent infections, and acute exacerbations. Despite its importance, representative experimental models remain limited. Human infection challenge studies may improve understanding of disease mechanisms and accelerate therapy development. The Bronchiectasis and Rhinovirus Research to Investigate Exacerbation Responses (BARRIER) trial is the first human rhinovirus challenge study in bronchiectasis and investigates the immunopathogenesis of exacerbations. This analysis aimed to identify recruitment barriers and evaluate strategies introduced to improve enrolment.
Methods Recruitment data from July 2024 to June 2026 across three North London sites were reviewed. Screening and recruitment logs were analysed to identify reasons for declining participation, inter-site variation, and eligibility constraints. Descriptive statistics summarised recruitment outcomes and participant characteristics.
Results A total of 350 eligible patients were invited. Reluctance to participate was observed in 311(89%) individuals. The main barriers were time constraints (20%), concerns about deliberate viral inoculation (13%), reluctance to undergo bronchoscopy due to previous negative experiences or unfamiliarity (10%), and language barriers (5%). Around 40% of screened individuals were excluded due to positive rhinovirus serology. Among those completing screening, 11 of 17 participants identified as White British or White Other, and 72% were female, indicating limited representativeness.
Conclusion Recruitment to human challenge studies in bronchiectasis is challenging. Although pre-trial engagement suggested acceptability, concerns about viral inoculation, invasive procedures, and time commitments were key barriers. Underrepresentation of minority ethnic groups persisted, with language barriers explaining only part of this disparity. In response, a non-bronchoscopy arm was introduced, recruitment sites were expanded, and additional resources were allocated. Mapping recruitment against deprivation indices is ongoing to identify underserved populations. Understanding barriers is essential to improve inclusivity, feasibility, and future therapeutic development for bronchiectasis.

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P173 BRONCHIECTASIS DOESN'T COME WITH A USER MANUAL - DO INTERDISCIPLINARY CONVERSATIONS IMPROVE DECISION MAKING?

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The prevalence of bronchiectasis is rising - in 2019 it was estimated that there were 212,000 current patients in the United Kingdom.[1] Antimicrobial decision-making in bronchiectasis continues to pose problems in the context of a multitude of organisms, difficulties in diagnosing acute exacerbations and burgeoning resistance. Consequently, there are often difficulties in maintaining a unified approach.

In February 2025, this NHS trust established a regular bronchiectasis/microbiology multidisciplinary team (MDT) meeting with the objectives of improving both stewardship and patient outcomes. Between February 2025 and May 2026, 54 patients were discussed across a total of 11 MDTs. MDT attendees included respiratory and microbiology physicians, along with bronchiectasis physiotherapists. MDT outcomes were reviewed retrospectively to assess whether discussions influenced decision-making.

Outcomes measured:

- The number of patients for whom there was a meaningful change in plan, as defined by the reviewing clinician
- The number of patients for whom advice on acute or future antimicrobials was provided

- The number of suggested antimicrobial courses that were concordant with guidelines (NHS Tayside Bronchiectasis Antimicrobial Guidance)
- The number of suggested courses of intravenous antimicrobials including: inpatient-mediated or Outpatient Parenteral Antimicrobial Therapy (OPAT)

Of those discussed, 75.93% (N=41) had a meaningful change to their ongoing management. 62.97% (N=34) of MDT outcomes included advice on acute or future courses of antimicrobials - of those, 76.47% (N=26) of antimicrobials were guideline concordant. 34.62% (N=9) of suggested antimicrobials were intravenous (inpatient or OPAT).

These results suggest that a regular MDT offers an opportunity to improve patient care. Objectively assessing guideline concordance is difficult and discordance is often attributable to nuanced patient factors, specific sensitivities or multi-organism infections. Future work is needed to assess whether the introduction of this MDT improves stewardship and patient outcomes and reduces exacerbations in the longer-term. Additionally, we wish to review whether providing a single point of access improves the patient journey through OPAT services.

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P174 FEASIBILITY AND ACCEPTABILITY OF USING A SHARED DECISION MAKING INTERVENTION TO SUPPORT PATIENT CHOICE OF AIRWAY CLEARANCE TECHNIQUES IN BRONCHIECTASIS

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10.1136/thorax-2026-BTSabstracts.334

Background Bronchiectasis is a chronic lung condition with symptoms including chronic productive cough, mucus retention and recurrent chest infections. Airway clearance techniques (ACT) are a guideline recommended treatment in bronchiectasis. There are several different ACTs with limited evidence supporting the efficacy of one ACT over another. Adherence to ACTs is poor (~30-50%). Shared decision making (SDM) is where clinicians and patients make personalised treatment decisions together, using the best available evidence. SDM can improve adherence to treatment in chronic conditions. SDM to support patient choice is emphasised in bronchiectasis guidelines.

Aims To test the feasibility and acceptability of using a SDM intervention to support patient choice of ACTs in bronchiectasis physiotherapy consultations.

Methods We conducted a two-centre prospective feasibility study. The SDM intervention consisted of a patient decision aid and SDM training package. Study duration was 12 weeks. Consecutive sampling using bronchiectasis clinic lists were used. Feasibility outcomes included recruitment and retention. Interventional outcomes included decisional conflict score (DCS), decisional regret score (DRS) and shared decision making _questionnaire (SDM-Q-9). Acceptability outcomes included the theoretical framework of acceptability questionnaires (TFAQ-physiotherapist and TFAQ-patient). Additional outcomes included self-reported adherence via weekly text message and quality of life-bronchiectasis (QoL-B) scores.

Results Thirty patients were recruited and 27 (90%) completed the study. Study outcomes are displayed in table 1. DCS and DRS median scores were 17/80 and 7/25 respectively indicating low decision conflict and regret. The SDM-Q-9 median score was 100 indicating high perceived levels of SDM within consultations. TFAQ-patient and physiotherapist mean acceptability scores were 35.47 and 38.33 respectively, indicating high levels of acceptability. 218/221 (99%) text message responses from 30 patients reported they were adherent to their ACT.

Abstract P174 Table 1 Characteristics and results of clinic assessments

Outcome	Mean (SD)	Mean 95% CI	Median (IRQ)	Median 95% CI	
DCS			17 (3.5)	17.51-21.56	
DRS			7 (5)	(5-9.5)	
SDM-Q-9			100 (2)	90.91-100	
TFAQ-patient	35.47 (4.11)	34.00-36.94			
TFAQ-physiotherapist	38.33 (1.53)	34.53-40.00			
Outcome		**Study visit 1 (n=30)**	**Study visit 2 (n=27)**	**Study visit 3 (n=25)**	**MCID scores (difference in mean scores between visit 1 and visit 3)**
QoL-B domain	Respiratory	58.30 (16.74)	64.59 (15.90)	54.69 (18.62)	9.90*
	Physical Functioning	53.09 (27.45)	57.10 (28.01)	49.11 (29.16)	7.99
	Health perceptions	40.43 (16.46)	45 (16.32)	38.33 (21.40)	6.67
	Vitality	38.27 (16.97)	47.11 (14.08)	34.81 (23.66)	12.30*
	Emotional functioning	74.38 (20.92)	76.33 (19.94)	70.83 (25.50)	5.50
	Treatment burden	74.22 (17.19)	68.12 (22.30)	69.59 (22.78)	-1.47
	Social functioning	50 (26.45)	51 (26.06)	49.72 (24.51)	1.28
	Role functioning	56.79 (17.18)	58.67 (20.83)	55.78 (28.20)	2.89

Categorical data are summarised as absolute numbers or frequencies and percentage rates. Continuous data are presented as mean (SD) if normally distributed, and median (IRQ) if not normally distributed as determined using the Shapiro-Wilk test.
*Met the minimally clinical important difference for the specific QoL-B domain; DCS: decision conflict score (scores range from 16-80. A lower score indicates a lower decisional conflict); DRS: decision regret score (scores range from 1-25. A lower score indicates lower decisional regret); SDM-Q-9: shared decision making questionnaire for patients (scores range from 0-100. A higher score indicates a higher perceived level of shared decision making); TFAQ: theoretical framework of acceptability questionnaires (scores range from 5-40. A higher score indicates a higher level of acceptability); QoL-B: quality of life-bronchiectasis score (a higher score indicates a higher quality of life)

Discussion This is the first study testing a SDM intervention to support patient choice of ACTs in bronchiectasis. Data suggests the intervention was feasible to deliver and is acceptable to patients and physiotherapists. There are also signals in the data that SDM supports high adherence to ACTs in bronchiectasis.
Conclusion Study data suggests it may be feasible to conduct a definitive trial using the SDM intervention, with some iterations to the intervention and study methods.

P175 AIRWAY CLEARANCE THERAPY SWITCHING PATTERNS IN PRIMARY CILIARY DYSKINESIA: A TERTIARY CENTER COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.335

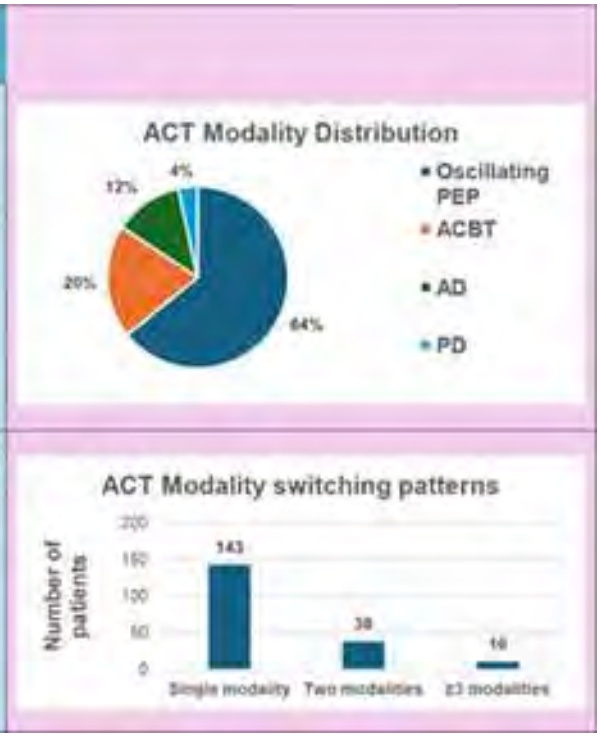
Background Airway clearance therapy (ACT) in Primary Ciliary Dyskinesia (PCD) is often adapted over time in response to symptoms, exacerbations, physiotherapy review, and patient preference. However, field phenomenon of ACT modality switching have not been well described. Understanding how and when patients transition between techniques can inform personalised physiotherapy pathways. We evaluated ACT switching behaviour over 12 months in a large tertiary PCD cohort.
Methods We retrospectively analysed all ACT entries for 191 PCD patients reviewed between May 2025 and April 2026 to identify modality switching, multimodality use, and temporal patterns. ACT modalities were grouped as oscillating positive expiratory pressure (oscillating PEP; Acapella/Flutter), Active Cycle of Breathing Technique (ACBT), Autogenic Drainage (AD), and Postural Drainage (PD).
Results Modality switching was highly prevalent. A large proportion of patients had two or more ACT modalities recorded across the year. Oscillating PEP remained the most common starting modality ($p < 0.001$), with many patients transitioning to ACBT or AD during periods of clinical instability. The dataset contains numerous examples of sequential modality use (e.g., *'Oscillating PEP (acapella) physiotherapy'* followed by *'ACBT'* or *'AD'*), indicating dynamic physiotherapy adaptation. Table 1 shows the ACT modality and switching statistics.

Switching patterns fell into three broad groups:

1. Stable singlemodality users — predominantly oscillating PEP, with consistent use throughout the year.
2. Intermittent switchers — alternating between oscillating PEP and ACBT, often around exacerbationrelated FEV_1 changes.
3. Highcomplexity switchers — using ≥3 modalities (e.g., oscillating PEP → ACBT → AD), typically associated with lower lung function or physiotherapyled optimisation.

A minority of patients had no recorded modality, reflecting either stable disease or limited documentation. Temporal clustering of switching was observed around winter months, consistent with seasonal exacerbation patterns.
Conclusions ACT modality switching is common in PCD care and reflects the dynamic nature of physiotherapy management. Oscillating PEP remains the dominant baseline technique, but transitions to ACBT and AD are frequent, particularly in patients with greater clinical complexity. These findings highlight the need for flexible, personalised ACT pathways and support the integration of switching behaviour into physiotherapy assessment and service design.

Category	Subgroup	Patients (n)	Percentage (%)	Statistical Significance
ACT Modality Distribution	Oscillating PEP	168	88.0%	Chi-square comparison across modalities show preference for Oscillating PEP χ^2 = 142.6, p < 0.001
	ACBT	53	27.7%	
	AD	32	16.8%	
	PD	9	4.7%	
ACT Switching Behaviour	Single modality users	143	74.9%	Preference for single-modality use is statistically significant at χ^2 = 154.44, p < 0.0001
	Two modality users	38	19.9%	
	≥3 modality users	10	5.2%	



Abstract P175 Table 1 Airway clearance modality and switching statistics in primary ciliary dyskinesia

P176 NEBULISED THERAPY AS AN INDEPENDENT DRIVER OF HIGH INTENSITY AIRWAY CLEARANCE IN BRONCHIECTASIS

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10.1136/thorax-2026-BTSabstracts.336

Background The 2025 European Respiratory Society bronchiectasis guideline recommends universal airway clearance training and selective use of nebulised therapy for patients with frequent exacerbations or chronic infection. In routine practice, however, the relationship between nebulised therapy and the intensity of airway clearance techniques (ACTs) is not well described. It is unclear whether nebulised therapy itself contributes to higher physiotherapy burden, or whether this simply reflects underlying disease severity. We examined this association to identify a practical, measurable hightreatmentburden phenotype within the treatabletraits framework.
Methods We analysed longitudinal data from 460 adults with bronchiectasis attending a UK tertiary service. Variables included FEV_1 % predicted, annualised exacerbation frequency, intravenous (IV) antibiotic exposure, ACT frequency (sessions/week), ACT modality, and use of nebulised therapy (hypertonic saline or nebulised antibiotics). Highintensity ACT was defined as ≥14 sessions per week. Multivariable logistic regression assessed whether nebulised therapy independently predicted highintensity ACT after adjustment for age, sex, lung function, exacerbation frequency, and IV antibiotic exposure.
Results Patients using nebulised therapy had more severe disease than nonusers, with lower mean FEV_1 (52% vs 66% predicted), higher median exacerbation frequency (3 vs 1 per year), and greater IV antibiotic exposure (48% vs 21%) (all $p<0.001$). Highintensity ACT was performed by 62% of nebulisedtherapy users compared with 24% of nonusers ($p<0.001$).

In adjusted analysis, nebulised therapy remained independently associated with highintensity ACT (OR 2.3, 95% CI 1.5–3.6, $p<0.001$). Lower FEV_1 (per 10% decrease: OR 1.4, $p=0.002$), higher exacerbation frequency (per exacerbation/year: OR 1.3, $p=0.004$), and IV antibiotic exposure (OR 1.9, $p=0.006$) were also independently associated.

A hightreatmentburden phenotype—defined as nebulised antibiotic use plus ≥14 ACT sessions/week—accounted for 18% of the cohort and had the lowest mean FEV_1 (45% predicted) and highest IV antibiotic exposure (71%).
Conclusions Nebulised therapy is independently linked to highintensity physiotherapy, even after accounting for objective markers of severity. This identifies a clear hightreatmentburden phenotype and supports the need for integrated physiotherapy–nebuliser pathways and prospective evaluation of treatment burden in bronchiectasis care.

P177 THE IMPACT OF DOMICILIARY HIGH-FLOW NASAL OXYGEN THERAPY IN ADULTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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10.1136/thorax-2026-BTSabstracts.337

Introduction and Objectives Bronchiectasis is a chronic progressive respiratory disease characterised by impaired mucociliary clearance and recurrent exacerbations, imposing a significant burden on patients and healthcare systems. High-flow nasal therapy (HFNT), which delivers heated and humidified air at high flow rates, has been proposed to improve mucociliary clearance and reduce exacerbation frequency in this population, although its effectiveness remains unclear. This systematic review aimed to evaluate the impact of long-term domiciliary HFNT in adults with non-cystic fibrosis bronchiectasis.
Methods A systematic search of MEDLINE, Embase, CENTRAL, SCOPUS, Web of Science and CINAHL was conducted up to February 2026 in accordance with PRISMA 2020 guidelines. Eligible studies included randomised controlled trials (RCT) and observational cohort studies evaluating domiciliary HFNT in adults aged 18 years or older with radiologically confirmed non-cystic fibrosis bronchiectasis reporting at least one pre-specified clinical outcome. Risk of bias was assessed using RoB 2 for randomised studies and ROBINS-I for non-randomised studies. A random-effects meta-analysis was performed for exacerbation rate where comparative data were available.
Results Seven studies (n=303 participants) were included. Meta-analysis of three comparative studies demonstrated a significant reduction in exacerbation rate with HFNT compared with standard care (pooled rate ratio 0.53, 95% CI 0.34–0.84; $p = 0.007$), though substantial heterogeneity was observed ($I^2=85\%$). Reductions in hospitalisation rates, improvements in health-related quality of life and symptom burden were also reported. Lung function improvements were inconsistent and physiological studies provided supporting mechanistic evidence.
Conclusions This review demonstrates that domiciliary HFNT is associated with reduced exacerbation frequency and hospitalisation rates in non-cystic fibrosis bronchiectasis, supporting its potential as an adjunctive therapy in this population. However, the evidence base remains limited by observational study designs and substantial heterogeneity. Large, well-designed randomised controlled trials are needed to confirm these findings and define the optimal role of HFNT in bronchiectasis management.

P178 OUTCOMES OF IV ANTIBIOTIC MONOTHERAPY FOR PSEUDOMONAS AERUGINOSA IN BRONCHIECTASIS: A 10-YEAR OPAT COHORT

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10.1136/thorax-2026-BTSabstracts.338

Introduction Pseudomonas aeruginosa (PsA) infection in bronchiectasis is associated with increased exacerbations, antimicrobial resistance and mortality. Guidelines support use of IV antibiotics for PsA eradication of new isolates and management of severe exacerbations. However, longitudinal clinical and microbiological outcomes following IV antibiotic monotherapy are poorly described.
Methods We conducted a single-centre retrospective cohort study of adult bronchiectasis patients treated with IV antibiotic monotherapy via a respiratory outpatient parenteral antibiotic therapy (OPAT) service between December 2015 and May 2025. Patients were classified as PsA 'colonised' (≥1 positive sputum

culture ≥1 year before initial IV therapy) or 'new isolate'. Longitudinal clinical (further IV-treated exacerbations, time to next IV, mortality) and microbiological outcomes (PsA eradication and antimicrobial resistance) were assessed using data collected from electronic medical and microbiology records.

Results 115 patients were included: 67 with new isolates (41.8% male, mean age 67 years) and 48 colonised (35.4% male, mean age 71 years). More colonised patients required subsequent IV antibiotics than new isolates (60.4% vs 52.0%), with similar time to next IV antibiotic (15.0 vs 14.2 months). Kaplan–Meier analysis showed >30% require further IV antibiotics by 1 year, >50% by 2 years and ~70% by 5 years following their initial OPAT. Post-OPAT exacerbations requiring IV antibiotics were lower in colonised patients compared to new isolates (0.70 vs 1.18 per year), as was mortality (14% vs 19%).

PsA eradication (no further PsA positive sputum and ≥1 negative sputum) occurred in 21.5% of patients with new isolates. Rates of increased antimicrobial resistance following OPAT were similar between those with new and those with colonised isolates: ciprofloxacin 24.5% vs 33.3%, meropenem 10.2% vs 10.3% and piperacillin–tazobactam 18.4% vs 20.5%.

Conclusion IV antipseudomonal monotherapy achieves modest PsA eradication, without significantly impacting antimicrobial resistance profiles. It effectively provides patients with exacerbation free time. Further research from centres using alternative strategies such as dual IV antibiotics would help contextualise these clinical and microbiological findings.

P179 SERUM AZUROCIDIN-1 IS ASSOCIATED WITH FREQUENT EXACERBATIONS IN PATIENTS WITH BRONCHIECTASIS: PRELIMINARY DATA FROM THE EMBARC-BRIDGE STUDY

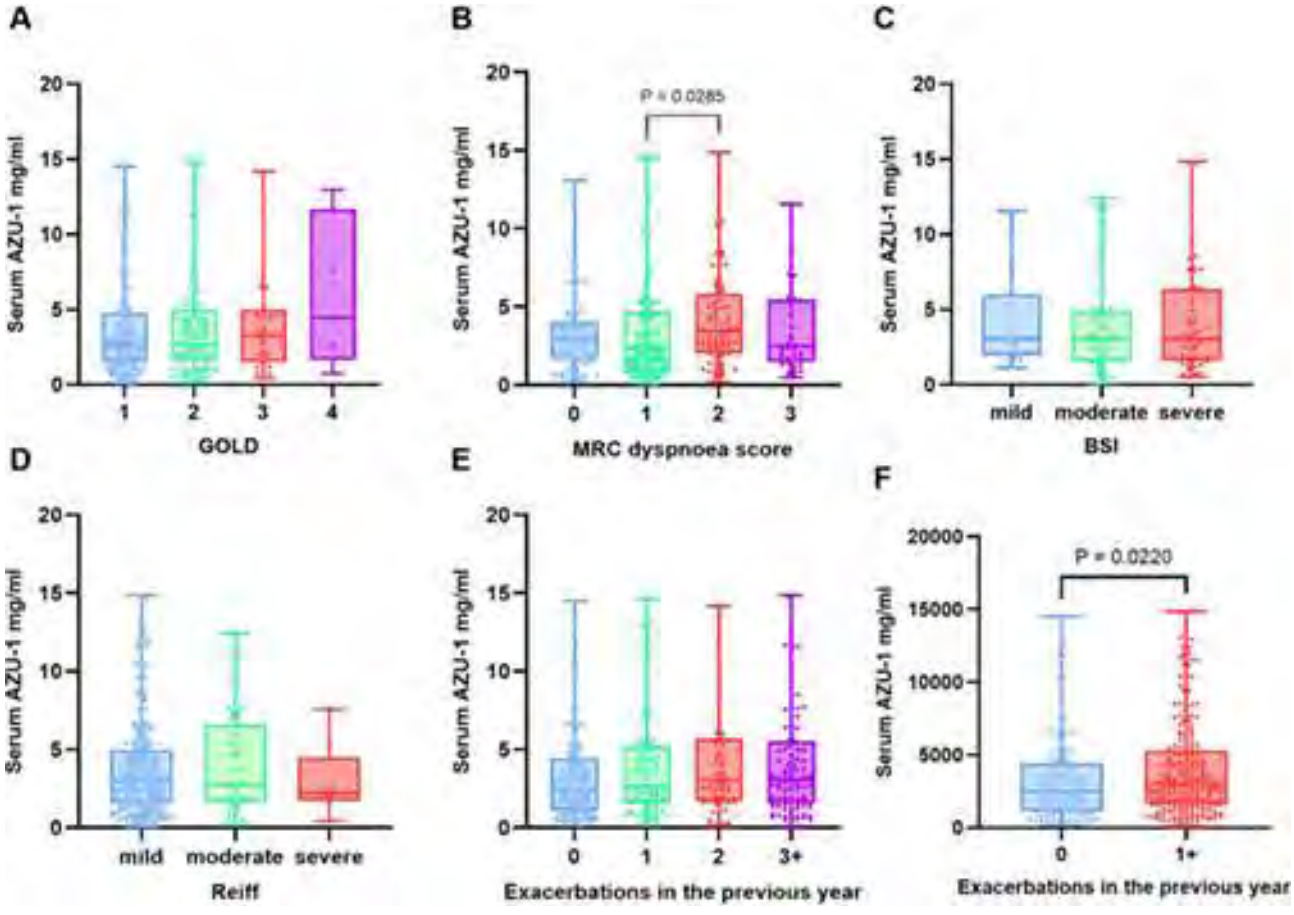
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10.1136/thorax-2026-BTSabstracts.339

Background Recent studies have shown that azurocidin-1 (AZU-1) in sputum is strongly associated with bronchiectasis severity, lung function impairment, bacterial load, and exacerbation frequency, suggesting an important role in airway inflammatory pathology. Azurocidin-1 is a target of DPP-1 inhibition which works systemically, and therefore the implications of systemic AZU-1 are also clinically important.

Objective To investigate the association between serum AZU-1 levels and clinical characteristics, disease severity, lung function, and exacerbation phenotype in a large cohort of patients with bronchiectasis.

Methods We conducted a cross-sectional study including 387 patients with bronchiectasis enrolled in the pan European EMBARC-BRIDGE cohort study. Serum AZU-1 was measured using ELISA. Clinical variables included age, sex, BMI, lung function (FEV_1 and FVC), bronchiectasis severity (BSI and Reiff), quality of life (QoL-B), MRC dyspnoea score, sputum volume, and exacerbation history in the previous year. Associations were



Abstract P179 Figure 1

assessed using Spearman correlation, Mann–Whitney U test, Kruskal–Wallis test, ANOVA, and ROC analysis.
Results Serum AZU-1 was not associated with age, BMI, lung function, bronchiectasis severity (BSI and Reiff), quality of life, sputum volume, or disease duration (all $P > 0.05$). A weak positive correlation was observed between serum AZU-1 and MRC dyspnoea score ($r = 0.138$, $P = 0.006$), although no consistent differences were observed across severity groups. Nevertheless patients with exacerbations (≥1 exacerbation in the previous year) had significantly higher serum AZU-1 levels than those without (3013.4 vs 2511.9 ng/mL, $P = 0.022$). Serum AZU1 was significantly correlated with annual exacerbation frequency ($p=0.042$).
Conclusion Serum AZU1 correlates with exacerbation frequency in clinically stable patients with bronchiectasis. Although serum AZU1 demonstrates limited value as a standalone biomarker this suggests a potential role for systemic neutrophils in exacerbation risk.

P180 LIVEBE: LIVING LIFE WITH BRONCHIECTASIS - RESULTS FROM A PATIENT SURVEY ON HOW BRONCHIECTASIS AFFECTS PATIENT LIVES

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10.1136/thorax-2026-BTSabstracts.340

Background/Aims LiveBE set out to improve our understanding of how the lives of people with bronchiectasis are changed by their disease.
Methods This was carried out via a predominately quantitative questionnaire. It was divided into sections including: basic demographics, time to diagnosis and current symptom burden via the CAAT score; how living with bronchiectasis affects home life, outdoor life, social and relaxation, mental health, the health professional relationship, work and non-respiratory symptoms. The questionnaire was designed by clinicians working with patients with bronchiectasis and obtained significant patient input from 3 patients. We obtained ethical consent from the University of Dundee.
Results We report preliminary results from 98 patients with an average age of 72 years; 62% were female and 38% male. 41% of those questioned reported symptoms for over 10 years yet only 22% of those had a diagnosis for over 10 years. 69% of patients reported they felt they had control of their bronchiectasis 'all' or 'most' of the time, 32% felt their ability to control their bronchiectasis had improved over time. 48% had changed their social activities; the main reason stated was 'because symptoms made the activity difficult'. 73% of patients reported that they intentionally exercised to increase their heart rate with 70% of this group finding this had a positive impact on their bronchiectasis. 30% reported their bronchiectasis had impacted their mental health with 'anxiety- future worry' the most commonly reported. 44 of our patients reported some level of urinary incontinence; 33 were female and 11 male, similar to the overall split. Both stress and urge incontinence symptoms were reported. Of these only 40% have discussed this with a medical professional and 30% have been offered treatment. We will report on the link between severity symptom burden via the CAAT score and additional reported symptom burden such as future anxiety, perceived disease control and urinary incontinence.
Conclusions Key outcomes for further exploration include how to help patients feel more in control of their disease; how to support and promote healthy exercise opportunities and how to help normalise symptoms and medical treatment for urinary incontinence.

Innovation across the lung cancer diagnostic pathway

P181 CLINICAL IMPACT OF THORACIC RADIOLOGIST INPUT IN A LUNG NODULE VIRTUAL CLINIC: IMPROVING TRIAGE AND PATIENT OUTCOMES

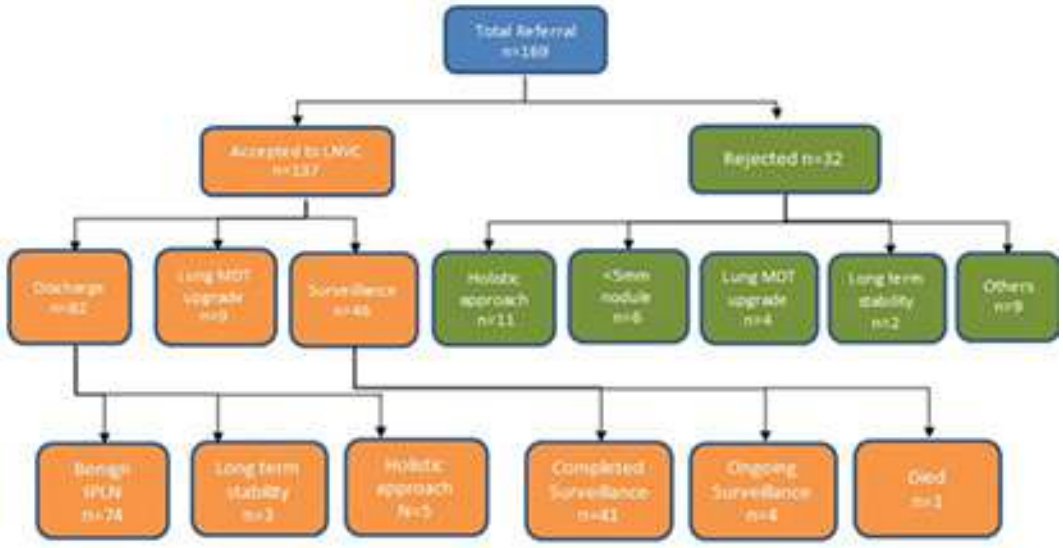
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10.1136/thorax-2026-BTSabstracts.341

Background CT activity increased by 60% 2016/17 to 2023/24 resulting in increased solitary pulmonary nodule (SPN) detection. While most SPNs are benign, some represent early, treatable lung cancer. Many thoracic CT scans are reported by non-thoracic radiologists or outsourced services, contributing to variability in interpretation. In 2022, a dedicated radiology SPN referral code linked to a referral mailbox was introduced at a tertiary teaching hospital to streamline access to the Lung Nodule Virtual Clinic (LNVC), led by respiratory physicians and thoracic radiologists. We evaluated the referrals to assess triage and outcomes.
Methods We retrospectively reviewed all referrals to the LNVC mailbox January to December 2022. Referral sources included internal referrals and primary care clinicians. All referrals were triaged by a respiratory physician; accepted cases underwent joint review with a thoracic radiologist in LNVC. Demographic, nodule characteristics and management outcomes were analysed.
Results 169 referrals were received: 136 internal, 33 primary care. Median age was 70 years; 82 male and 87 female. Most nodules were solid (n=136), with n=32 part solid. WHO performance status was documented in 24 cases.

Respiratory clinicians triage accepted 137 referrals with 32 referrals rejected due to poor performance status or comorbidities (n=11), nodules <5 mm (n=6), long-term stability (n=2), escalation to lung MDT (n=4), and other reasons (n=9), including no discrete nodule identified, resolved changes, and infection-related (TB).

Of accepted cases, 82 (59.85%) were discharged after first LNVC review, predominantly due to being characterised as benign intrapulmonary lymph nodes (n=74), long-term stability (n=3), and holistic approach (n=5). No delayed cancer diagnoses occurred among discharged cases. Nine were upgraded to the lung cancer pathway and 46 (33.57%) entered BTS-guided surveillance; 41 completed follow-up and were discharged, four remain under surveillance, and one died.
Conclusion A dedicated lung nodule radiology code and mailbox, linked to LNVC supported by thoracic radiologists, enables effective triage based on imaging characteristics and patient factors. This streamlines care by reducing unnecessary investigations and hospital visits, while alleviating pressure on lung cancer services without compromising safety. The model



Abstract P181 Figure 1 Breakdown of LNVCreferrals

demonstrates the value of specialist radiologist input and virtual clinics in optimising resource utilisation.

REFERENCE

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P182 ANTERIOR MEDIASTINAL MASSES IDENTIFIED THROUGH LUNG CANCER SCREENING: HOW SHOULD WE MEASURE, HOW SHOULD WE MANAGE AND WHEN TO REFER?

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Introduction and Objectives Anterior mediastinal masses (AMMs) detected incidentally during lung cancer screening (LCS), are commonly benign (e.g. thymic cysts) but occasionally are clinically significant (e.g. thymoma, thymic carcinoma). Advances in thoracic surgical techniques, e.g. robotic-assisted procedures, have made resection possible (and safer) for smaller thymomas, hence early referral is recommended. MRI can help differentiate between pathologies based on tissue characteristics, but spatial resolution is poor in small lesions. Guidelines suggest a 2cm threshold for MRI, but measurement planes are not standardised. We reviewed local AMM management to ensure compliance with recent guideline updates and to evaluate consistency of size reporting in respect to measurement planes.

Methods All anterior mediastinal masses identified since the start of the local LCS programme were reviewed. Initial

Abstract P182 Table 1 Summary of diagnostic and management outcomes

Initial SRM classification		Initial SRM size		Initial management		Ultimate diagnosis		Diagnostic basis
AMM, undetermined	70	>2cm	44	2WW referral	16	Thymic cancer	2	2 histology
				Enhanced surveillance	16	Thymoma	2	2 histology
				Routine LCS follow up	10	Cyst	14	10 MRI, 4 histology
						Other / unknown	26	1 histology, 1 PET-CT, 2 NM scan, 20 LDCT, 2 data unavailable
		<2cm	26	2WW referral	3	Thymic cancer	1	1 histology
				Enhanced surveillance	16	Thymoma	0	-
				Routine LCS follow up	7	Cyst	1	1 MRI
						Other / unknown	24	23 LDCT, 1 data not available
AMM, likely cyst	14	>2cm	11	2WW referral	1	Thymic cancer	0	-
				Enhanced surveillance	2	Thymoma	0	-
				Routine LCS follow up	8	Cyst	11	3 MRI, 8 LDCT
						Other / unknown	0	-
		<2cm	3	2WW referral	0	Thymic cancer	0	-
				Enhanced surveillance	0	Thymoma	0	-
				Routine LCS follow up	3	Cyst	3	3 LDCT
						Other / unknown	0	-

management and final diagnosis were reviewed. Imaging was reassessed by local radiologists to record size in axial and sagittal planes.

Results Between 01/08/2022 and 30/09/2025, 31,464 people underwent low dose computed tomography (LDCT) as part of the local LCS programme. 84 AMMs were identified (0.3% incidence). Mean age was 68 years with 37% female. Diagnostic and management outcomes are shown in table 1. In terms of additional imaging, 20 MRIs were requested, 7 via the screening review meeting (SRM), 13 via 2 week-wait (2WW) services (15 AMM >2cm, 2 AMM <2cm, 3 cyst >2cm). Of these, 15/20 confirmed thymic cysts. Additionally, 3 NM scans were requested via SRM and 2 PET-CTs via 2WW. Overall, we diagnosed 2 thymomas, 3 thymic carcinomas, 29 thymic cysts, 2 thyroid tissues, 1 thymic remnant and 47 undetermined anterior mediastinal masses (ongoing surveillance). Mean AMM size in axial plane was 24.6mm (range 8-80mm, median 21mm) compared with 32mm in sagittal plane (range 12-97mm, median 25mm).

Conclusions AMMs identified at LCS represent thymic cysts, thymomas and thymic malignancies. Further imaging, particularly with MRI, helps differentiate cases suitable for conservative management but is limited to lesions greater than 2cms. There is significant variation between measurements in the axial versus sagittal planes, and therefore reporting should be standardised for all AMM lesions. Our practice is to use the longest axis of measurement to determine management.

P183 EMERGENCY ADMISSIONS WITH LUNG CANCER – WHO AND WHY?

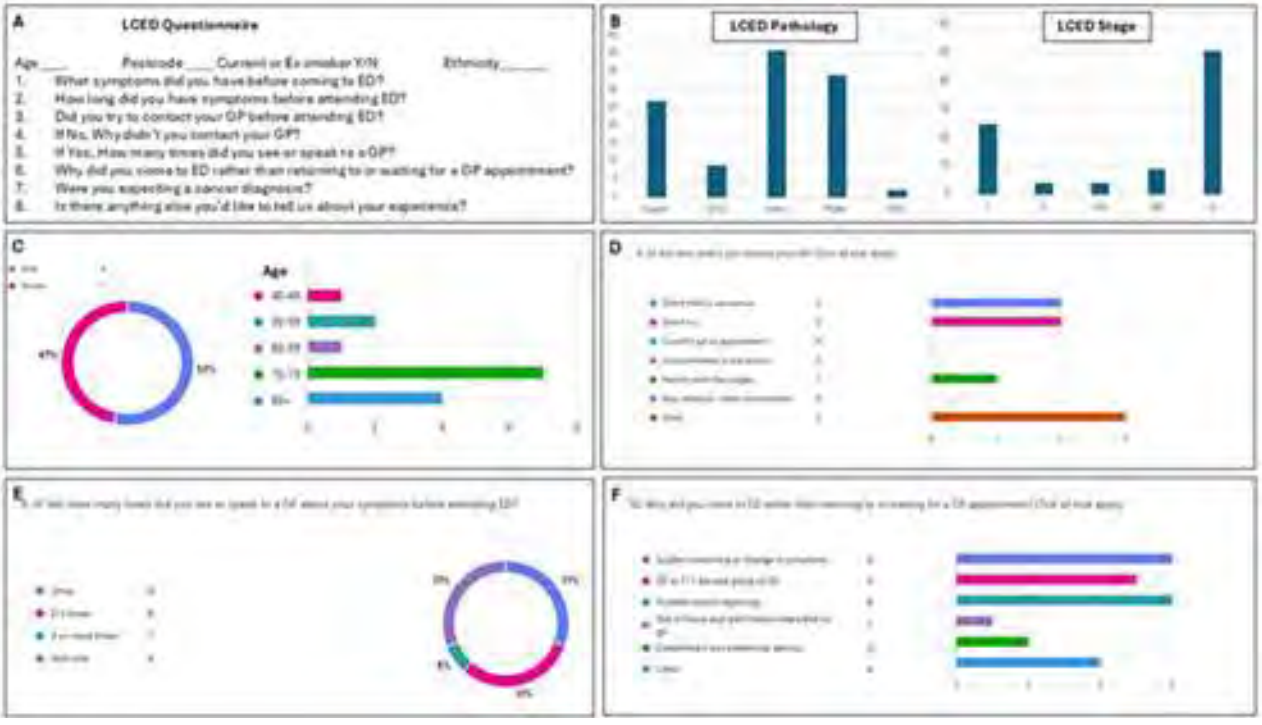
S Raza, K Verbeke, F Kon, J Ong, A Talwar, CL Tey, G Ghidoni, A Moore, JE Park. *Oxford University Hospital NHS Foundation Trust, Oxford, UK*

10.1136/thorax-2026-BTSabstracts.343

Introduction Despite Lung Cancer Screening (LCS), a significant percentage of lung cancer (LC) patients present at emergency admission (LCED) and have poorer outcomes[1]. Previously, for our population, 60% LCED were outside the LCS age, highlighting the need to understand symptomatic lung cancer presentation. We sought to further assess LCED and why patients present to the emergency department (ED).

Methods LC Patients diagnosed in 2024 were identified via Infoflex. Electronic patient records were reviewed to confirm a diagnosis of small cell or non small cell lung cancer and record admission route, postcode, performance status, smoking status, stage, pathology and whether the patient remained alive at review (September 2025). From April 2026 15 LCED patients were prospectively identified at lung MDT. Patients were contacted by telephone, asked about their presentation (figure 1A) and demographics collected.

Results 113 (25%) of 451 LC cases were LCED. Only 39 (34.5%) of LCED were current or ex-smokers aged 55-74 (range: 50-100 years). 43% of LCED patients lived in the 6 most deprived postcodes. 75% of the LCED group were dead at



Abstract P183 Figure 1 A. Questionnaire LCED patients. B. Pathology subtype and stage of lung cancer diagnosed at emergency presentation. C. Demographies of emergency admission patients questioned. D. Responses for not contacting primary care. E. Responses for frequency of contacting primary care. F. Responses for reason for attending emergency department

Abbreviations: ED - emergency department, LCED- lung cancer emergency admissions, Squam-Squamous cell, SCLC-small cell lung cancer, Adeno-adenocarcinoma, Radiol- radiological diagnosis, NOS- not otherwise specified.

time of review compared to 45% in the non-ED group. Tumour subtypes and stage are shown in figure 1B.

6 of 15 (40%) patients questioned after LCED were aged 55-74 (figure 1C) and 60% had tried to contact their GP prior to admission. Fatigue and general unwellness, weight loss and cough were the most common symptoms, with no patients expecting a diagnosis of cancer. 5 (33%) of patients questioned lived in the 2 most deprived postcodes. Figures 1D-F illustrate responses for interaction with primary care and reasons for attending ED.

Conclusion Locally LCED remains an important group, presenting at a higher stage, with poorer outcomes despite local roll out of LCS. LCED patients did not expect a diagnosis of cancer at presentation, did not think the symptoms were serious or did not try to contact primary care. These data, which we aim to build upon, show that there is a need for continued, targeted education around lung cancer and symptoms, especially in localities of highest deprivation.

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P184 DISCUSSING THE EFFECTIVENESS OF GEOGRAPHIC MAPPING ON GEOSPATIAL ANALYSIS OF LUNG CANCER IN BERKSHIRE

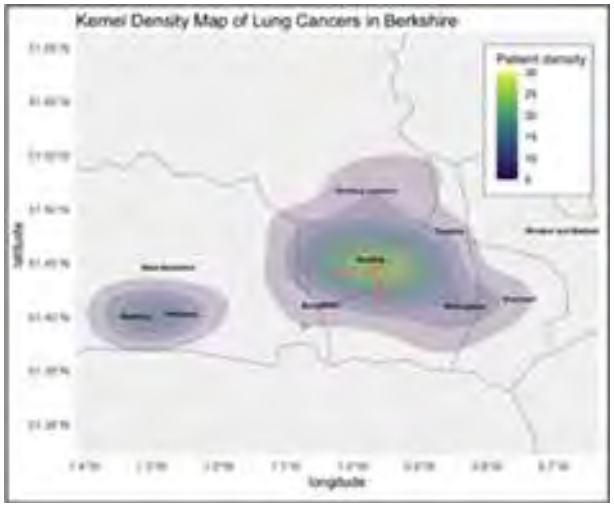
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10.1136/thorax-2026-BTSabstracts.344

Geospatial analysis is an effective method for understanding the geographic distribution of medical conditions. By understanding patterns behind the distribution of illnesses, researchers can identify areas with higher incidence, and better allocate resources to identifying and treating patients suffering from those conditions. Lung cancers are the leading cause of cancer deaths, accounting for 34,771 deaths in the United Kingdom between 2017 and 2019 alone.[1] Earlier lung cancer detection improves prognosis, and identifying high-incidence areas enables targeted screening and more efficient resource allocation. This study evaluates the effectiveness of geographic mapping of lung cancers in Berkshire, and its applicability to other medical conditions.

Patient data were obtained from a hospital's lung cancer referral list. Only cases with confirmed primary lung malignancies (n=314) were selected. Patient residential postcode data was converted into coordinates using R and the plugin 'Tidygeocoder'. The coordinate data was inserted onto the map of Berkshire and analysed using spatial and statistical packages. Point mapping, hexagonal binning (Hexbin), and kernel density estimation (KDE) was performed for spatial analysis.

Analysis using point mapping and Hexbin identified clusters of cases across Berkshire, with the most prominent located around Reading, Sonning Common, Wokingham, and Thatcham. KDE demonstrated that historical patterns of lung cancer distribution corresponded closely with newer case locations, with 11 of the 12 cases (91.7%) identified through screening falling within high-density regions generated through historical data. Cancer stage differed significantly by referral source (χ^2 = 12.1, p = 0.007; Cramér's V = 0.26), with emergency admissions demonstrating the highest proportion of late-stage (Stage III–IV) disease (82.1%), whereas incidental findings and secondary care referrals more commonly diagnosed early-stage (Stage I–II) disease (61.5% and 42.4%, respectively).



Abstract P184 Figure 1 Kernel density map of lung cancer cases, with new cases from screening represented by red points

Geospatial mapping identified clusters of lung cancer cases across Berkshire and may provide a useful approach for informing targeted screening and healthcare resource allocation. Similar methodologies could be explored for the spatial analysis of other diseases.

REFERENCE

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P185 SELF REQUEST CHEST X RAY PATHWAY: A LOW COST STRATEGY TO ACCELERATE LUNG CANCER DETECTION IN SYMPTOMATIC PATIENTS

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Background Lung cancer screening programmes focus on detecting early disease in asymptomatic highrisk populations. However, many patients continue to present symptomatically. A selfrequest chest Xray (CXR) pathway may provide a rapid, accessible diagnostic route; reduce barriers to care, and help address healthcare inequalities. Optimising patient engagement strategies is also crucial to maximise uptake of such services.

Method This pilot study was conducted across three GP practices in Coalville between November 2024 and November 2025. Patients aged ≥40 years with potential lung cancer symptoms were invited to selfrequest for CXR. A multimodal communication strategy was implemented, including SMS invitations, GP practice websites, posters, leaflets, and social media campaigns. Data collection included uptake, time intervals (referraltoimaging and imagingtoreport), clinical outcomes, and the effectiveness of communication methods.

Result A total of 355 patients accessed the pathway, with 324 CXRs performed (6.23 per week); 31 patients were ineligible. The mean time from referral to CXR was 4.65 days, and from CXR to report was 2.37 days.



Abstract P185 Figure 1

Suspicious findings for lung cancer were identified in 6 patients (1.85%), with 2 cases subsequently confirmed (0.62%). One patient remains under surveillance and one was unfit for further investigation. Additional findings included inflammatory or infective changes in 13 patients (4.01%) and one pneumothorax. The mean patient age was 62.03 years.

Over 30,000 SMS invitations yielded approximately 1% uptake. SMS messaging demonstrated the highest engagement compared to other methods. Younger individuals were more responsive to digital invitations, while uptake was relatively lower in older populations. A multimodal approach improved overall awareness but did not surpass SMS alone in effectiveness.

Conclusion A selfrequest CXR pathway is a feasible, effective model for early identification of lung cancer in symptomatic patients, enabling rapid access to diagnostic imaging while reducing reliance on primary care. Although uptake remains modest, SMSbased invitations are the most effective engagement strategy. Lower participation among older populations highlights the need for nondigital approaches to ensure equitable access. Future work should focus on scaling the pathway and improving integration within cancer referral systems to support earlier diagnosis and better resource use.

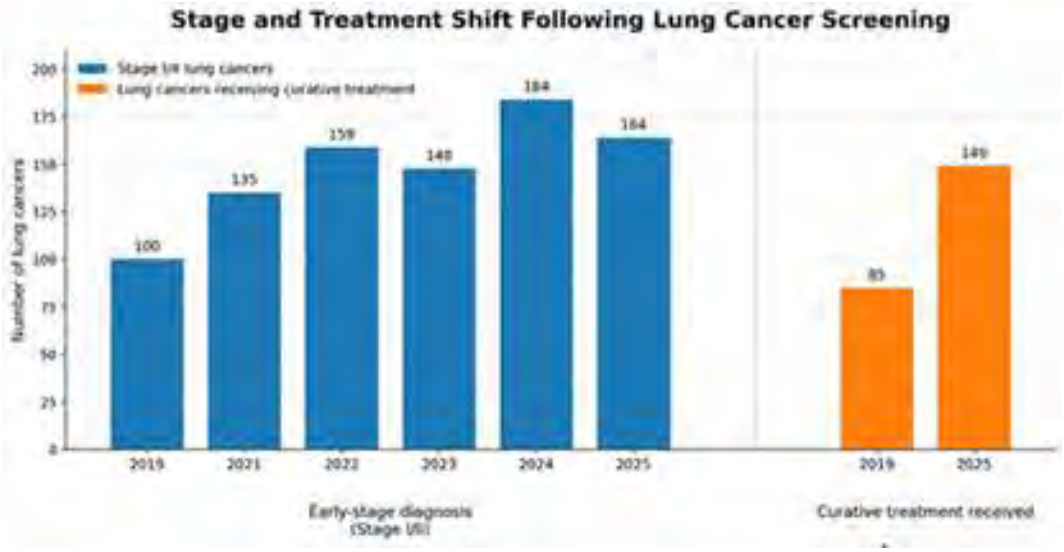
P186 FROM LATE DIAGNOSIS AND PALLIATION TO EARLY DETECTION AND CURE: THE SUSTAINED IMPACT OF A LUNG CANCER SCREENING PROGRAMME

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10.1136/thorax-2026-BTSabstracts.346

Introduction and Objectives Our hospital trust serves a population of around 445,000 people with high level of social deprivation, smoking prevalence, lung cancer incidence and mortality. In 2018, the area recorded England's lowest proportion of stage I/II lung cancer diagnoses at presentation, at 23%. The lung cancer screening programme (LCS) began as a pilot in one locality in 2021 and expanded to another in 2022, before becoming part of the current national screening programme.

We evaluated the effectiveness of LCS in improving early lung cancer detection and increasing curative treatment by analysing longitudinal trends in stage at diagnosis and treatment received.



Abstract P186 Figure 1

Methods Data were obtained from the Trust Lung Cancer Database and the LCS Database. Missing information was retrieved from clinic letters and MDT outcomes. Staging data from 2021 onwards were compared with 2019 as the pre-LCS, pre-COVID baseline. The contribution of LCS to early-stage diagnosis was analysed. Treatment received was compared between 2019 and 2025, after four years of programme implementation.
Results Early-stage lung cancer detection was 27% in 2019. This increased to 35% in 2021, 42% in 2022, 36% in 2023 and 43% in 2024, and 36.5% in 2025. To date, LCS has diagnosed 356 lung cancers, of which 280 (78.7%) were stage I/II and 279 (78.4%) received curative treatment.

The contribution of LCS to all stage I/II cancers was 20% in 2021, 54% in 2022, 39% in 2023, 23% in 2024 and 30% in 2025.The proportion of all patients receiving curative treatment increased from 23% in 2019 to 33.2% in 2025. In 2025, 165 patients, representing 36.7% of all lung cancers, were retrospectively identified as LCS-eligible but had missed the opportunity to participate because of non-registration, non-contact or non-engagement.
Conclusions Four years after implementation of LCS, the proportion of lung cancers detected at stage I/II increased by 9.7%, with a similar 10% increase in patients receiving curative treatment. This improvement has been sustained over five years compared with pre-LCS data. Improving registration data, contact processes and engagement is likely to sustain and strengthen this shift towards earlier diagnosis and curative treatment.

P187 INTERVAL AND INCIDENT LUNG CANCERS IN A LUNG CANCER SCREENING PROGRAM

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10.1136/thorax-2026-BTSabstracts.347

Background The Lung Cancer Screening program is been running in our local region since August 2022. To inform on planning and benchmark against national figures, we sought to define interval and incident lung cancers in the program at T1 (24 months) into the program after an index negative low-dose CT (LDCT).
Methods We reviewed our prospectively collected local dataset, and cross referenced with our local cancer registry data. The program commenced in August 2022, and completed baseline scans in the eligible screening population in November 2024. Interval scans were defined as T0+(3-15). Scans identified as T1 were performed 24 months following a normal T0 (index) LDCT.
Results A T0 LDCT scan was performed in 22,483 patients. During the index cycle (T0+x), 228 cancers were diagnosed at T0 (1.01%) and 116 cancers during interval imaging (T0+3-15).

Of these interval cancers most were diagnosed at the 3 month [n=61(53%)] or at 12 month [n=20(17%)] intervals. Stage I disease occurred in 97(84%) patients, while 7(6%) were stage IV. A shift in T stage occurred in 33 patients. A further 6 had nodal disease and 3 had metastatic disease.

For those completing a T1 scan, 35 lung cancers were diagnosed from 7,222 scans - an incidence of 0.48% (95%CI 0.34-0.64%). Stage I/II disease was present in 80% and 14% at Stage III/IV. In the those aged >70 years the incidence was 0.63% and accounted for 54% of incident (T1) lung cancers diagnosed. This compared to an incidence of 0.52% in the 55-59 age range, 0.24% in the 60-64 age group and 0.45% in the 65-69 age group. Compared with prevalent (T0) lung cancers, the stage distribution did not differ significantly: prevalent vs incident: 77%vs 80% Stage I/II and 20% vs 14% Stage III/IV; (p=0.44), but trended towards a reduction in late stage cancers.
Conclusion A significant proportion of interval scans show stage progression, and incident lung cancers after 24 months in the LCS program are approximately half of prevalent cancers. Earlier interventions and scanning cycles may be warranted to improve outcomes.

P188 ARE EARLY CANCER DETECTION AND CURE MISSED FROM THE LUNG CANCER SCREENING PROGRAMME?

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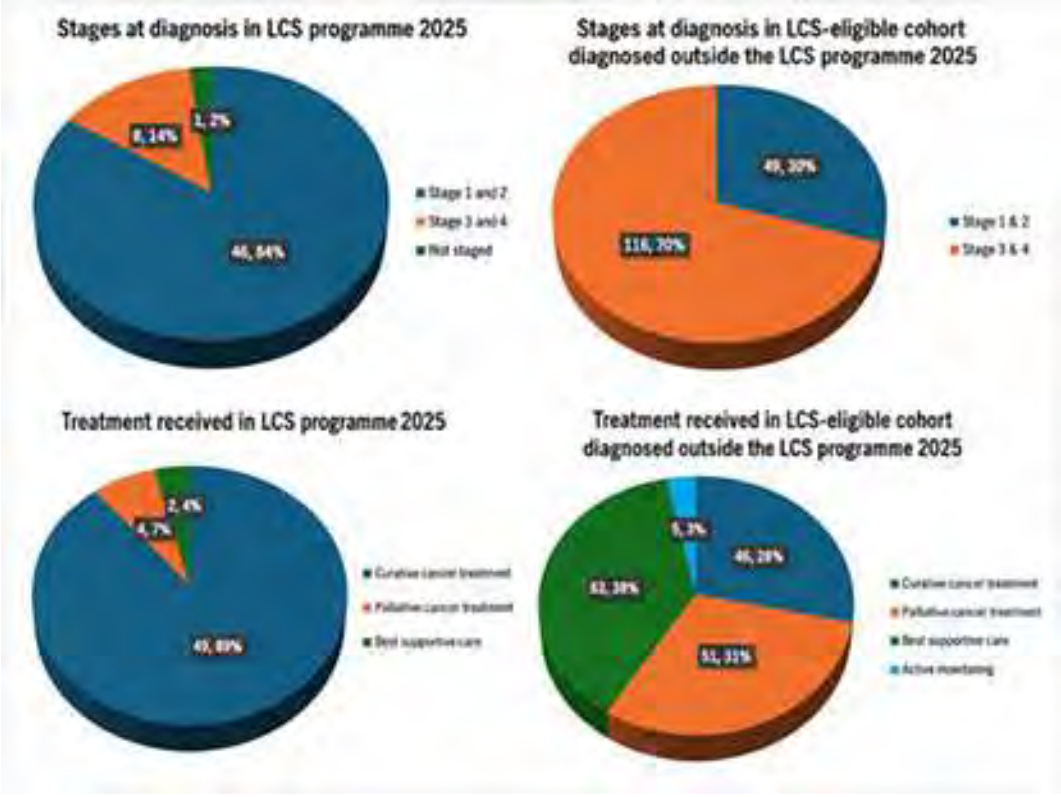
10.1136/thorax-2026-BTSabstracts.348

Introduction and Objectives The national lung cancer screening (LCS) programme has been established in our institutions since April 2021 and August 2022 as pilot sites. To date, 356 lung cancers have been detected through LCS; 280 (78.65%) were early stage and 279 (78.37%) acheived cure. However, many patients still received lung cancer diagnosis outside LCS in 2025.

We explored demographics and reasons for non-participation among missed eligible patients, defined as those diagnosed outside LCS who met eligibility criteria: ever-smokers aged >55 and <75 years.
Methods The Trust lung cancer database for 2025 identified this cohort. Reasons for non-participation were obtained from the LCS database. A comparable 2022 snapshot dataset was used.
Results In 2025, 394 of 449 lung cancers (87.75%) were diagnosed outside LCS. Of these, 165 (36.7% of total cancers) were missed eligible patients. Most, 132 (80%), were not registered with the programme; 22 (13%) could not be contacted; 9 chose not to participate; 1 did not attend; and 1 was deceased at contact. Similar findings were seen in 2022, with non-registration the main reason for missing LCS (71/94, 75.5%).

The non-registered cohort was similar to the whole outside-LCS group by gender, age and deprivation. However, three postcode areas accounted for one-third of unregistered cases, and some postcodes had four times the rate of potentially missed earlier cancer diagnosis due to non-registration. Patients declining participation were younger (59 vs 68 years) and more deprived (average decile 2.0 vs 3.5). Only seven patients identified as non-White British, limiting interpretation.

Among missed LCS-eligible cancers, 116 (70%) presented at stages 3–4, and only 46 (28%) received curative treatment. In contrast, among LCS-detected cancers in 2025, 46 (84%) were early stages and 49 (89%) received curative treatment.
Conclusions Reliable GP-register data, especially coded smoking history, is crucial to maximise LCS uptake. Missing, miscoded or non-searchable smoking data may explain many missed



Abstract P188 Figure 1

opportunities. Engagement campaigns since 2022 have encouraged uninvited eligible individuals to come forward, but missed eligible numbers remain high. Targeted community engagement remains important, but urgent review of smoking-status coding and GP data extraction is needed to improve LCS uptake and outcomes.

P189 ENHANCING EARLY LUNG CANCER DIAGNOSIS - EFFICACY OF A STRATEGY TO INCREASE PRIMARY CARE REFERRALS

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10.1136/thorax-2026-BTSabstracts.349

Introduction NICE NG12 guidelines1 recommend urgent suspected cancer (USC) referral for any patient over 40 with haemoptysis or with chest X-ray (CXR) suspicions of cancer.

Based on NLCA data, Airedale NHSFT has a high proportion of patients with advanced lung cancer, poor performance status at diagnosis, and diagnosed via emergency presentation. In an attempt to improvethis, local referral criteria were broadened five years ago to recommend USC referral and CT for patients with any feature of lung cancer.

Following concerns that this variation from national guidance may cause confusion, increase primary care workload and result in unnecessary CTs, we reviewed 12 months of USC referrals to assess outcomes.

Methods 12 months of GP lung USC referrals from August 2024 onwards were reviewed. Data were collected regarding symptoms, CXR/CT result, and final diagnosis/stage.

Results 270 referrals were identified. 84 had confirmed cancer of which 74 were lung cancer/mesothelioma. Outcomes of diagnostic pathways based on referral reason are presented in table 1.

Conclusion In our population the incidence of cancer in patients fulfilling NG12 criteria was 48.5% compared to 3.3% in those not fulfilling these criteria. Only one of 270 patients had an early-stage lung cancer that was not visible on CXR.

Our findings suggest that widening USC criteria beyond NICE recommendations increases fast-track CT referrals but does not significantly impact lung cancer diagnoses/outcomes. Limited radiology resources could potentially be better utilised in expediting tests for those fulfilling NICE referral criteria or in increasing CXR capacity.

We note our population had a lower rate of lung cancer not seen on CXR than that reported in the literature2. It is possible our population differs from earlier study populations as screening is detecting some early-stage lung cancers.

We propose to align our lung USC criteria with NICE NG12. We plan to re-purpose resources to introduce a same day CT for patients with a suspicious CXR and to develop self-referral

Abstract P189 Table 1 Outcomes of diagnostic pathways based on referral reasons

	No cancer	Thoracic Malignancy	Extra-thoracic Malignancy
All referrals	186	74	10
Patients with plain film suggestive of cancer	53	72	9
Patients with haemoptysis	49	7	1
Patients without haemoptysis and no plain film suspicion of cancer	86	2	1

CXR alongside a primary care education programme to increase rates of early lung cancer detection.

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P190 WHEN IT'S NOT LUNG CANCER: ALTERNATIVE DIAGNOSES AND DIAGNOSTIC DELAYS

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10.1136/thorax-2026-BTSabstracts.350

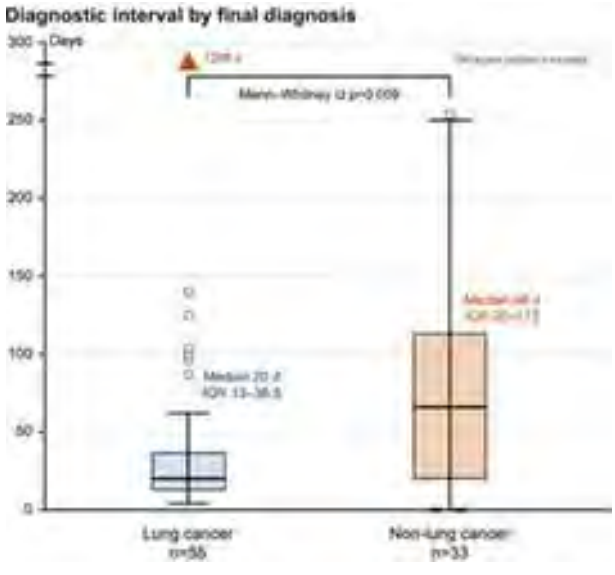
Introduction and Objectives Patients with imaging suspicious for lung cancer enter multidisciplinary team (MDT) surveillance before receiving a definitive diagnosis. While many are diagnosed with lung cancer, a substantial minority have alternative diagnoses. The diagnostic duration and burden associated with these mimics are poorly described. We evaluated diagnostic interval, investigation burden and alternative diagnoses among patients entering a lung cancer MDT surveillance pathway.

Methods We performed a retrospective service evaluation of patients entered onto a district general hospital lung cancer MDT monitoring list between August and November 2025. Patients with unknown outcomes were excluded. Diagnostic interval was measured from first lung cancer physician review to establishment of final diagnosis. Final diagnoses were classified as lung cancer or non-lung cancer. Non-lung cancer diagnoses were manually recoded into clinically relevant mimic categories. Continuous variables were compared using Mann–Whitney U testing and categorical variables using Fisher exact or chi-square testing.

Results Eighty-eight patients were analysed; 55 (62.5%) had lung cancer and 33 (37.5%) had non-lung cancer diagnoses. Median diagnostic interval was more than threefold longer in patients without lung cancer (66 days, IQR 20–113) than those with lung cancer (20 days, IQR 13–36.5; p=0.009; rank-biserial r=-0.33).

Non-lung cancer diagnoses comprised alternative malignancy (30.3%), infection (27.3%), inflammatory disease (21.2%), benign structural lung or pleural disease (12.1%), airway disease (3.0%), vasculitis/connective tissue disease (3.0%) and cardiovascular disease (3.0%). The longest diagnostic intervals occurred in benign structural disease (median 223 days), inflammatory disease (82.5 days) and vasculitis/connective tissue disease (81 days). Alternative malignancies and infection were diagnosed more rapidly. Non-lung cancer patients underwent more CT examinations (median 2 versus 1), although this did not reach statistical significance (p=0.056). EBUS was more frequently performed in lung cancer patients (38.2% versus 12.1%; Fisher p=0.014). Documented patient distress did not differ between groups (20.0% versus 16.4%; p=0.779).

Conclusions More than one-third of patients entering lung cancer MDT surveillance did not have lung cancer. These patients were exposed to the greatest diagnostic delay with comparable investigative burden and associated distress. Earlier recognition of lung cancer mimics may reduce this burden on both the healthcare service and patients.



Abstract P190 Figure 1 Time from first lung cancer physician review to final diagnosis by final diagnostic group. Boxes indicate IQR, centre lines indicate medians and whiskers extend to 1.5xIQR; points are outliers. The capped triangle denotes one 1298-day outlier shown off-scale and included in analysis

P191 STREAMLINING LUNG MULTI-DISCIPLINARY TEAM MEETINGS USING AI BASED CLINICAL DECISION SUPPORT SYSTEM: RETROSPECTIVE DATA ANALYSIS TO VALIDATE STANDARD OF CARE

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10.1136/thorax-2026-BTSabstracts.351

Introduction and Objectives Increasing lung cancer caseloads overwhelm multidisciplinary team (MDT) meetings, delaying crucial treatment targets. In alignment with the 2026 National Cancer Plan, NHS England emphasises implementing Standards of Care (SoC) to reduce MDT workloads by triaging out protocolisable cases. To address this challenge, we implemented an artificial intelligence (AI)-based clinical decision-support-system (CDSS) to streamline lung MDTs at a UK tertiary hospital. As a foundational implementation step, this study evaluates the CDSS in developing and validating SoC criteria for Non-Small-Cell Lung Cancer (NSCLC), which comprises the majority of the MDT caseload.

Methods To establish the SoC pathway for NSCLC, we utilised the Deontics AI-based CDSS, integrating MDT approved evidence from national and international NSCLC guidelines. In addition to these guidelines, the CDS platform incorporated local clinical trial options. To validate the system, we inputted patient data from the five-month period between September 2025 and January 2026 and performed a comparative analysis between the system's recommendations and the documented outcomes of the MDT meetings.

Results Over a five-month study period, 142 unique patients were discussed, with 97 (68%) identified as suspected or confirmed NSCLC cases eligible for analysis. A longitudinal review of these patients encompassed 344 total MDT discussions. The CDS identified substantial opportunities for MDT streamlining. Sixty-six of 344 discussions (19.2%) met predefined Standards of Care criteria and could potentially be triaged out of full MDT discussion. Within this SoC cohort, concordance was

Abstract 191 Table 1 Barts lung MDT retrospective data analysis details: CDSS = clinical decision support system. MDT = multidisciplinary team meeting, NSCLC = non-small-cell-lung-cancer

Description	Numbers (percentage)
Total patient cohort reviewed across 17 consecutive MDT sessions between 26th Sept 2025 to 27th Jan 26 in 2026	142
Cumulative MDT discussions for the 142 patients during their clinical journey	492
Excluded from Analysis	148(30%)
– Mediastinal tumours	6 (1%)
– Metastatic lung disease from non-thoracic origins	48 (9%)
– Cancers other than suspected/confirmed NSCLC	38 (8%)
– Infectious or benign conditions	20 (4%)
– Records with insufficient staging data	36 (8%)
Included in Analysis – 97 patients and 344 MDT discussions	**344** (70%)
– Pulmonary nodules	45 (13%)
– NSCLC confirmed via imaging or biopsy	299 (87%)
○ Stage I	101 (34%)
○ Stage II & III	78 (26%)
○ Stage IV or recurrent disease	120 (40%)
Triage Outcome dataset	344
– Identified as eligible for Standard Of Care (SoC) by CDSS	66 (19.2%)
– Identified as ineligible for SoC and will be discussed in MDT by default	259 (80.8%)
Guideline-based CDSS applicability to MDT context	344
– Applicable: Staging, workup, treatment, and surveillance decisions	**302** (87.7%)
– Inapplicable: Isolated pathology or imaging reviews	42 (22.3%)
Concordance analysis dataset	302
– Cases where MDT decision was postponed due to lack of results	46 (15.2%)
– Cases where MDT discussion resulted in the MDT decision (recommendation)	**256** (84.7%)
○ MDT decision and CDSS recommendations were concordant	237 (93%)
○ MDT decision and CDSS recommendations were discordant	19 (7%)
Clinical trial eligibility dataset – note that it is per patient, so denominator is 97	97
– Potential eligible candidates identified by CDSS	33 (34%)
– Potential eligible candidates documented by MDT	4 (4%)

nearly 100%, with the single discordant recommendation identified being likely due to a MDT outcome transcription error. Furthermore, the platform markedly improved clinical trial screening, identifying 33 potentially eligible patients (34%) in contrast to only 4 (4%) noted in MDT documentation. The tool also highlighted current MDT inefficiencies, noting that 15.2% of discussions failed to reach a recommendation, necessitating re-discussion, primarily due to pending pathology. Overall, the CDS demonstrated 93% concordance with MDT recommendations, with the discordant cases attributed to patient factors and guideline coverage gaps.

Conclusions The study demonstrates technology can help implement SoC, reduce MDT caseload and support implementation of NHS England's MDT streamlining agenda.

P192 LUNG CANCER WITH CYSTIC AIR SPACES (LCCA) – A COMPREHENSIVE REVIEW OF THE LITERATURE

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10.1136/thorax-2026-BTSabstracts.352

Background Lung cancer associated with cystic airspaces (LCCA) is an increasingly recognised yet under-characterised manifestation of non-small cell lung cancer (NSCLC), comprising approximately 1–4% of cases.[1] It remains absent from major international staging frameworks and tumour classifications, generating persistent uncertainty across diagnosis, staging and management.

Methods We undertook a narrative review using a systematic search strategy (PubMed/MEDLINE and Cochrane Library, 2000 onward), with snowball sampling, forward-citation searching, and expert curation across thoracic oncology, radiology, pathology and surgery, following a modified PRISMA 2020 framework.

Results LCCA reflects tumour–airspace interaction rather than a discrete molecular subtype, arising through overlapping mechanisms including tumour growth within pre-existing airspaces and lepidic spread. Adenocarcinoma predominates, with strong associations with smoking and emphysema. The natural history follows stepwise evolution from thin-walled cysts to progressive wall thickening and mural nodularity; because a cystic scaffold may mask growth, size-based criteria are unreliable. When matched for pathological stage, survival is comparable to other NSCLC subtypes, suggesting that poorer outcomes largely reflect diagnostic delay rather than aggressive biology, though increasing solid components confer higher recurrence risk. LCCA is prone to misclassification as benign disease, accounting for 22.7% of missed or delayed diagnoses in the NELSON screening trial.[4] Several radiological classification systems standardise description but lack consensus.[1-3] FDG-PET sensitivity is limited where solid components are small, and tissue sampling carries low yield and elevated complication risk, though CT-guided approaches are safe in experienced hands. Staging extrapolated from part-solid pathways risks underes-

timating tumour burden, generating discordance with pathological stage since radical treatment targets the whole lesion. Treatment aligns with conventional NSCLC—surgery, radical radiotherapy and SABR, and systemic therapy in advanced disease.

Conclusion LCCA as a distinct, under-recognised entity whose heterogeneous appearances and diagnostic pitfalls demand dedicated attention. Formal recognition, consensus on nomenclature, classification and staging, and prospective registry data are essential to optimise outcomes and inform guideline development.

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P193 THE VALUE OF CIRCULATING TUMOUR DNA IN SECONDARY CARE THORACIC ONCOLOGY: SEARCH AND RESCUE

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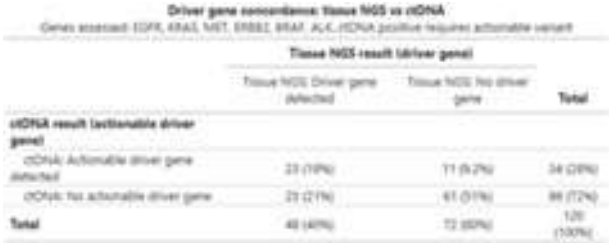
10.1136/thorax-2026-BTSabstracts.353

Plasma cell-free DNA generated by rapid cellular division, apoptosis, and immune-mediated lysis of malignant cells, termed circulating tumour DNA (ctDNA), proposes a new avenue for genomic tailoring of cancer treatments. The role of ctDNA in lung cancer pathways is evolving, with NHS England initiating national rollout of ctDNA testing in 2025. Leveraging our unique position as a ctDNA pilot site, we aimed to analyse the current clinical utility of ctDNA against next-generation sequencing (NGS) within lung cancer pathways.

Patients referred for ctDNA testing at University Hospitals of Morecambe Bay NHS Foundation Trust between August 2023 and October 2025 were included in a retrospective cohort analysis. After excluding non-thoracic malignancies, the primary cohort was n=196. ctDNA results, number of tissue biopsies, NGS results, treatment decisions, and their respective timings were collected. Time-to-event analyses and ctDNA/NGS concordance were performed in R v4.6.0.

Of 59 patients with an actionable mutation who underwent ctDNA and tissue NGS testing, 11 had their druggable mutation detected by ctDNA alone (figure 1). For 38% of patients, ctDNA was their sole access to genomics. ctDNA results returned faster than tissue NGS, with a pseudomedian of –20 days from referral (Wilcoxon signed-rank p<0.001). Time to treatment between patients who received both modalities versus ctDNA only did not differ significantly (61 and 52 days, respectively; Kruskal-Wallis p=0.074). 34 patients required greater than one tissue biopsy. Within this subset, ctDNA results arrived faster with a pseudomedian of –40 days (Wilcoxon signed-rank p<0.0001); however, the 11-day difference in time to treatment was similarly non-significant statistically (Kruskal-Wallis p = 0.964).

ctDNA rescued 1 in 6 patients who would otherwise have been without personalised cancer therapy, and a significant proportion accessed genomics via ctDNA alone. Within this cohort, faster ctDNA results did not translate into a significant reduction in time to treatment, possibly reflecting oncologists awaiting tissue NGS or PD-L1 staining before making treatment decisions. Whilst tissue remains important, particularly for diagnosis, ctDNA testing is less invasive, faster, and reliable at identifying druggable mutations. This represents a powerful adjunct within lung cancer pathways, expanding access to genomics and consequently bespoke cancer therapy.



Abstract P193 Figure 1

P194 THE IMPACT OF CONCORDANCE BETWEEN LIQUID AND TISSUE BIOPSY FOR THE MOLECULAR PROFILING OF ACTIONABLE MUTATIONS IN NON-SMALL CELL LUNG CANCERS (NSCLC)

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10.1136/thorax-2026-BTSabstracts.354

Introduction Tissue biopsy is the benchmark for diagnosing Non-small cell lung cancers but is often limited by invasiveness and patient frailty. Liquid biopsy (circulating tumour DNA) offers a safer, faster alternative for molecular profiling. This study evaluates concordance between these modalities to bridge the gap between clinical frailty and precision medicine.

Aims To analyse concordance, identify demographics and actionable mutation trends, evaluate PD-L1 detection, and compare turnaround time for results in a District General Hospital in UK.

Methods Out of 111 in the cohort exclusions were made for small cell lung cancers(3) and cases where tissue biopsy was unfeasible(25). Hence this retrospective study evaluated 83 Non small cell lung cancer patients (Jan–Dec 2025).Metrics included Overall Percentage Agreement (OPA), Sensitivity, Specificity, and Cohen's Kappa.

Results The cohort with mean age of 68.4 and female predominance was primarily adenocarcinoma (63 cases). Concordance was 89.16% (34 True Positive, 40 True Negative, Kappa 0.78), showing substantial agreement. Sensitivity was 87.18% and specificity 90.91%. KRAS and EGFR were most frequent actionable mutations. Mean Turnaround time was 16 days for liquid biopsy versus 27 days for final tissue molecular profiling. Liquid biopsy flagged actionable mutations in 22 cases out of 46 PD-L1 positive patients.

Conclusion Liquid biopsy is a dependable surrogate for actionable mutations, offering a high concordance with tissue biopsy. However, regular adoption remains limited. While excellent for rapid mutation screening, tissue remains crucial for PD-L1 assessment. Further studies are required to evaluate the long-term clinical impact of early utilization of liquid biopsy.

P195 OUTCOMES OF IMAGING SURVEILLANCE IN VIRTUAL LUNG CANCER TRIAGE: SAFETY, DISCHARGE RATES, AND CANCER DETECTION WITHIN THE NATIONAL OPTIMAL LUNG CANCER PATHWAY

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10.1136/thorax-2026-BTSabstracts.355

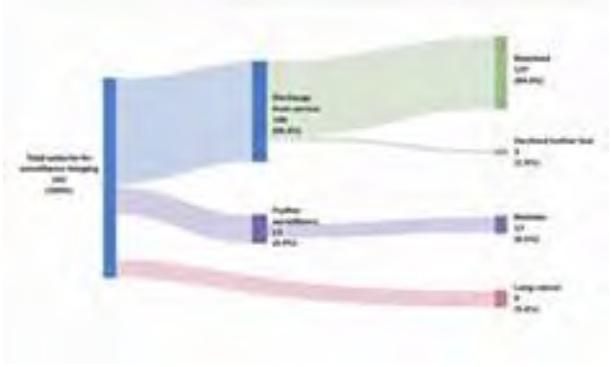
Background Lung cancer is the leading cause of cancer-related mortality in the UK. The National Optimal Lung Cancer Pathway (NOLCP) aims to identify and manage patients with suspected lung cancer in a standardised efficient manner. This includes lung triage where all lung cancer referrals are reviewed. Here, we assessed the outcomes of patients initially managed with surveillance imaging from triage.

Methods We retrospectively reviewed NOLCP referrals at a tertiary teaching hospital between January 2025 and January 2026. Patients allocated to imaging surveillance were included. Referrals originated from primary care (two-week wait), direct-to-CT, and inpatient services. All were virtually triaged by respiratory physicians, with outcomes communicated via clinic letters or Lung Specialist Nurses.

Results 2,217 cases were reviewed in virtual triage during this period, of which 162 (7%) referrals were triaged for further surveillance imaging. Of these, 88 patients were male and 74 female, with a median age of 71.5. Radiological findings included 51 (31.4%) solid nodules, 51 (31.4%) consolidations, 21 part-solid nodules, 17 ground-glass opacities, 12 tree-in-bud nodularity, 6 isolated lymphadenopathy cases, and 4 pleural effusion/thickening. 63 patients received a course of antibiotics in view of consolidative change suspicious for infective aetiology.

Following initial triage, 61 patients underwent repeat CT imaging at 4–6 weeks, 29 at 8 weeks, 66 at 3 months, 3 at 6 months, and 1 at 9 months. Two patients were referred directly to the lung nodule clinic for ongoing surveillance.

Following surveillance, 140 patients (86.4%) were discharged, including 137 with resolved findings and 3 who declined further testing. 13 patients (8.0%) remained under surveillance for pulmonary nodules, while nine patients (5.6%) were diagnosed with lung cancer. No cases were upstaged during surveillance; stages ranged from IA to IIIA, and all received curative-intent treatment.



Abstract P195 Figure 1 Outcomes of surveillance imaging in 162 patients

Conclusion Virtual triage using imaging characteristics and patient clinical symptoms is an effective and safe approach to managing selected suspected lung cancer referrals with interval imaging. Most patients were safely discharged following surveillance, while cancers detected remained suitable for curative treatment with no evidence of upstaging. This helps reduce unnecessary clinic appointments and investigations, easing lung cancer service pressures.

Ventilation: frailty and comorbidity in patients with respiratory failure

P196 COMBINATION OF NON-INVASIVE VENTILATION OUTCOME (NIVO) AND ROCKWOOD CLINICAL FRAILTY SCALE (CFS) SCORES IN PREDICTING IN-HOSPITAL MORTALITY IN COPD EXACERBATIONS REQUIRING NON-INVASIVE VENTILATION

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10.1136/thorax-2026-BTSabstracts.356

Introduction and Objectives The NIV Outcome (NIVO) score is a validated prognostic tool for patients admitted with COPD exacerbations requiring non-invasive ventilation (NIV).[1] The NIVO score does not include frailty, which has been independently associated with a 60% higher risk of one-year mortality in patients with acute hypercapnic respiratory failure treated with NIV.[2] We aim to prove that the Rockwood Clinical Frailty Scale (CFS) provides complementary prognostic information to NIVO using joint categorical analysis as the primary method.

Methods NIVO and CFS scores as well as in-hospital mortality were recorded for 49 COPD patients treated with NIV between February and August 2025. The primary analysis used a 2×2 joint risk matrix categorizing patients by NIVO (low <4 vs high ≥4) and CFS (low <6 vs high ≥6) to determine whether frailty identifies additional mortality risk within each NIVO stratum. Spearman's rank correlation and Receiver Operating Characteristic (ROC) and Area Under the Curve (AUC) were supporting analyses.

Results In-hospital mortality was 24.5% (12/49). The 2×2 matrix demonstrated a statistically significant mortality gradient: patients with both low NIVO and CFS had zero mortality (0/10), while those with both high scores had 36% (9/25). Within the low-NIVO stratum, high frailty increased mortality from 0% to 22.2% (2/9), indicating that frailty identifies residual risk not captured by NIVO alone. Spearman: NIVO r=0.369 (p=0.009), CFS r=0.368 (p=0.009) and combined r=0.455 (p=0.001). ROC-AUC: NIVO 0.743, CFS 0.737 and combined 0.802.

Conclusions Combined use of NIVO and CFS in a joint categorical framework is superior to either score alone, identifying a zero-mortality low-risk group and 36% mortality high-risk group with frailty adding prognostic value within each NIVO stratum. These findings support prospective multicentre validation of combined NIVO and CFS in COPD exacerbations requiring NIV to develop a formally weighted composite prognostic tool.

Abstract P196 Table 1 Primary analysis: 2×2 joint risk matrix for mortality with NIVO × CFS categories in COPD cohort (n=49)

	FRAILTY LOW (CFS < 6)	FRAILTY HIGH (CFS ≥ 6)
NIVO LOW (Score < 4)	**n=10 – 0 deaths** **0% mortality** *Safest group with zero deaths*	**n= 9 – 2 deaths** **22.2% mortality** *Frailty reveals hidden risk.*
NIVO HIGH (Score ≥ 4)	**n= 5 – 1 death** **20.0% mortality** *NIVO drives risk*	**n=25 – 9 deaths** **36.0% mortality** *Highest risk*

NIVO low: score <4 vs NIVO high: ≥4 (median), CFS low: score <6 vs CFS high: ≥6 (median). Each quadrant is independently derived and no arithmetical combination of scores is required.

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Abstract P197 Table 1

Category	Group	%
Indication	COPD	51.4
	Acute cardiogenic pulmonary oedema	17.3
	Other	17.9
	Obesity-related respiratory failure	9.8
	Neuromuscular / chest wall	3.5
Outcome	Discharged	68.8
	Death	29.5
	Readmission (NIV)	1.7
Frailty (CFS)	1–3 (fit–vulnerable)	24.3
	4–5 (mild–moderate)	42.8
	≥6 (severe)	32.9
Age (years)	<65	31.2
	65–74	32.9
	75–84	27.7
	≥85	8.1

P197 FRAILTY INDEPENDENTLY PREDICTS MORTALITY IN PATIENTS RECEIVING ACUTE NON-INVASIVE VENTILATION FOR TYPE 2 RESPIRATORY FAILURE: A MULTICENTRE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.357

Introduction Acute non-invasive ventilation (NIV) for type 2 respiratory failure (T2RF) carries significant in-hospital mortality. Whilst chronological age is a recognised predictor of poor outcome, frailty captures cumulative physiological reserve and may add prognostic value. We aimed to determine whether the Rockwood Clinical Frailty Scale (CFS) predicts in-hospital mortality independently of age in patients commenced on acute NIV for T2RF.

Methods Retrospective data were collected from consecutive adults commenced on acute NIV for T2RF at two UK hospitals during 2024–25. Baseline indication, CFS, age, and in-hospital outcome were recorded. The relationship between frailty, age and mortality was assessed using the Mann–Whitney U test and logistic regression. A multivariable model included both CFS and age, with discrimination assessed using the area under the receiver operating characteristic curve (AUROC).

Results 173 patients received acute NIV (Luton & Dunstable 59.5%, Harlow 40.5%). The commonest indication was COPD (51.4%); overall in-hospital mortality was 29.5%. Frailty was prevalent: 75.7% of patients scored CFS 4 or above, with 32.9% scoring 6 or above indicating severe frailty. Mortality increased stepwise with frailty severity, from 14.3% in CFS 1–3, to 29.7% in CFS 4–5, and 40.4% in CFS 6 or above. Median CFS was higher in non-survivors than survivors (5 vs 4.5, p=0.007), with each one-point rise in CFS associated with a 38% increase in the odds of death (OR 1.38, 95% CI 1.09–1.75). Increasing age was also associated with mortality (OR 1.07 per year, 95% CI 1.04–1.11). In the multivariable model, frailty remained an independent predictor (CFS OR 1.31, 95% CI 1.01–1.70, p=0.041) after adjustment for age (OR 1.07, 95% CI 1.03–1.11, p<0.001). The combined model achieved moderate-good discrimination (AUROC 0.74).

Conclusion Frailty measured using the Rockwood CFS independently predicted in-hospital mortality in patients receiving acute NIV for T2RF, adding prognostic value beyond age alone. Routine CFS assessment at NIV initiation may aid early risk stratification and inform ceiling-of-care discussions. Integration of frailty assessment into NIV escalation pathways and prospective validation in larger cohorts, including comorbidity adjustment, represent important next steps.

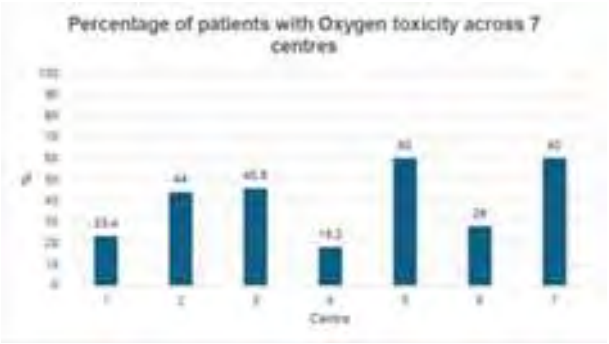
P198 PREVALENCE AND INFLUENCE OF OXYGEN TOXICITY IN COPD EXACERBATIONS REQUIRING NON-INVASIVE VENTILATION

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10.1136/thorax-2026-BTSabstracts.358

Introduction Administration of excess oxygen during COPD exacerbations is associated with higher rates of ventilation and mortality.[1] Oxygen therapy titrated to SpO_2 88-92% is associated with better survival, including in normocapnic patients.[2]

Methods Within the 7 centre non-invasive ventilation outcomes scores directed weaning RCT (ISRCTN: 64639614), we assessed the prevalence of oxygen toxicity (OT) prior to initiation of NIV for ECOPD and compared baseline characteristics and outcomes in OT and no oxygen toxicity (NOT) subgroups. OT was defined by the highest $Pa0_2$ on arterial/ cap-



Abstract P198 Figure 1

illary blood gases (ABG) prior to NIV: <10kPa= NOT; 10.1-13.3kPa= moderate OT; and >13.3kPa= severe OT.

Results 55/164 (33.6%) participants had OT, which was severe in 27. The prevalence of OT varied substantially across centres (18.2%-60%). OT was associated with a lower prevalence of cardiovascular co-morbidities (OT= 47.3%, NOT= 77.1%, p<0.01). There was no association between OT and eMRCD, LTOT, FEV1, BMI or age. OT was associated with more severe baseline respiratory acidaemia (RA), Median (IQR) pre-NIV pH: OT= 7.25 (7.20-7.29); NOT= 7.30 (7.26-7.32) p<0.001, $PaC0_2$: OT= 9.25 (7.9-10.6)kPa, NOT= 8.70 (7.9-10.9)kPa, p=0.25. Despite showing a greater fall in $PaC0_2$ and rise pH on NIV, a higher proportion of OT patients still had persistent RA on 1-2 hr (OT= 69.8%, NOT= 49%, p=0.01) and 4-6hrs ABG (OT= 46.3%, NOT= 27.5%, p=0.02). Median (IQR) time from NIV initiation to meeting weaning criteria, OT= 37.4 (31.5-44.1), NOT= 35.4 (27.3-40.7) hours p=0.16, and inpatient death (OT= 5.5. %, NOT= 1.8%, p=0.20) were numerically worse with OT. Relapse (OT= 5.5%, NOT= 4.6%, p=0.81) was similar. Mann-Whitney U and Chi-square as appropriate

Conclusion Oxygen toxicity remains common in COPD patients requiring NIV, with marked variation between centres. OT was associated with worse respiratory acidaemia pre-NIV, and higher rates of persistent respiratory acidaemia up to 6 hours after initiation of NIV; OT does not lead to rapidly reversible RA or less adverse outcomes. OT will be important data capture in future national audits. Simple condition-based target oxygen saturations for all COPD patients from ambulance pickup should reduce the risk of OT.

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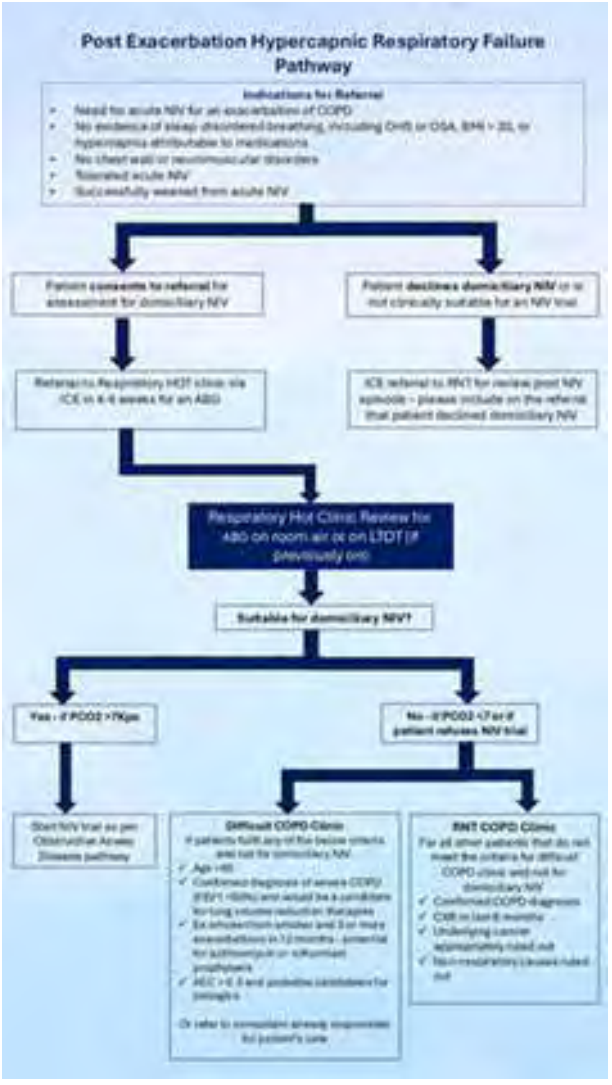
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P199 EVALUATING THE IMPACT OF A POST-EXACERBATION RESPIRATORY FAILURE PATHWAY ON HOME MECHANICAL VENTILATION REFERRALS AND CLINICAL OUTCOMES: MIND THE GAP

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10.1136/thorax-2026-BTSabstracts.359

Background Acute Hypercapnic Respiratory Failure (AHRF) secondary to chronic obstructive pulmonary disease (COPD)



Abstract P199 Figure 1

exacerbation is associated with significant morbidity, mortality and recurrent hospital admissions. Guidance recommends assessing patients with persistent hypercapnia for Home Mechanical Ventilation (HMV) following an episode of AHRF requiring acute non-invasive ventilation (NIV). A post exacerbation respiratory failure pathway was implemented (figure 1), and clinicians were advised to refer accordingly. We have evaluated the referral patterns and clinical outcomes.

Methods Patients admitted between July - December 2025 with COPD exacerbation requiring acute NIV were identified through admission database. Patient demographics, acute NIV outcome, discharge process and referrals for HMV to the Respiratory HOT clinic (4-6 weeks) following discharge were obtained by electronic patient records.

Results Over a six-month period, 139 patients were admitted with COPD exacerbation needing acute NIV. 30.2% (n=42) were on HMV initiated previously and 10.8% (n=15) were commenced on HMV during admission due to failure to wean from acute NIV. 21.6% (n=30) were deemed appropriate for HMV assessment in the post exacerbation period. 47% (n=14) were referred for further assessment (n=11 attended, 1=declined and 2= unable to attend due to illness). None of

the patients who attended had persistent hypercapnia (Pco2 > 7) on arterial blood gas assessment.

However, the clinic reviews enabled optimisation of other COPD management (pulmonary rehabilitation referral-91%; smoking cessation advice- 91%; inhaler optimisation-82%; consideration or initiation of azithromycin or roflumilast therapy-64%). This intervention led to reduction in hospital readmissions as compared to those who were not referred (36.3% v/s 50%, P= 0.69) but none required acute NIV. In those who were not referred, 25% (n=4) died and 25% (n=4) were eventually established on HMV via referrals outside the pathway.

Conclusion Non-adherence to the post-exacerbation pathway is associated with a high rate of missed clinical optimisation, likely disease progression and increased mortality. While clinic attendance successfully optimises COPD care, stricter electronic or system-wide safeguards are urgently required to prevent eligible patients from falling through the referral gap. Introduction of a standardised discharge referral pathway and targeted staff education may improve patient identification and reduce referral inconsistency.

P200 CHANGES IN RESPIRATORY PARAMETERS OBTAINED FROM REMOTE NIV MONITORING ARE ASSOCIATED WITH EXACERBATIONS IN PATIENTS WITH COPD

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10.1136/thorax-2026-BTSabstracts.360

Background Remote access to data provided by non-invasive ventilators (NIV) can help to understand patient adherence to long-term NIV (LT-NIV). Only a limited number of studies analysed the value of remote monitoring to identify exacerbations in patients with chronic hypercapnic COPD in LT-NIV, and the results are contradictory.

Methods We investigated 65 patients with COPD who were established on Lumis 150 (ResMed UK) ventilators between May 2025 and January 2026. We used the Airview platform to gather data on average hours of usage, tidal volume (TV), respiratory rate (RR), leaks, spontaneous cycle (%), spontaneous trigger (%), minute ventilation (MV), residual apnoea-hypopnoea index (AHI) and rapid shallow breathing index (RSBI) at days 28-21 before (T1), days 7-0 before (T2), days 0-7 after (T3) and days 21-28 after (T4) the onset of an exacerbation.

Results Fifty-nine exacerbation in 25 patients were identified. When the first exacerbation of the 25 patients was analysed, there was a significant increase in minutes of daily use (from 552 ± 200 mins to 592 ± 231 mins, p=0.04) and respiratory rate (14.5 ± 1.4/min to 14.7 ± 1.5/min, p=0.01) between T1 and T2. When all exacerbations were analysed together, there was a significant increase in RSBI (from 20.4 ± 7.4/min/L to 21.4 ± 8.2/min/L, p=0.01) in the same period. Interestingly, following the event, the daily use significantly increased (552 ± 200, 592 ± 231, 593 ± 207 and 621 ± 184 mins, T1, T2, T3 and T4, respectively, p=0.01). There was no difference in the other parameters.

Conclusions Patients with COPD increase their NIV use during exacerbations which is associated with a slightly higher respiratory rate. Whether this signal on remote monitoring can be used for early intervention at the onset of exacerbation needs to be tested in future studies. The reason for increase in daily NIV use by more than 1h one month after exacerbation compared to the baseline value is not known, but could represent either increased motivation for LT-NIV use, or the fact that patients become more dependent on ventilators.

P201 AGREEMENT AND ACCURACY OF VENTILATOR-DERIVED VCO_2 FOR ESTIMATING RESTING ENERGY EXPENDITURE IN MECHANICALLY VENTILATED CRITICALLY ILL ADULTS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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10.1136/thorax-2026-BTSabstracts.361

Background Accurate determination of resting energy expenditure (REE) is essential in critically ill patients to avoid the adverse consequences of underfeeding and overfeeding. Indirect calorimetry (IC) remains the reference standard for measuring REE but is limited by cost, availability, and technical requirements. Ventilator-derived carbon dioxide production (VCO_2) has emerged as a practical alternative for estimating REE in mechanically ventilated patients; however, its accuracy and agreement with IC remain uncertain. We performed a systematic review and meta-analysis to evaluate the performance of VCO_2-derived REE compared with IC in critically ill adults.

Methods A systematic literature search was conducted to identify studies evaluating VCO_2-derived REE against IC in mechanically ventilated critically ill adults. Studies involving pediatric populations, predictive equations without VCO_2 measurements, and non-comparative studies were excluded. The primary outcome was acceptable agreement between VCO_2-derived REE and IC-derived REE. Secondary outcomes included mean bias, limits of agreement, and measures of reliability.

Results Six studies involving 531 mechanically ventilated adult patients met the inclusion criteria. Study designs included prospective observational, retrospective validation, and comparative studies. Reported agreement varied considerably across studies. Acceptable agreement ranged from 7% to 89%, with a crude pooled agreement of 64.6% (317/491 observations). Mean REE estimated by VCO_2-derived methods generally approximated IC-derived REE; however, substantial inter-study variability was observed. Extractable bias data from three studies demonstrated a preliminary pooled mean bias of approximately +32.5 kcal/day, suggesting limited systematic overestimation at the population level. Despite this, wide limits of agreement and inconsistent performance across studies indicate significant variability in individual patient estimates.

Conclusions Ventilator-derived VCO_2 provides a feasible and readily available approach for estimating REE in mechanically ventilated critically ill adults when IC is unavailable. Although average bias appears small, substantial variability exists between studies and individual patients. Current evidence supports the use of VCO_2-derived REE as a pragmatic surrogate rather than a replacement for indirect calorimetry. Further prospective studies using standardized methodologies are required to establish its clinical utility and reliability.

P202 MORBID OBESITY AND ACUTE NIV: PRESERVED ACUTE OUTCOMES AND IMPROVED ONE-YEAR SURVIVAL

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Background Morbid obesity is increasingly prevalent among patients admitted with acute hypercapnic respiratory failure (AHRF) and may influence both acute and long-term outcomes following non-invasive ventilation (NIV). However, outcome data from real-world ward-based NIV services remain limited. We aimed to compare the characteristics and outcomes of morbidly obese and non-morbidly obese patients receiving acute NIV for AHRF.

Methods We analysed audit data from adults receiving acute NIV for AHRF at a tertiary centre between April 2019-March 2020 and April 2024-March 2025. Morbid obesity was defined as body mass index (BMI) >40 kg/m^2. Acute NIV success, in-hospital mortality, one-year mortality, length of stay and new home NIV setup were compared between morbidly obese and non-morbidly obese patients. Outcomes were also explored across the two study periods.

Results Among 390 admissions (340 patients), 73 (19%) occurred in patients with morbid obesity and 314 (81%) in those without, with similar proportions across both study periods. Morbidly obese patients were younger than non-morbidly obese patients [median age (IQR) of 68 (17) vs 71 (14) years] and had higher BMI [49 (11) vs 26 (16) kg/m^2]. Pre-existing CPAP use was more common in the morbidly obese group (15% vs 2%, p<0.001). Obesity-related respiratory failure was the predominant cause of AHRF among morbidly obese group (69%), whereas COPD predominated in the non-morbidly obese group (74%, p<0.001). Acute NIV success was similar between groups (77% vs 73%), and remained consistently high across both service periods. In-hospital mortality demonstrated a non-significant trend towards being lower in morbidly obese patients (11% vs 18%, p=0.165). However, one-year mortality was markedly lower in the morbidly obese cohort (25% vs 47%, p<0.001). New home NIV setup was more than twice as common among morbidly obese patients (33% vs 14%, p<0.001), while length of stay was comparable.

Conclusion Morbidly obese patients accounted for almost one-fifth of acute NIV admissions and demonstrated preserved acute NIV outcomes. They experienced significantly lower one-year mortality and were more likely to transition to home NIV compared with non-morbidly obese patients. These findings suggest that obesity-related AHRF represents a distinct and potentially more treatable phenotype within acute NIV services.

P203 EVOLUTION OF ACUTE NIV PRACTICE FOLLOWING THE COVID-19 PANDEMIC: CHANGES IN CASE-MIX, CARE PATHWAYS AND OUTCOMES

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Background Acute non-invasive ventilation (NIV) is a cornerstone therapy for acute hypercapnic respiratory failure (AHRF). The COVID-19 pandemic altered healthcare delivery, disease management and patient behaviour, potentially changing the population requiring acute NIV. We aimed to compare diagnostic characteristics, admission trajectories and outcomes of patients receiving acute NIV before and after the COVID-19 pandemic.

Methods We analysed prospectively collected data from our Acute NIV quality database for adults receiving acute NIV for AHRF at a tertiary centre during two time periods: pre-covid (April 2019- March 2020, n=187 patients, 206 admissions)

Abstract P203 Table 1 Patient characteristics and outcomes summary

	Pre-Covid (n=188 patients with 206 admissions)	Post-Covid (n=177patients with 208 admissions)	P value
DEMOGRAPHICS (Unique Patients, n=365)			
Age, years, median (IQR)	68 (60-75)	72 (64-79)	**0.001***
Sex, male, n (%)	74 (39)	76 (43)	0.557
BMI, kg/m^2, median (IQR)	27 (21-38)	29 (22-37)	0.267
Clinical Frailty Score, median (IQR)	5 (4-6)	4 (4-6)	**0.034**
DIAGNOSIS (All Admissions, n=414)			
COPD, n (%)	134 (65)	134 (65)	0.098
ORRF, n (%)	48 (23)	53 (26)	
NMD, n (%)	13 (6)	4 (2)	
CWD, n (%)	6 (3)	6 (3)	
CPE, n (%)	2 (1)	1 (1)	
Substance/drug, n (%)	1 (1)	8 (4)	
Other, n (%)	2 (1)	2 (1)	
PHYSIOLOGY BEFORE NIV (All Admissions, n=414)			
pH, median (IQR)	7.27 (7.22-7.31)	7.26 (7.20-7.30)	0.089
pCO_2, kPa, median (IQR)	9.6 (8.5-11.6)	10.0 (8.4-11.7)	0.341
LOCATION FOR NIV START (All Admissions, n=414)			
A&E, n (%)	115 (56)	118 (57)	**0.05**
Acute medical assessment units, n (%)	30 (15)	18 (9)	
Wards, n (%)	42 (20)	63 (30)	
ICU, n (%)	0(0)	1 (1)	
Transfer to Critical care	23 (11)	1 (1)	**<0.001**
OUTCOMES (All Admissions, n=414)			
Acute NIV success, n (%)	159 (77)	146 (70)	0.133
New domiciliary NIV setup, n (%)	40 (19)	30 (14)	0.221
Length of stay, days, median (IQR)	9 (5-17)	9 (6-16)	0.823
In-hospital mortality, n (%)	38 (18)	36 (17)	0.862
1-year mortality, n (%)	92 (45)	92 (44)	0.935

BMI body mass index; *CPE* cardiogenic pulmonary oedema; *COPD* chronic obstructive pulmonary disease; *CWD* chest wall disease; *NMD* neuromuscular disease; *ORRF* obesity-related respiratory failure.

and post-covid (April 2024-March 2025, n=178 patients, 208 admissions). Unique patients were analysed for demographic comparisons, while all admission episodes were used to compare physiological, diagnostic distribution, admission trajectories and outcomes between groups.

Results A total of 414 acute NIV admissions of 365 patients were analysed (table 1). Patients in post-covid cohort were older despite lower frailty scores. COPD remained the predominant indication for acute NIV (65%) and the principal contributor to recurrent admissions in both periods (74%), followed by Obesity-Related Respiratory Failure (24% both). Neuromuscular Disease admissions non-significantly declined, whereas substance/drug-related respiratory failure increased. Ward-based NIV (75.5% vs 58.5%) was more common as ceiling of care in post era, while NIV starts at high dependency unit (12.1% vs 1.0%) was more common in pre ($p<0.001$). NIV was increasingly initiated on hospital wards in the post-COVID period, with fewer at the front-door and fewer transfers to critical care units. Overall, NIV success, new domiciliary NIV setups, in-hospital and one-year mortality was 74, 17, 17 and 44%, respectively, and remained unchanged between groups despite these changes.

Conclusion The population receiving acute NIV has changed following the COVID-19 pandemic, with patients being older and a significantly higher proportion of patients receiving ward based NIV as ceiling-of-care. Despite these changes short- and long-term outcomes remained stable, suggesting successful adaptation of acute NIV services to evolving post-pandemic demands.

P204 OUTCOMES OF HIGH FLOW NASAL OXYGEN THERAPY IN A RESPIRATORY SUPPORT UNIT OUTSIDE CRITICAL CARE

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10.1136/thorax-2026-BTSabstracts.364

Introduction and Objectives High flow nasal oxygen therapy (HFNO) is being increasingly used for the management of acute respiratory failure – both in critical care and in ward settings. The aim of this study was to determine the clinical outcomes of HFNO outside critical care in a respiratory support unit (RSU) in a large tertiary teaching hospital.

Methods Based on local protocol, HFNO can be considered outside critical care for acute type 1 respiratory failure requiring 40% or more conventional oxygen, with a variety of underlying aetiology. It is delivered by trained nursing staff in a 12-bedded RSU within the respiratory wards. The decision to commence HFNO is made by a respiratory consultant, and a ceiling of treatment must be agreed and documented prior to commencement.

All cases between December 2023 and January 2026 were included in this study.

Results 44 patients (50% male) with a median (IQR) age of 76 (62-81) years were included. 70% had pneumonia with underlying cardiorespiratory co-morbidities (interstitial lung disease 18%, chronic obstructive pulmonary disease 36%, and heart failure 16%). 20% had pneumonia without underlying cardiorespiratory co-morbidities and 9% had Covid pneumonitis. 38 patients (86%) were deemed not for escalation to critical care prior to commencement of HFNO. Median (IQR) duration of HFNO was 6 (3-9) days. HFNO was continued in 39% even following a decision to palliate. 6 patients who were deemed for escalation were eventually transferred to critical care, including 3 who were intubated; however, 4 of them died. Overall, inpatient mortality was high at 59%, with a further 9% dying within 8 weeks of discharge (see table 1).

Conclusions Our experience shows a relatively high mortality with HFNO, in a cohort predominantly comprising of patients with pre-existing cardiorespiratory co-morbidities who are deemed not for escalation to critical care. Patients with pneumonia without underlying cardiorespiratory comorbidities had a relatively better survival compared to the other groups. Further studies are needed to define stricter patient selection criteria, so that this resource-intensive form of respiratory support can be delivered more effectively outside critical care.

Abstract P204 Table 1 Indication for HFNO based on aetiology, and subsequent outcomes

Indication for HFNO	n, %	Escalated to critical care (n, %)	Inpatient death (n, %)	Death within 8-weeks post-discharge (n, %)
Pneumonia with underlying ILD	8 (18%)	1 (12.5%)	6 (75%)	1 (12.5%)
Pneumonia with underlying COPD	16 (36%)	2 (12.5%)	8 (50%)	2 (12.5%)
Pneumonia with underlying HF	7 (16%)	1 (14%)	6 (86%)	0
Pneumonia without underlying cardiorespiratory co-morbidities	9 (20%)	2 (22%)	4 (44%)	0
Covid pneumonitis	4 (9%)	0	2 (50%)	1 (25%)
Total	44	6 (13.6%)	26 (59%)	4 (9%)

ILD: Interstitial lung disease; COPD: Chronic obstructive pulmonary disease; HF: heart failure

P205 CLINICAL OUTCOMES OF NON-INVASIVE VENTILATION IN PATIENTS WITH POST-TUBERCULOSIS LUNG DISEASE PRESENTING WITH ACUTE HYPERCAPNIC RESPIRATORY FAILURE

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Introduction and Objectives Post-tuberculosis structural lung disease is increasingly recognized as an important cause of chronic respiratory morbidity, particularly in high tuberculosis burden countries, and may predispose patients to acute hypercapnic respiratory failure (AHRF). However, evidence regarding the effectiveness of non-invasive ventilation (NIV) in this subgroup remains limited. We aimed to evaluate the clinical and

Abstract P205 Table 1

Variable	Overall Cohort (n=49)	p value
Baseline characteristics		
Age, years (Mean ± SD)	56.0 ± 15.0	—
Male sex, n (%)	33 (67.3)	—
Smokers, n (%)	25 (51.0)	—
Baseline SpO_2 on room air, % (Mean ± SD)	73.0 ± 12.4	—
Physiological response to NIV		
pH improvement	7.30 ± 0.06 → 7.43 ± 0.05	<0.001
$PaCO_2$ clearance, mmHg	67.8 ± 12.5 → 53.4 ± 8.2	<0.001
Pulse rate improvement, bpm	114.1 ± 17.7 → 89.6 ± 11.5	<0.001
Respiratory rate improvement, /min	26.8 ± 3.1 → 18.9 ± 2.4	<0.001
Clinical outcomes		
NIV success, n (%)	47 (95.9)	—
NIV failure requiring intubation, n (%)	2 (4.1)	—
Total NIV duration, days (Mean ± SD)	6.04 ± 2.26	—
Weaning duration, days (Mean ± SD)	3.96 ± 1.63	—
NIV-related complications, n (%)	25 (51.0)	—
NIV duration with vs without complications, days	7.28 ± 2.21 vs 4.75 ± 1.48	<0.001
LTOT at discharge, n (%)	24 (49.0)	—
Domiciliary NIV at discharge, n (%)	5 (10.2)	—

physiological outcomes of NIV in patients with post-tuberculosis lung disease presenting with type II respiratory failure.

Methods A prospective observational study was conducted in 49 patients with post-tuberculosis lung disease presenting with acute hypercapnic respiratory failure managed with NIV. Demographic, clinical, radiological and serial arterial blood gas (ABG) parameters were analysed. Serial changes in pH, $PaCO_2$ and HCO_3^- during NIV therapy were assessed using repeated measures ANOVA.

Results The mean age was 56.0 ± 15.0 years, with male predominance (67.3%). NIV was successful in 47/49 patients (95.9%), while 2 patients (4.1%) required invasive mechanical ventilation. Overall mortality was 2.0%.

Significant serial improvement in ABG parameters was observed during NIV therapy. Mean arterial pH improved from 7.30 ± 0.06 at baseline to 7.43 ± 0.05 at discharge ($p<0.001$), while mean $PaCO_2$ decreased from 67.8 ± 12.5 mmHg to 53.4 ± 8.2 mmHg ($p<0.001$). Respiratory distress improved significantly, with reduction in pulse rate (114.1 ± 17.7 vs 89.6 ± 11.5 bpm, $p<0.001$) and respiratory rate (26.8 ± 3.1 vs 18.9 ± 2.4 breaths/minute, $p<0.001$).

NIV-related complications occurred in 25 (51.0%) patients which were minor and manageable but associated with longer NIV duration (7.28 ± 2.21 vs 4.75 ± 1.48 days, $p<0.001$). The mean duration of NIV therapy, including weaning days, was 6.04 ± 2.26 days. At discharge, 49.0% patients required long-term oxygen therapy and 10.2% required domiciliary NIV support.

Conclusions NIV is an effective and well-tolerated modality for acute hypercapnic respiratory failure in patients with Post tuberculosis lung disease, with high success rates and significant improvement in gas exchange parameters. Despite favourable acute outcomes, a substantial proportion of survivors remained oxygen-dependent at discharge, highlighting the persistent chronic respiratory burden following tuberculosis.

P206 COMPREHENSIVE CLINICAL PROFILE AND RESPIRATORY OUTCOMES OF PATIENTS WITH LIMB-GIRDLE MUSCULAR DYSTROPHY IN A SPECIALISED LONG-TERM VENTILATION SERVICE

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10.1136/thorax-2026-BTSabstracts.366

Introduction Limb-girdle muscular dystrophy (LGMD) is a clinically diverse group of neuromuscular disorders characterized by progressive weakness, often leading to respiratory insufficiency, bulbar dysfunction, and cardiac complications. This study evaluates the clinical characteristics, comorbidities, and outcomes of an LGMD cohort managed within a specialised long-term ventilation service (LTVS) in Wythenshawe Hospital, Manchester.

Methods A retrospective analysis of 34 patients diagnosed with LGMD was performed. Data regarding demographics, comorbidities, cardiac investigations, diaphragmatic involvement, nutritional support (PEG), and respiratory interventions were analysed. Descriptive statistics were utilized to summarize patient characteristics and respiratory outcomes.

Results The cohort (N=34) comprised 41.2% (n=14) males and 58.8% (n=20) females. Mean age was 44.4 ± 14.2 years with a mean BMI of 28.3 kg/m^2. Autosomal Recessive types of LGMD were predominant at 82% (n=28) in the cohort. Documented comorbidities included hypertension (n=12), type 2 diabetes mellitus (n=5), heart failure (n=3), COPD (n=2), and ischaemic heart disease (n=2). Clinical investigations revealed various cardiac abnormalities, including left ventricular hypertrophy and conduction defects and only 2.9% (n=1) had a pacemaker. Diaphragmatic involvement was documented in 11.8% (n=4) of the cohort.

Longterm-Home Non-Invasive-Ventilation (LTH-NIV) was utilized by 67.6% (n=23) of patients. Mean pCO2 levels demonstrated improvement following NIV initiation, decreasing from 6.69 kPa pre-setup to 5.84 kPa at the latest clinical review. Cough augmentation was provided to 58.8% (n=20) of patients to facilitate secretion management. Furthermore, 5.9% (n=2) of the cohort had undergone PEG (Percutaneous Endoscopic Gastrostomy) insertion to address significant weight loss and bulbar dysfunction while another 11.8% (n=4) had refused PEG insertion. Overall survival remained high at 94.1% (32/34).

Conclusion Patients with LGMD present with a significant burden of cardiovascular comorbidities and progressive respiratory decline. The presence of diaphragmatic involvement and bulbar dysfunction necessitates a proactive, multidisciplinary approach, including timely PEG consideration for nutritional support. The implementation of LTH-NIV and cough augmentation effectively manages hypercapnic respiratory failure, supporting stable longitudinal outcomes in this medically complex population.

P207 RESPIRATORY SUPPORT OF FACIOSCAPULOHUMERAL MUSCULAR DYSTROPHY

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10.1136/thorax-2026-BTSabstracts.367

Background Facioscapulohumeral muscular dystrophy (FSHD) is a rare genetic condition that causes progressive muscle weakness. Respiratory muscle involvement is rare, with only around 1% of patients requiring ventilatory support. Therefore, optimal respiratory management of FSHD is poorly described in the literature.

Aim To better understand the need for long-term respiratory support in a single centre cohort of patients with FSHD.

Methods We performed a retrospective case note review of 25 patients (10 men) with FSHD who were referred and followed up (median follow up 80 months) at our tertiary centre since 2007. We collected data on blood gases, sleep studies, non-invasive ventilation (NIV) and continuous positive airway pressure (CPAP) use.

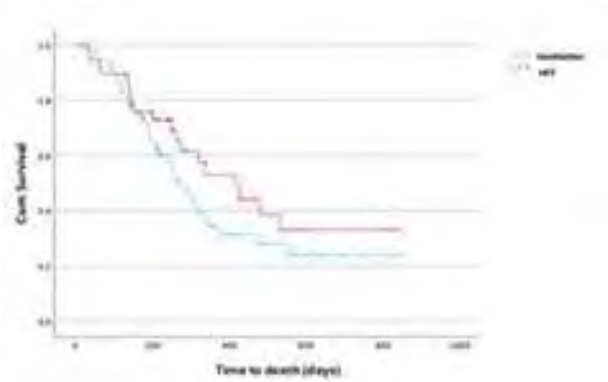
Results At the time of the first assessment, the prevalence of OSA and hypercapnia ($pCO_2 \geq 6kPa$) were 44% and 40%, respectively; and only further one patient developed hypercapnia during follow up. Four patients had bulbar symptoms, and 18 complained about respiratory symptoms, 14 patients had impaired cough peak flow. Hypercapnia was more frequent in those who had impaired cough peak flow and required cough augmentation (69 vs. 11%, $p<0.01$). Positive airway pressure (PAP) was commenced in 11 patients (2 CPAP, 9 NIV). Four patients were always adherent to PAP, 1 never and 6 showed variable adherence; and there was a trend for a relationship between PAP adherence and better hypercapnia control ($p=0.09$). Adherence did not relate to the presence of respiratory or bulbar symptoms, age, gender or comorbidities, but tended to higher in those with impaired cough peak flow ($p=0.06$). We recorded two deaths during the follow up (at 139 months and 95 months), both patients had hypercapnia and showed variable adherence to PAP; however, due to the low numbers no association between mortality and clinical factors could be explored.

Conclusions A multidisciplinary approach, including respiratory assessment should be part of the routine management of FSHD. As hypercapnia in FSHD is related to impaired cough strength, patients with frequent chest infections or difficulties with secretion management should be checked for hypercapnia.

P208 A SINGLE CENTRE EXPERIENCE OF NON-INVASIVE VENTILATION (NIV) AND HIGH FLOW THERAPY (HFT) FOR PATIENTS WITH MOTOR NEURON DISEASE (MND) UNDERGOING A PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG) INSERTION WITH PROPOFOL SEDATION

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10.1136/thorax-2026-BTSabstracts.368



Abstract P208 Figure 1

Introduction NICE MND guidance[1] recognises the importance of respiratory risk assessment and timely gastrostomy for patients living with MND but with limited guidance on the respiratory support and sedation strategy during the PEG insertion. Advancing muscle wasting, weakness with weight loss and declining respiratory function are associated with increased procedural risk.

Methods An electronic record system (EPIC) was retrospectively reviewed between January 2024- December 2025 for survival data and peri-procedural complications in MND patients undergoing a new PEG insertion at a tertiary complex home ventilation centre. Anaesthetic-led sedation was supported with NIV, tracheostomy or HFT as determined by the presence or absence of respiratory failure. All patients were discussed in a pre-screening MDT attended by the anaesthetic, gastroenterology, respiratory and nutrition teams.

Results 93 patients (65±9.9 years, BMI 22.7±3.9 kg/m2, 53% male) underwent a new PEG insertion. 56% (52) had limb onset presentation, 33% (31) bulbar onset, 6% (6) mixed onset and 4% (4) respiratory onset. 58% (53) were supported with NIV, 41% (38) with HFT and 3 were tracheostomy ventilated. Mean % weight loss at time of PEG was 15 ±9.1%.

92% (86/93) patients with a mean Americian Society of Anaesthesiologists (ASA) Score of 3.5 (n=57) were sedated using propofol (mean dosing 243±102mg) as led by an anaesthetist. Peri-procedural events included transient hypotension (19% managed with metaraminol/ ephedrine) and 6 % had transient oxygen desaturations. 2% of patients developed post-procedural pneumonia but there were no cases of new onset respiratory failure in the first 24 hours. Two patients had a peristomal haematoma and 4 cases of bleeding were reported.

Median survival was 319 days (95%CI 254-383). Ventilation supported survival was 276 days (95%CI 210-341) and HFT supported was 418 days (95%CI 265-570). 30-day survival was 99% with a 90-day survival of 90%.

See figure 1.

Conclusion These data demonstrate that anaesthetic-led sedation with propofol, with appropriately selected HFT or NIV for high-risk patients with motor neuron disease undergoing new PEG tube placement can be achieved safely when patients are pre-screened by an experienced MDT.

REFERENCE

1. Motor neuron disease: assessment and management. NICE guideline [NG42].

P209 ANALYSIS OF OUTCOME OF PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG) TUBE INSERTION UNDER NON-INVASIVE VENTILATION (NIV) SUPPORT FOR MOTOR NEURON DISEASE (MND) PATIENTS: A THREE-YEAR RETROSPECTIVE STUDY

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10.1136/thorax-2026-BTSabstracts.369

Introduction MND is a progressive neurodegenerative disease in which malnutrition is common. Percutaneous Endoscopic Gastrostomy (PEG) or Radiologically Inserted Gastrostomy (RIG) is routinely used to maintain nutritional status, but often requires NIV support during placement. We evaluated outcomes of PEG insertion in patients referred to the West Midlands Complex Home Ventilation Service.

Methods We conducted a retrospective analysis of patients referred to our regional ventilation unit for PEG/RIG insertion from April 2022 to April 2025. Data were collected using electronic case records.

Results 182 patients (98 males) with a median age of 68 years underwent PEG insertion during the above period. The mean weight was 66.9 kg. 167 patients were admitted electively, 10 were inpatient transfers, and 5 were emergency admissions. The median length of stay was 4 days. One patient failed both PEG and RIG and was transitioned to the palliative care pathway. 132 patients required NIV support during the procedure; 92 had been on NIV prior to admission, and 40 (39.2%) required NIV for the first time during gastrostomy insertion, of whom 10 (25%) were discharged on NIV after the intervention. A further 8 patients were initiated on domiciliary NIV during the admission but did not require NIV for gastrostomy insertion. The mean FVC and SNIP were 2.33 litres and -31 cm H_2O, respectively. 18 patients (9.9%) had complications following the procedure, of which 4 (2.2%) had major complications. 3 patients (1.6%) died during the admission, and 8 patients (4.4%) died within 30 days of PEG insertion. All patients who died had been referred for a new PEG tube insertion.

Discussion PEG insertion within a specialised ventilation unit is associated with low complication and mortality rates. NIV was well tolerated, including in patients requiring new initiation, the majority of whom were successfully weaned before discharge. These findings support the safety and feasibility of PEG insertion with NIV in appropriately selected patients with MND.

Abstract P209 Table 1

Parameters (April 2022 to April 2025)	Values
Total number of MND patients for PEG insertion	182
Male: Female	98:84
Median Age	68 years
Type of Admission	Elective – 167, IP transfer – 10, Emergency - 5
Mean weight	66.9 kg
Mean FVC	2.33 litres
Mean SNIP	-31 cm H_2O
Median length of stay	4 days
Type of procedure (PEG/RIG)	PEG – 177 (New – 170, Replacement – 11), RIG – 4, Failed both – 1
In-region / Out-of-Area referrals	42/140
NIV prior to admission	92
NIV support during procedure	132 (92 old + 40 new)
No of patients newly started on Domiciliary NIV during admission	18
Complications (Major/minor)	Major – 4, Minor – 14
Inpatient death	3 (Peritonitis sec. to perforation, Progression of MND, other)
30-day mortality	8
Total deaths	11 (7 females, 4 males);
Major complications	Organ injury, Bleeding, Other
Minor complications	Abdominal pain, Pneumoperitoneum, Pneumonia, Bleeding from PEG site, Haematoma, Fall, Covid, C.difficile infection

Outcome of PEG insertion in MND patients from a complex ventilation unit

P210 OXIMETRY PLUS PULSE WAVE TONOMETRY FOR DETECTING RESPIRATORY FAILURE IN PATIENTS WITH AMYOTROPHIC LATERAL SCLEROSIS (ALS); INTERIM REPORT

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10.1136/thorax-2026-BTSabstracts.370

Background Oximetry alone has proved unreliable for the detection of respiratory failure in patients with ALS. Some modern devices also add pulse wave tonometry (O2/PWT) allowing detection of changes in autonomic output and assessment of cardiopulmonary desynchrony which might prove more effective in identifying respiratory failure. This would allow for timely initiation of non-invasive ventilation (NIV). If O2/PWT were effective it would allow assessments to be done at home which would benefit patients who are often immobile.

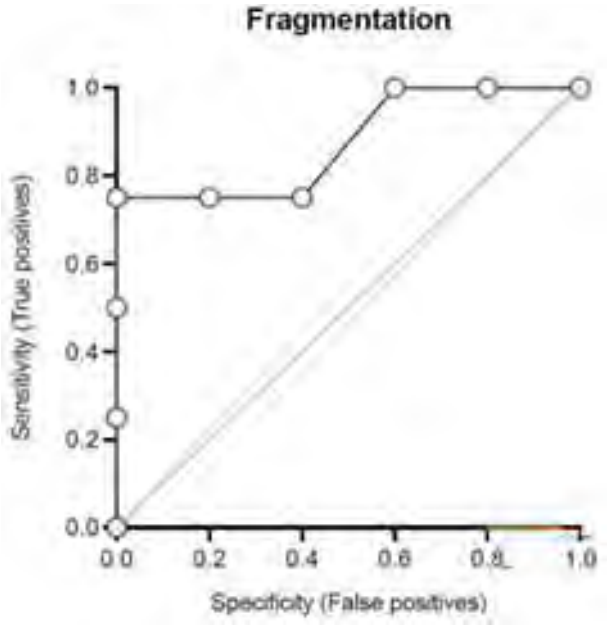
Objective To test the feasibility of using O2/PWT to assess respiratory failure in patients with ALS.

Methods The study received ethical approval, and patients gave informed consent to participate. The study was registered as NCT07502677. We studied consecutive patients with a neurology confirmed diagnosis of ALS not on NIV attending for inpatient assessment

We undertook standard clinical measures including transcutaneous CO2 (tCO_2) monitoring, maximal sniff nasal inspiratory pressure (SNIP), forced vital capacity (FVC) and daytime blood gases. O2/PWT was measured (SleepImage USA), the primary variable of interest was sleep quality index (SQI) and sleep Fragmentation (defined as $eLFC_{BB}$ >15%) as a secondary outcome.

A decision to initiate NIV was made the morning after by a consultant physician who did not have sight of the O2/PWT data. Receiver Operating Curve (ROC) analysis was undertaken to compare parameters of interest.

Results Of a planned recruitment target of 15, 10 patients have been recruited since March 2026, 1 was excluded due



Abstract P210 Figure 1

to technical failure. 9 data sets are therefore presented. Their median age was 64years and NIV was recommended in 4.

ROC analysis was completed. Amongst the measured parameters those with a positive predictor for NIV initiation were SNIP (AUC 1.0), bicarbonate (HCO_3) (AUC 0.95) and median tCO_2 (AUC 0.875). ROC analysis failed to show SQI as a positive predictor, however sleep fragmentation was predictive of being offered NIV (see figure 1, AUC=0.875).

Conclusion Although our study was incomplete at the time of submission, O2/PWT was feasible in ALS patients and could in principle be done at home. The data suggest sleep fragmentation may have value as a predictive test for being offered NIV in this context.

P211 LONGITUDINAL CLINICAL AND THERAPEUTIC TRAJECTORIES IN THOSE WITH MOTOR NEURONE DISEASE RECEIVING TRACHEOSTOMY VENTILATION AT A SPECIALIST COMMUNITY LONG-TERM VENTILATION UNIT: A RETROSPECTIVE QUALITATIVE STUDY

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10.1136/thorax-2026-BTSabstracts.371

Introduction Tracheostomy ventilation (TV) in motor neurone disease (MND) is rare in the UK (<1% of MND patients), frequently crisis-driven. Longitudinal data describing lived experience, symptom trajectories, and the evolving complexity of specialist care following TV are minimal. Our aim is to characterise the care needs of this complex population.

Methods A retrospective qualitativve longitudinal review of eight people living with MND (plwMND) admitted to a specialist community long-term ventilation unit between 2020 and 2025 requiring TV. Data were extracted from electronic clinical records using a structured framework reviewing ventilator management and respiratory care requirements, psychological wellbeing and mood, cognitive function, sleep disturbance, pain, acute hospital admissions, mobility, cuff deflation status, communication methods, route of nutrition and hydration, and advance care planning (ACP). TV duration was defined as time from tracheostomy insertion to discharge, death, or date of data extraction (April 2026). Reflexive thematic analysis and individual trajectory reconstruction were used to characterise longitudinal patterns.

Results The majority of plwMND underwent tracheostomy following an acute crisis (n=7). None had previously completed ACP. In this small mixed cohort, median duration of TV support was 22 months. PlwMND had varying requirements related to ventilator setting changes with no correlation to MND subtype or time from tracheostomy. All required airway clearance support. Most plwMND had significant saliva management needs requiring various treatment approaches, impacted by their individual goals. Interventions with glycopyrronium and/or botulinum toxin injections were dynamic and highly individualised over months to years. Therapy input supported quality of life and engagement in meaningful activities; cuff deflation enabled communication and oral intake for selected patients with MDT support. Data collection for acute admissions, pain, sleep, mood and infection domains is ongoing, and complete findings will be presented.

Conclusions Living with TV in MND is heterogeneous, intervention-intensive, and characterised by evolving symptom burden; not a uniform terminal trajectory. Oral secretion management requires ongoing multimodal adaptation and relates to patient goals and participation in meaningful activities. Physical and psychological burden is substantial and may be under-recognised in clinical practice. These findings highlight the value of specialist multidisciplinary infrastructure and iterative ACP in this population.

Opportunities to improve asthma care

P212 EVALUATION OF SCREENING TOOLS TO IDENTIFY INAPPROPRIATE SABA USE IN PATIENTS WITH ASTHMA: THE CHECK-SABA STUDY

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10.1136/thorax-2026-BTSabstracts.372

Objective This observational study aimed to evaluate the ability of two screening tools, a simplified set of quick rules(Quick Rule) and a comprehensive self-administered questionnaire, to detect inappropriate short-acting beta-agonist (SABA) use. Additionally, the prevalence of inappropriate SABA use according to current guideline recommendations(GINA and GEMA) was assessed. Methods: Consecutive adult outpatients with asthma using inhaled SABA as rescue therapy were recruited. All assessmentswere performed during a single visit. Patients completed an ad-hoc questionnaire evaluating SABA use. Investigators thenapplied a simplified 3-item Quick Rule. Finally, guideline-based clinical assessment was conducted and considered the reference standard for defining inappropriate SABA use. Results: Among 739 evaluable patients, 545 (73.7%) were classified as having inappropriate SABA use according to the

clinical assessment. The 3-item Quick Rule showed the best performance, with high agreement with the reference standard (κ=0.72) (see table 1). In contrast, the full self-questionnaire overestimated inappropriate use (98.4%, κ =0.06). Investigator-based judgement reduces its prevalence (59.90%), highlighting some discrepancies with guideline-defined criteria. Conclusion: Approximately three out of four asthma patients using SABA demonstrate inappropriate use according to current guidelines. A simple 3-item Quick Rule provides an accurate and pragmatic screening tool, showing strong agreement with clinical assessment and high sensitivity for detecting inappropriate use. Its implementation in routine care could facilitate early identification of at-risk patients and improve alignment with guideline-based asthma management.

P213 USING FENO TO PREDICT THE RISK OF ASTHMA EXACERBATIONS: IMPACT OF 'OTHER' FACTORS

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10.1136/thorax-2026-BTSabstracts.373

Introduction Fractional exhaled nitric oxide (FeNO) is a marker of airway inflammation. High FeNO levels have been associated with an increased risk of future asthma exacerbation.[1] NICE recommends use of FeNO in diagnosing and monitoring asthma.[2] We elected to review the real-time impact of non-asthma factors on FeNO and, on the future exacerbation risk within 12 months.

Methods We identified 100 patients that were reviewed in the outpatient asthma clinic. Baseline data such as smoking status, FeNO and treatment including compliance was collected. Electronic patient record was used to record the number of asthma exacerbations in community and hospital over 12 months.

Results The average FeNO was lower in smokers compared with non-smokers (20.6 vs 42.8). Smokers were more likely to have an asthma exacerbation (55.6% vs 51.6%). BMI ≥30 was associated with a lower average FeNO (34.4 vs 44.8) but a higher frequency of exacerbations in 12 months (average 1.9 vs 1.4).

In patients with FeNO <50, 41 (56.9%) had at least 1 exacerbation. 19 (26.4%) had ≥3 exacerbations. 9 (12.5%) patients had a breathing pattern disorder (BPD). 5 (55.6%) of these had ≥3 exacerbations in 12 months. 7 (9.7%) were known to have anxiety related symptoms. 2 (28.6%) of these had ≥3 exacerbations. 5 (6.9%) had vocal cord dysfunction. 2 (40%) of these had ≥3 exacerbations.

In those with FeNO ≥50, 10 (35.7%) had at least 1 exacerbation in 12 months. 4 (14.3%) had ≥3 exacerbations. 1 (3.6%) patient had BPD and 2 (7.1%) had anxiety related symptoms. These patients had <3 exacerbations.

Conclusion Obesity and smoking are associated with a lower FeNO but a higher risk of future asthma exacerbation. Anxiety related symptoms and breathing pattern disorders are associated with a higher exacerbation risk even in patients with FeNO <50. Therefore, FeNO should be used as an adjunct to a comprehensive asthma review.

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P214 DEVELOPING INTERVENTIONS TO TACKLE ETHNICITY-BASED DISPARITIES IN ASTHMA MANAGEMENT IN BRITISH SOUTH ASIANS 'TEAMS STUDY'

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10.1136/thorax-2026-BTSabstracts.374

Background asthma affects ~8% of the British South Asian (SA) population. SA patients are more likely to be deprived and experience poor clinical outcomes. Self-management is a key driver of asthma control and multiple factors including cultural, social, religious and poor health literacy have been postulated as reasons underpinning poor clinical outcomes.

Objectives to determine facilitators and barriers for asthma management amongst British SA patients and to develop culturally tailored supportive interventions for asthma management for British SA patients.

Methods SA patients (≥16 years; n=30) with physician-diagnosed asthma and HCPs (n=30) including respiratory consultants, general practitioners, nurses and physiotherapists with experience in management of SA patients participated in semi-structured interviews to identify facilitators/barriers in asthma management. Interviews were transcribed verbatim and analysed using reflexive thematic analysis. A behavioural analysis of data was conducted, using a 'person based' approach for intervention development, employing the Behaviour Change Wheel and COM-B model to develop a logic intervention model. Further assessment of the intervention was conducted involving 2 focus groups of 9 SA patients and 9 HCP.

Results the themes of barriers identified from SA patient and HCP interviews were: socioeconomic and environmental, illness and medication perceptions, mental health and emotional burden, treatable traits and comorbidities, socio-cultural, demographic and family-related factors, low social awareness of asthma, and healthcare access and communication.

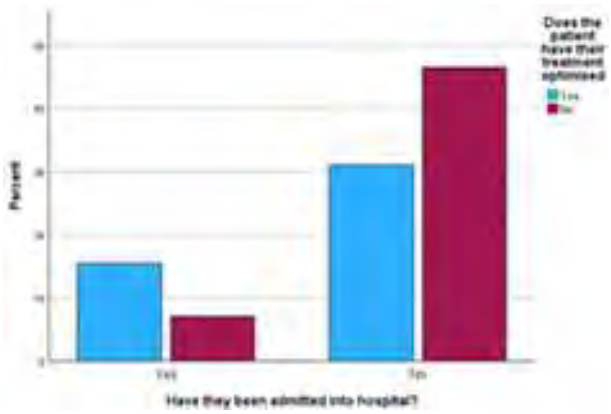
A culturally supportive composite multi-modal interventions was developed.The SAATHI tool (South Asian Asthma Treatment, Help and Information) iscomprisedofpatient information, infographics in simple native language, podcasts, and YouTube videosand aninteractive artificial intelligence tool with an Avatar that maintains identity with patients and aligns with age, sex, ethnicity, language including dialect and literacy to maximise patient engagement and treatment adherence. Focus groups of patients and HCPs supported the SAATHI tool and emphasised the need for targeting the intervention at patients, theirfamilies and communities.

Conclusion Culturally tailored composite multimodal interventions are needed to address health inequalities and inequities in the management of asthma amongst British SAs. The developed SAATHI will require refinement and co-production of the content and future testing for clinical and cost effectiveness in an RCT prior to its implementation.

P215 AN EVALUATION OF INHALER OPTIMISATION IN ASTHMA PATIENTS DURING HOSPITAL ATTENDANCE: CLINICAL EFFECTIVENESS, SUSTAINABILITY AND PATIENT INVOLVEMENT

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10.1136/thorax-2026-BTSabstracts.375



Abstract P215 Figure 1 Treatment optimisation in A&E-only vs ward-admitted patients Graph showing treatment optimisation in A&E-only (left) and ward admitted (right) patients. Blue represents the patients that have had their treatment optimised and red represents patients who have not had their treatment optimised

Failing to optimise asthma treatment for patients can lead to exacerbations which have both clinical and environmental consequences. Hospital attendance can provide an opportunity to optimise inhaler treatment and technique in line with the 2024 NICE/BTS/SIGN asthma guidelines, which could lead to clinical and sustainability benefits.

The aim was to determine whether asthma patients have their treatment optimised in line with the 2024 updated asthma guidelines during an A&E or hospital admission, whether this has sustainability benefits and patients' opinion of inhaler changes.

A retrospective review of electronic patient records of all adult patients attending A&E with asthma between November 2025 and March 2026 was carried out. Data was recorded and analysed using Microsoft Excel and SPSS v30. Patients were sent an electronic post-discharge questionnaire via Microsoft Forms through the hospital patient app, to review their hospital experience. Carbon footprint estimations were conducted using PrescQIPP data to assess the carbon impact of inhaler use.

71 patients were identified who had attended A&E. 16 of those were admitted to the ward. 46.5% of patients had their treatment optimised in line with current asthma guidance. Optimisation was significantly higher (68.8%) in admitted patients compared to patients visiting A&E only (40.0%) (X^2=8.56, p=0.014) (figure 1). 50% of ward-admitted patients received an asthma bundle review compared to only 3.6% of patients who just attended A&E (X^2=22.0, p<0.01). The 10 questionnaire respondents had a median inhaler confidence score of 10 (range: 6-10). Switching to new guideline-led treatment could cause a reduction of 31,662 gCO2e per patient per year.

Inhaler optimisation was not carried out for all patients. Patients admitted onto the ward were more likely to be optimised compared to A&E-only patients, suggesting a major gap in A&E care. Carbon footprint estimates indicated that guideline-led treatment can reduce carbon emissions and limited questionnaire findings suggested that patient involvement remains important in asthma care. Outcomes have supported the introduction of pre-labelled inhalers and education packs in A&E for optimisation during discharge. Structured pathways in hospitals are necessary to ensure full asthma optimisation.

P216 HIGH ICS/FORMOTEROL INHALER UTILISATION AND POTENTIAL MEDICINES OVERSUPPLY: A NATIONAL PRESCRIBING ANALYSIS 2022–2025

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10.1136/thorax-2026-BTSabstracts.376

Background Anti-inflammatory reliever (AIR) and Maintenance-and-Reliever Therapy (MART) strategies are recommended to improve asthma outcomes and reduce short-acting β_2-agonist (SABA) overuse. High ICS/formoterol utilisation may identify patients at risk of poor disease control, inappropriate prescribing, or medicines oversupply. We evaluated trends in high ICS/formoterol inhaler utilisation during increasing adoption of AIR/MART prescribing.

Methods Pseudonymised ICS/formoterol data for England, 2022–2025, was extracted under the NHS England–PrescQIPP CIC data sharing agreement and analysed. Clinical indication, treatment regimen and inhaler usage data were unavailable; high dispensing volumes were interpreted as markers of potential excessive use or oversupply rather than confirmed inhaler use. Patients receiving ≥16 inhalers annually were categorised as high-utilisation users and stratified into 16–23, 24–35, 36–51 and ≥52 inhalers/year. Relative risks (RR) were calculated using 2022 as the reference year.

Results The proportion of patients receiving ≥16 ICS/formoterol annually declined from 4.7% in 2022 to 4.1% in 2025; an 11% relative reduction (RR 0.89; χ^2 p<0.001), whilst absolute patient numbers increased by 30%, from 80,879 to 105,539.

The largest subgroup comprised patients receiving 16–23 inhalers annually, increasing from 61,754 to 81,566 (+32.1%). Patients receiving 24–35 inhalers/year increased from 18,215 to 22,901 (+25.7%), while very high utilisation (36–51 inhalers/year) increased from 798 to 961 (+20.4%). More than 100 patients each year received ≥52 inhalers annually.

In 2025, 23,973 patients received ≥24 inhalers annually, including 1,072 receiving ≥36 inhalers. These represented

Abstract P216 Table 1

Annual ICS/Formoterol Inhalers	Clinical Interpretation	2022 n (%)	2023 n (%)	2024 n (%)	2025 n (%)
16–23 inhalers/year	Above expected utilisation/over-supply	61,754 (3.55%)	65,183 (3.47%)	73,100 (3.53%)	81,566 (3.15%)
24–35 inhalers/year	Marked utilisation/over-supply	18,215 (1.05%)	18,915 (1.01%)	21,017 (1.01%)	22,901 (0.88%)
36–51 inhalers/year	Severe utilisation/over-supply	798 (0.046%)	827 (0.044%)	909 (0.044%)	961 (0.037%)
≥52 inhalers/year	Extreme utilisation/over-supply	112 (0.006%)	119 (0.006%)	115 (0.006%)	111 (0.004%)
Total ≥16 inhalers/year	Potential MART utilisation/over-supply	**80,879 (4.65%)**	**85,044 (4.53%)**	**95,141 (4.59%)**	**105,539 (4.08%)**

<1% of all ICS/formoterol users, but accounted for a disproportionate burden of prescribing.

Conclusions Although the proportion of high-utilisation ICS/formoterol users declined since 2022, more than 100,000 patients continued to receive ≥16 inhalers annually and almost 24,000 received ≥24 inhalers. These findings identify a substantial population exposed to high ICS/formoterol supply volumes that may reflect excessive reliever use, medicines oversupply, poor disease control or suboptimal prescribing. Even if some prescribing relates to COPD, the highest dispensing categories remain suggestive of potential oversupply and warrant further investigation. Targeted review of high-utilisation patients may improve outcomes and reduce avoidable healthcare costs.

P217 AIRWAY VARIABILITY ACROSS METHACHOLINE CHALLENGE AND BRONCHODILATOR RECOVERY IMPROVES SENSITIVITY FOR DIAGNOSING ASTHMA

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Introduction Bronchodilator reversibility (BDR) and methacholine challenge testing (MCT) are established tools for diagnosing asthma. However, due to crossover effects, these tests need to be conducted across two separate visits. Whether post-MCT BDR can serve as a surrogate for an independent BDR or MCT remains unknown.

Aims i) Determine the diagnostic concordance between post-MCT BDR and an independent BDR. ii) Determine the diagnostic performance of post-MCT BDR FEV_1 variability ≥20% for diagnosing asthma, compared to standard PD_{20} criteria.

Methods 93 patients (52 with Asthma, 41 without asthma) from the RADicA study, underwent standard BDR testing and, on a separate occasion, MCT. Asthma diagnosis (reference standard) was made by a panel of asthma specialists using clinical history, physical examination, spirometry, BDR, FeNO, peak expiratory flow variability, MCT, allergy testing, blood eosinophils, and response to 8 weeks inhaled corticosteroid treatment. Sensitivity and Specificity was calculated for BDR FEV_1 >12%, Post-MCT BDR >12%, MCT PD_{20} <200ug, and post-MCT BDR FEV_1 variability ≥20% [minimum FEV_1 post-MCT to maximum FEV_1 post-BDR or baseline (whichever is larger)].

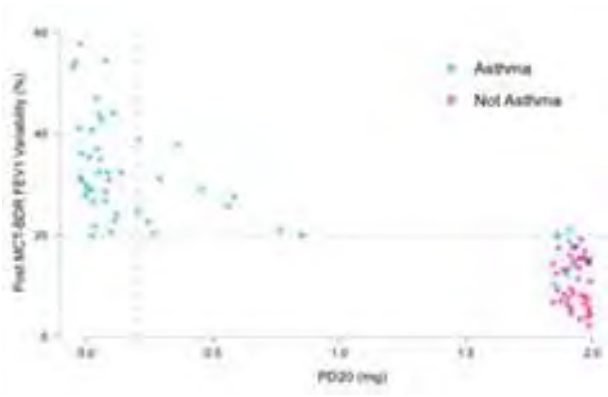
Results i) Post-MCT BDR FEV_1 remained below baseline in half of individuals and only two participants demonstrated >12% increase from baseline, compared to 13 when BDR was conducted independently. Consequently, sensitivity was very poor (table 1). Post MCT-BDR FEV_1 variability ≥20% diagnosed an additional 12 individuals with asthma, incl. 1 false positive, compared to PD_{20} <200ug (figure 1). Consequently, sensitivity improved whilst specificity reduced slightly (table 1).

Discussion Post-MCT BDR cannot be used as a substitute for an independent BDR. However, Post-MCT BDR FEV_1 variability ≥20% is a more sensitive criterion for diagnosing asthma than PD_{20} <200ug and retains very good specificity. Airway variability across methacholine challenge and bronchodilator recovery detects asthma in individuals who show partial bronchoconstriction to MCT and partial BDR, but do not meet established criteria for either and should be considered as an additional diagnostic criterion.

Abstract P217 Table 1

	Sensitivity, % (95% CI)	Specificity % (95% CI)
Independent BDR FEV_1 > 12%	25 (14-39)	100 (91-100)
MCT PD_{20} < 200ug	63 (49-76)	100 (91-100)
Independent BDR AND/OR MCT	69 (55-81)	100 (91-100)
Post MCT-BDR FEV_1 >12%	5 (1-16)	100 (93-100)
Post MCT-BDR FEV_1 variability ≥20%	85 (72-93)	97 (87-100)

BDR, bronchodilator reversibility; MCT, methacholine challenge test



Abstract P217 Figure 1

P218 BARRIERS AND FACILITATORS TO IMPLEMENTING ANTI-INFLAMMATORY RELIEVER-BASED ASTHMA CARE IN COMMUNITY PHARMACY: A QUALITATIVE STUDY

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10.1136/thorax-2026-BTSabstracts.378

Background and Aim Community pharmacists are well positioned to support implementation of recent UK asthma guidelines NG254 BTS/NICE/SIGN for anti-inflammatory reliever (AIR)-based regimens, requiring a shift away from short-acting beta-agonist (SABA)-based reliever inhalers. However, their role in delivering guideline-aligned asthma care and the challenges influencing implementation within routine practice, remain unclear.

Aim To explore community pharmacists' experiences of asthma care following updated UK guidelines, with a focus on role, barriers and facilitators related to AIR-based treatment.

Methods Theoretically underpinned, semi-structured interviews were conducted with community pharmacists in England. Data were analysed thematically using NVivo V15 software.

Results Thirteen community pharmacists participated, including six who also worked within general practice. Participants represented a range of organisational contexts, including large national chains and independent pharmacies across England.

Pharmacists described their role in asthma care as largely centred around contractual services, including new medication reviews, patient education, checking inhaler technique and advising the patient to re-visit primary care where concerns were identified. Participants emphasised their frequent patient contact, which created opportunities to support ongoing

asthma management. However, this was limited by their lack of authority to modify treatment.

Barriers to care delivery were: high workloads, time constraints, medication shortages and queue pressures, poor or variable knowledge of asthma guidelines, alongside limited learning opportunities and availability of guidance, fragmented systems, including limited access to clinical information beyond often incomplete Summary Care Records, competing demands and organisational targets, the challenges of patient expectations and reliance on SABA medication and emotional burdens of professional frustration, fatigue and workload stress.

Facilitators were accessibility and strong relationships with patients (although constrained by prescribing and escalation capabilities), the ability to identify high-risk patients (but without clear mechanisms to act, strong communication and counselling skills and rewarding opportunities to make a difference for patients.

Conclusions Current systems do not consistently enable community pharmacists to contribute fully to guideline-aligned asthma care. More integrated systems and proactive approaches, such as community pharmacy led reviews and emergency dispensing audits are needed to maximise the contribution of community pharmacy to improving asthma outcomes.

P219 CAN HOME SPIROMETRY AND FENO TESTING IMPROVE ASTHMA DIAGNOSIS? – A FEASIBILITY STUDY

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10.1136/thorax-2026-BTSabstracts.379

Introduction Although temporal variation is the hallmark of asthma, recommended diagnostic approaches largely rely on single clinic-based measurements. Ambulatory monitoring captures diurnal and day-to-day variability and may therefore enhance diagnostic accuracy. We evaluated the clinical feasibility and potential utility of home spirometry and fractional exhaled nitric oxide (FeNO) monitoring in asthma diagnosis.

Methods Symptomatic, untreated adults with GP-suspected asthma underwent diagnostic tests including bronchodilator reversibility, in-clinic FeNO, blood eosinophil counts and bronchial challenge. Participants measured spirometry and FeNO four times daily over one week; during the second week spirometry were measured twice daily. The reference standard was provided (asthma/not-asthma) by an expert panel of at least two asthma specialists based on clinical history and the results of all in-clinic testing; home spirometry (except for peak expiratory flow) and FeNO measurements were blinded to the panel.

Results Of 67 eligible participants, 51(76%) were recruited, and 38 had asthma confirmed or excluded by the panel. 1058 home spirometry measurements were obtained from 37(73%) participants; 848 home FeNO readings were obtained from 39(76%) participants. Among those completing at least one home measurement, median (IQR) adherence was 66.7(58.6-97.6)% for spirometry and 78.5(51.8-103.6)% for FeNO. Collection of health impact data for economic evaluation was feasible. In participants with a confirmed diagnostic outcome who completed home measurements (FeNO: n=32; spirometry: n=28), the putative home-testing metrics demonstrated high sensitivities at ≥90% specificity, and outperformed peak expiratory flow diurnal variability. Incorporating home testing into the BTS/NICE/SIGN 2024 diagnostic pathway had the potential to reduce reliance on bronchial challenge testing by 65%.

Conclusions Home spirometry and FeNO testing and the prospective collection of health-economic data in the diagnostic setting were feasible. Home-based testing strategy showed early potential to improve asthma diagnosis and pathway efficiency. These findings support further evaluation through an adequately powered diagnostic accuracy study and health-economic assessment.

P220 DEPEMOKIMAB DEMONSTRATES SUSTAINED LONG-TERM EFFICACY AND CONSISTENT PATIENT-REPORTED OUTCOMES OVER 2 YEARS IN PATIENTS WITH TYPE 2 ASTHMA IRRESPECTIVE OF BASELINE INHALED CORTICOSTEROID DOSE: AN INTEGRATED ANALYSIS OF THE PHASE III SWIFT-1/-2 AND THE OPEN-LABEL EXTENSION AGILE STUDIES

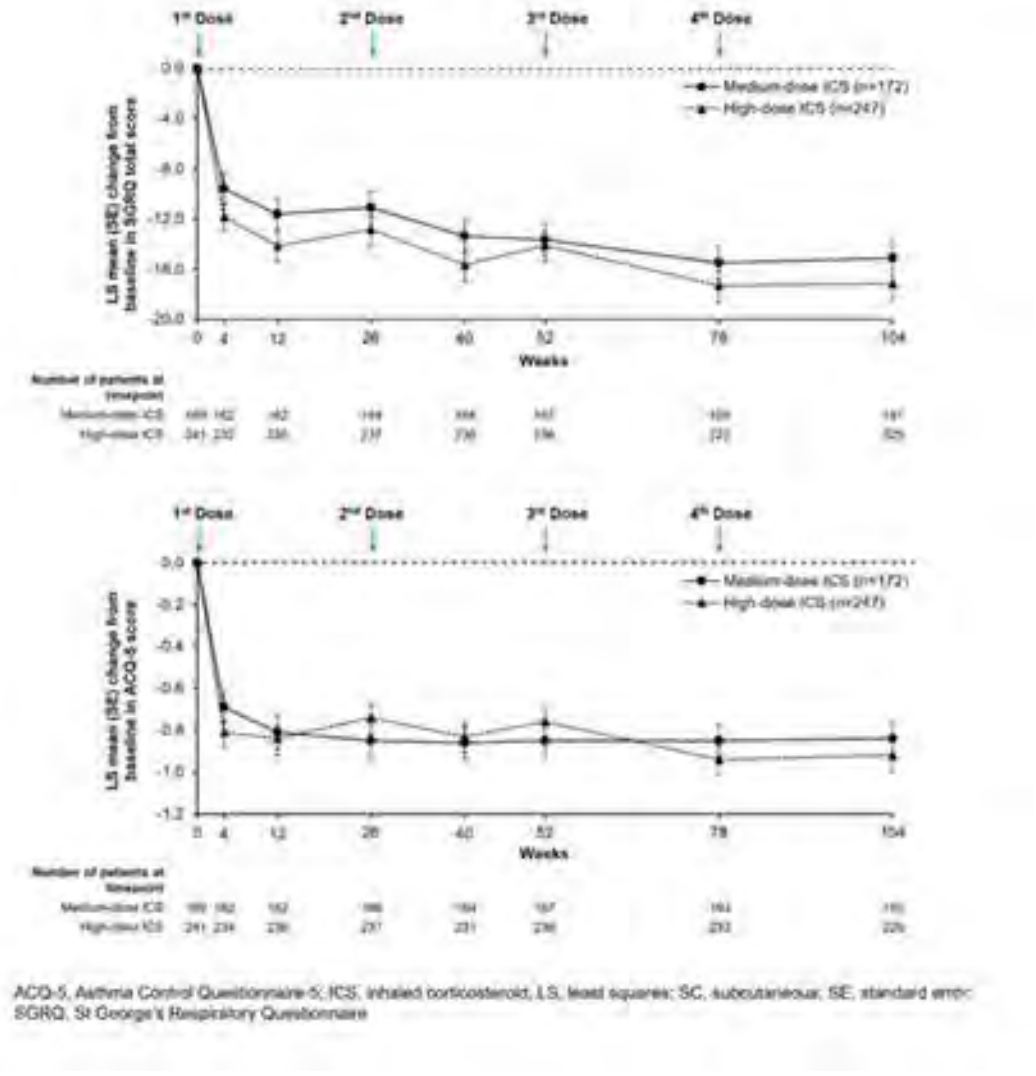
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10.1136/thorax-2026-BTSabstracts.380

Rationale High-dose inhaled corticosteroids (ICS) are associated with increased side effects and earlier initiation of biologics is desirable. 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 asthma. Depemokimab efficacy was demonstrated over 52 weeks in patients with type 2 asthma in the Phase III SWIFT-1/-2 studies. This integrated post hoc analysis of the SWIFT-1/-2 and AGILE open-label extension (OLE) studies evaluated the long-term efficacy of depemokimab by baseline ICS dose in patients with type 2 asthma.

Methods In SWIFT1/2, patients ≥12 on medium/highdose ICS were randomized 2:1 to depemokimab 100 mg or placebo every 26 weeks for up to 52 weeks. Completing patients entered AGILE OLE, continuing or switching to depemokimab for up to 104 weeks. Outcomes included annualised exacerbation rate (AER) and LS mean (SE) change from baseline (CFB) in SGRQ total score and ACQ-5 score by baseline ICS use (medium/high) over 2 years.

Results In SWIFT1/2 (N=762), depemokimab reduced exacerbations by 54% vs placebo (RR 0.46 [0.36,0.59]); 52week AER was 0.36 (0.27,0.48) for mediumdose and 0.65 (0.53,0.79) for highdose ICS. Improvements in SGRQ and ACQ-5 scores were observed with depemokimab regardless of baseline ICS dose: LS mean (SE) CFB at Week 52 with depemokimab in the medium- and high-dose ICS subgroups, respectively, was –14.38 [1.19] and –13.60 [1.01] for SGRQ and –0.89 [0.07] and –0.76 [0.06] for ACQ-5. 641 (84%) entered AGILE; 629 received ≥1 depemokimab dose (n=419 depemokimab/depemokimab; n=210 placebo/depemokimab). At baseline, 172 and 247 in the depemokimab/depemokimab group were on medium- and high-dose ICS. In the depemokimab/depemokimab group, AER was maintained over 2 years in both the medium- (0.41 [0.32, 0.52]) and high-dose (0.62 [0.52, 0.75]) ICS subgroups. CFB in SGRQ



ACQ-5, Asthma Control Questionnaire-5; ICS, inhaled corticosteroid; LS, least squares; SC, subcutaneous; SE, standard error; SGRQ, St George's Respiratory Questionnaire

Abstract P220 Figure 1 SGRQ and ACQ-5 scores were sustained in patients in the depemokimab/depemokimab group throughout the 2-year period irrespective of baseline ICS dose (N=419)

and ACQ-5 scores were sustained throughout the 2-year period for both medium- and high-dose ICS subgroups (figure 1).

Conclusions Twice-yearly depemokimab demonstrated sustained efficacy over 2 years, irrespective of baseline ICS dose. These results support the long-term efficacy of depemokimab in asthma and its potential initiation prior to high-dose ICS escalation.

Funding GSK (SWIFT-1/-2:206713/213744, NCT04719832/NCT04718103; AGILE:212895/NCT05243680).

Previously presented at ATS 2026.

P221 PHENOTYPING SEVERE ASTHMA ATTACKS IN HOSPITALISED PATIENTS

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10.1136/thorax-2026-BTSabstracts.381

Background Asthma attacks are important events associated with significant morbidity and mortality but are poorly understood. We aimed to describe the phenotypes of suspected severe asthma attacks in hospitalised patients during attacks and at recovery.

Methods This was a single centre observational study. We assessed patients as soon as possible after hospital admission and 2 weeks after discharge (recovery) with spirometry, oscillometry (FOT), exhaled nitric oxide (FeNO), blood eosinophils, induced sputum and Acute Asthma Quality of Life. Airflow obstruction was defined as FEV_1/FVC <0.7 and/or R5 >5 cmH_2O/L/s. Events were classified as likely Acute Asthma-Related Admissions (LAARA) if airflow obstruction was present. Attacks were classified as T2-high if FeNO ≥25ppb and/or blood eosinophil count ≥0.4 $x10^9$/L and/or sputum eosinophil count ≥3%.

Results 100 patients were recruited, 83 completed both lung function measures. 64/83 (77%) had airflow obstruction (LAARA). Of these 16/64 (25%) had obstruction on FOT, 21/64 (33%) on spirometry and 27/64 (42%) on both spirometry and FOT. 38/64 (59%) LAARA were T2-high but 25 (25.8%) of the cohort had taken corticosteroids prior to recruitment. Those with T2-high attacks demonstrated greater $\%FEV_1$ improvement at recovery than those with T2-low (mean Δ%FEV1 24% vs 9%).18/38 (47%) of LAARA were T2-high at recovery.

Conclusion 1/5 suspected asthma attacks had no evidence of airflow obstruction. Our results suggest heterogeneity of inflammatory phenotypes in attacks and greater improvement in lung function after corticosteroid treatment in those with T2-high attacks. Nearly 50% of likely attack patients were T2-high 2 weeks after hospital discharge and may be at higher risk of further attacks.

Advances in thoracic surgery and tobacco harm reduction

P222 ECHOCARDIOGRAPHIC PROBABILITY OF PULMONARY HYPERTENSION AND THE ASSOCIATION WITH 90-DAY MORTALITY AND POST-OPERATIVE COMPLICATIONS IN PEOPLE UNDERGOING LUNG CANCER RESECTION SURGERY

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10.1136/thorax-2026-BTSabstracts.382

Introduction Pulmonary hypertension (PH) may be associated with increased mortality in people undergoing lung resection.[1]
Aim To find the incidence of echocardiographic probability of PH in people undergoing lung cancer resection and its association with 90-day post-operative mortality and complications.
Method Retrospective observational data was collected from June 2023 to June 2025 for people undergoing lung cancer resection surgery. People with radiological diagnosis and subsequent negative histology were excluded. Echocardiographic PH probability was classified as intermediate/high (PH group) or low (non-PH group).
Results A total of 444 people were included in the study. However, only 250 people (56.3%) had echocardiographic evidence of PH grading (low to high) and hence were used in the final analysis.

The incidence of PH, defined as intermediate/high echocardiographic probability, was 10.0% (25/250). Mean tricuspid regurgitation velocity was 2.28 ± 0.599 m/s (n = 131).

People with PH had slightly higher rates than those without PH of: pulmonary complications (32.0% vs 29.3%), non-pulmonary complications (40.0% vs 27.1%), Clavien–Dindo grade ≥3 (24.0% vs 20.0%), and 90-day mortality (4.0% vs 2.2%). However, the differences were not statistically significant.

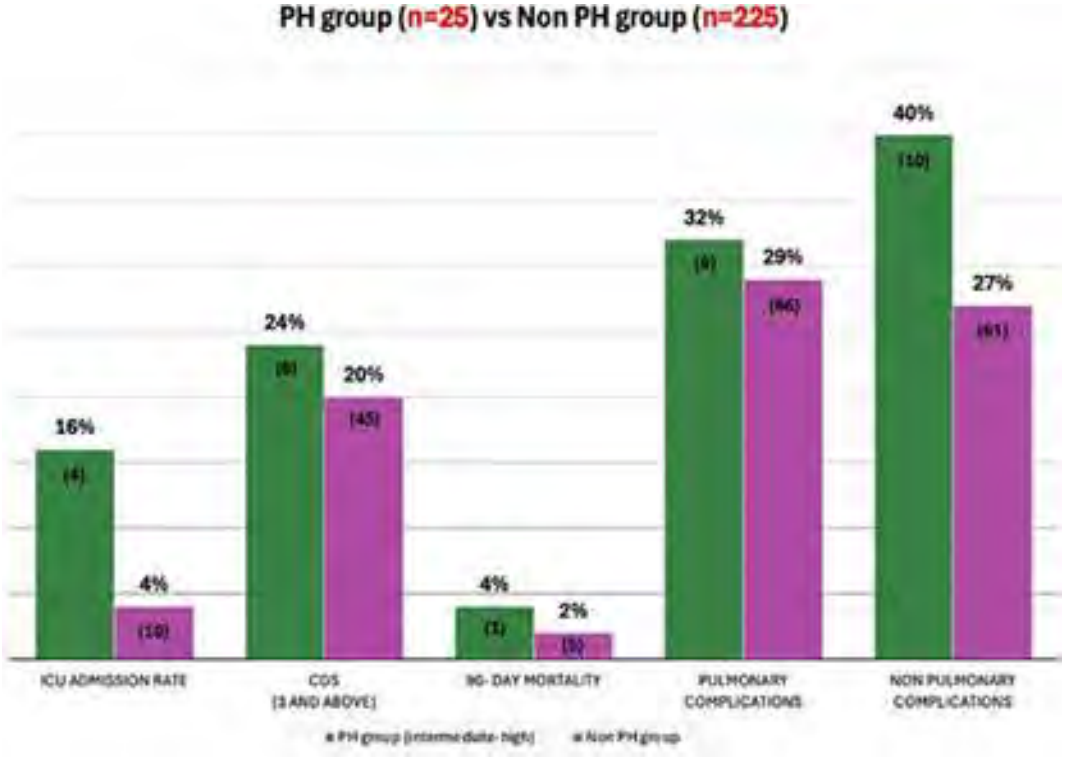
Mean DAOH90 was lower in the PH group than the non-PH group (77.4 ± 20.7 vs 81.1 ± 13.7 days, respectively).

Mean ICU length of stay was higher in the PH group than the non-PH group (0.72 ± 2.62 vs 0.16 ± 1.38 days, respectively) and demonstrated a weak but statistically significant positive correlation with PH grade (Spearman's rho = 0.153, p = 0.015).

The PH group had a higher rate of ICU admissions, with 16% of patients (4/25) requiring ICU care compared with 4.44% (10/225) in the non-PH group. This was statistically significant (p = 0.017).
Conclusions People with PH exhibited higher rates of post-operative pulmonary and non-pulmonary complications and 90-day mortality, although these differences were not statistically significant. PH was, however, significantly associated with increased ICU stay and rate of admission.

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Abstract P222 Figure 1

P223 LONG-TERM BRONCHOSCOPIC MANAGEMENT OF LOCALISED TRACHEOBRONCHIAL AMYLOIDOSIS

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10.1136/thorax-2026-BTSabstracts.383

Introduction and Objectives Tracheobronchial AL amyloid deposition is a rare disorder causing airway stenosis, respiratory symptoms and a need for recurrent airway intervention. Data describing long-term management, procedural burden, and the role of adjunctive therapies remain limited.[1] We aimed to characterise cohort demographics, anatomical distribution and airway interventions performed in patients with airway amyloidosis managed at a tertiary centre.

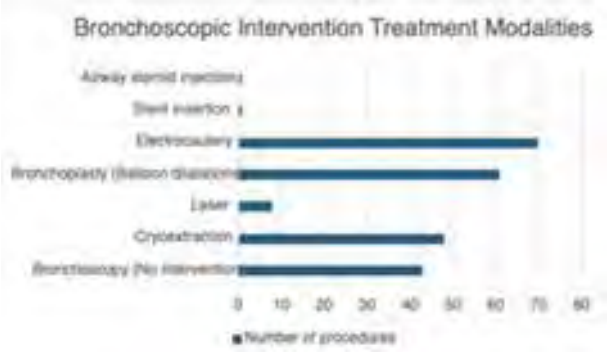
Methods We retrospectively analysed consecutive patients with tracheobronchial amyloidosis undergoing endobronchial assessment and treatment at our tertiary centre between 2015-2026. Demographic characteristics, smoking history, comorbid respiratory disease, disease distribution, therapeutic interventions, and outcomes were extracted from clinical records. Bronchoscopic procedures and use of endoluminal brachytherapy were reviewed.

Results Twenty-two patients were included: 12 male (55%), 77% Caucasian with a median age of 63.5 years (IQR 50–68.5) at first bronchoscopy. Twelve (54.5%) were ever-smokers and 45% had co-existing respiratory disease.

Disease was confined to the airways in 77% of patients. Central airway involvement was present in 82%, subglottic involvement in 32%, bilateral disease in 68%, and 64% had disease affecting three or more lobar bronchi.

A total of 153 bronchoscopies were performed: median of 7 procedures per patient and mean of 1.5 procedures per treatment year. Forty-three (28%) procedures required surveillance alone, 110 (72%) involved therapeutic intervention. Management combined interventional bronchoscopy to treat amyloid deposition and maintain airway patency; alongside adjuvant endoluminal brachytherapy to target the underlying B-cell clone.

Bronchoscopic intervention included electrocautery (70 procedures, 45.8%), balloon bronchoplasty (61, 39.9%), cryoextraction (48, 31.4%) and laser therapy (8, 5.2%). Only one patient required endoluminal stenting. Endoluminal brachytherapy was delivered to 15 patients (68%), accounting for 38 treatments with a mean of 2.5 sessions per treated patient.



Abstract P223 Figure 1 Bar graph to show Interventional bronchoscopy treatment modalities for managing airway amyloid deposition

Conclusions Tracheobronchial AL amyloidosis is characterised by extensive central airway disease and a high long-term procedural burden, with patients requiring repeated multimodal bronchoscopic intervention and adjuvant endoluminal brachytherapy. These findings support patient management within a specialist centre with access to advanced airway therapies and multidisciplinary expertise.

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P224 INVESTIGATING REGIONAL VARIATION IN REFERRAL FOR LUNG VOLUME REDUCTION: WHO SENDS US WHAT?

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Introduction Lung volume reduction (LVR) via endobronchial valves or surgery leads to improvement in quality of life and mortality for selected individuals with COPD. In the UK, LVR is delivered via specialist centres; regional referral practices differ. Despite the potential benefits of LVR, it is an under-utilised procedure.

Aims and Objectives We wished to investigate whether variation exists amongst hospitals referring individuals for LVR, and if we could identify systemic factors influencing it.

Methods We reviewed all referrals to our tertiary cardiothoracic centre LVR service over 1 year. We adjusted referral numbers to the size of the 12 referring hospital trusts' catchment, COPD prevalence, and average driving distance to our centre. Between-hospital referral rate variation was assessed using a likelihood ratio test.

Results In 2024, we were referred 116 cases, of which 86 were unique. Referrals per 100,000 population ranged from 0.29 to 5.13; referrals per estimated COPD population (catchment multiplied by prevalence) ranged from <0.01 to 0.2. Greater than 15-fold variation existed between the lowest and highest referrers ($P<0.001$), suggesting unbalanced referral rates. There was no significant relationship between referral rate and average driving time from our centre ($R^2=0.09$), regional COPD prevalence ($R^2=0.01$) or hospital size measured by bed capacity ($R^2=0.33$).

Conclusions Marked variation exists in LVR referral in our region, not explicable by hospital size, location or COPD prevalence. Some hospitals with the highest prevalence of COPD and socioeconomically deprived catchments had the lowest referral rates, suggesting these populations may be under-served. Further work is needed to explain this variation, address modifiable factors and improve clinician education on LVR.

P225 REAL-WORLD OUTCOMES OF A MULTIDISCIPLINARY HYPERINFLATION SERVICE FOR SEVERE EMPHYSEMA

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10.1136/thorax-2026-BTSabstracts.385

Introduction Severe emphysema-related hyperinflation significantly contributes to dyspnoea, exercise limitation and impaired quality of life. Lung volume reduction (LVR) strategies, including endobronchial valves (EBVs) and lung volume reduction surgery (LVRS), can improve symptoms and functional outcomes in carefully selected patients, making robust multidisciplinary team (MDT) patient selection essential.

Aim To review outcomes of patients discussed at a specialist hyperinflation MDT in a large teaching hospital between 2022 to 2025.

Methods A retrospective analysis was conducted of all patients discussed at hyperinflation MDT over a three-year period. Demographic data, MDT recommendations, intervention outcomes and radiological assessments were reviewed.

Results Eighty-one patients were discussed, of whom 48 (59%) were male. Mean age was 63 years (range 41-85). Following MDT review, 17 (21%) patients were considered for EBVs, 25 (31%) for LVRS, and 2 (3%) for bullectomy. Ultimately 9 patients underwent EBV insertion and 7 underwent surgery.

Thirty-seven (45%) patients were deemed unsuitable for intervention. Reasons included referral for lung transplantation (n=5), development of lung cancer (n=4), failure to meet LVR eligibility criteria (n=20), patient preference to decline treatment (n=5) and death before intervention (n=3).

StratX analysis was performed in 24 patients. Fissure completeness (complete/partially deficient/incomplete) was observed as follows; right oblique fissure10/9/5, right horizontal fissure 2/4/18 and left oblique fissure 18/5/1. Among 44 patients with planned or completed interventions, target lobes were; right upper lobe (n=20), right upper and middle lobes (n=1), right lower lobe (n=1), left upper lobe (n=11) and left lower lobe (n=11). All EBVs were performed by physicians under general anaesthesia using combined rigid and flexible bronchoscopy. 2 patients developed pneumothorax post EBV, all other procedures were uncomplicated.

Conclusions Our findings support multidisciplinary hyperinflation services as an effective framework for delivering LVR in severe emphysema. Right upper lobe was the most common target for LVR, and left oblique fissure was most frequently intact. LVRS remains a common MDT recommendation. Improved awareness of LVR eligibility criteria among respiratory physicians may enhance referral appropriateness and facilitate timely access to treatment.

P226 SMOKING CESSATION IN COPD: A MIXED-METHODS ANALYSIS OF BEHAVIOURAL, SOCIOECONOMIC AND HEALTHCARE DETERMINANTS IN REAL-WORLD PRACTICE

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10.1136/thorax-2026-BTSabstracts.386

Background Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide, with smoking representing the primary modifiable risk factor for disease progression.[1] Despite clear clinical benefits associated with cessation, a substantial proportion of patients continue smoking following diagnosis, suggesting that smoking persistence in COPD is influenced by complex behavioural, socioeconomic and healthcare-related factors.[2] This study aimed to investigate both smoking behaviour and the barriers and facilitators to smoking cessation, with interpretation informed by specialist consultation and patient and public involvement (PPI) discussions.

Methods A retrospective analysis of anonymised primary care data from the DataLoch Trusted Research Environment involving 57,079 COPD patients, was conducted to examine smoking prevalence across demographic, socioeconomic and clinical subgroups. Experiential perspectives were gathered through consultation with a smoking cessation specialist and PPI group feedback from people living with COPD. These perspectives were used to support interpretation and contextualisation of the quantitative findings.

Results 39% of patients were current smokers. A strong socioeconomic gradient was observed, with smoking prevalence decreasing from 50% in the most deprived quintile to 24% in the least. Smoking prevalence remained persistently high across disease severity groups, suggesting that increasing disease burden alone did not reliably translate into smoking cessation. Current smokers demonstrated significantly higher hospitalisation rates compared to non-smokers ($p<0.001$). PPI discussions and specialist perspectives highlighted addiction, psychological distress, social environment, stigma and inconsistent engagement with cessation services as key contributors to continued smoking behaviour. Comparison of quantitative findings with specialist and lived experience perspectives demonstrated strong alignment between deprivation-associated smoking patterns, patient-reported behavioural and healthcare barriers.

Conclusion Smoking appears to be strongly shaped by socioeconomic disadvantage, behavioural dependence and healthcare system factors. These findings highlight the need for flexible, psychologically informed and inequality-sensitive smoking cessation strategies, tailored towards high-risk COPD populations.

Abstract P226 Table 1 Triangulated findings identifying barriers and facilitators to smoking cessation in patients with COPD

Domain	PPI Findings	Service Provider Findings	Quantitative Link	Integrated Interpretation	Alignment
Addiction / Behaviour	Smoking viewed as habitual; relapse after reduction common.	Repeated quit attempts and relapse described as normal.	Smoking prevalence remained high across all severity groups (39–44%).	Persistent smoking reflects chronic nicotine dependence despite disease burden.	
Psychological Factors	Motivation fluctuated; shame and fear of failure common.	Readiness to quit strongly influenced engagement and relapse.	Smoking prevalence highest in middle age (~63%).	Emotional distress and relapse cycles reinforce continued smoking.	
Social Environment	Family and peer support could help or hinder quitting.	Social context strongly influenced cessation success.	Strong deprivation gradient (50% vs 24%).	Social inequality significantly influences smoking behaviour and cessation ability.	
Healthcare Professional Interactions	Clinician support varied; accountability was valued.	Advice from a Trusted Neutral Health Professional often triggered quit attempts.	Smoking prevalence varied widely across GP practices (12.3–92.5%).	Variation in clinician engagement may influence smoking outcomes.	
Service / System Factors	Rigid programmes and limited long-term support criticised.	Service variation and waiting times affected access.	Geographic and practice-level variation observed.	Service structure influences accessibility and effectiveness of support.	
Treatment / Intervention Factors	Gradual reduction preferred over abrupt quitting.	Behavioural support and flexible approaches viewed as important.	Smoking persisted in high-risk COPD groups.	Current interventions may not align with patient preferences.	

Evidence Alignment:

Green – Strong Convergence

Orange – Partial Convergence

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P227 THE PRESCRIPTION OF HOME OXYGEN TO PEOPLE WHO VAPE: A CROSS-SECTIONAL SURVEY STUDY

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Introduction and Objectives While there is anecdotal evidence of fires associated with vape use in the presence of home oxygen therapy (HOT), there is no United Kingdom guidance on the prescription of HOT to people who vape.

Our project identifies and characterises the practice and opinions of current Home Oxygen Service (HOS) prescribers on prescription of HOT to people who vape.

Methods A cross-sectional electronic survey study with a Microsoft Forms questionnaire was sent to HOT prescribers in England, using purposive and snowballing sampling. The questionnaire comprised mainly closed Likertscale items centring on prescribers' practices and opinions, supplemented by an openended question to capture freetext responses.

Results There were 195 respondents from all English regions. 14.4% reported prescribing HOT to current cigarette smokers;

27% of respondents reported prescribing HOT to current vapers. Northeast England reported the highest rate of prescribing to vapers (46% of respondents). Those who prescribed to vapers were significantly more likely to prescribe to smokers also (37% vs 7%, p<0.001).

Those prescribing HOT to vapers most commonly reported lack of evidence of risk (96%) and that risk assessments were being undertaken on a case-by-case basis (96%). Those not prescribing HOT to vapers most commonly cited concern over fire risk (94%). 10% reported experience of adverse events. Most respondents (76%) reported being either 'Unhappy' or 'Completely Unhappy' about prescribing to vapers; even among those prescribing to vapers, only 10% reported being 'Happy' or 'Completely Happy' about prescribing.

Free text responses characterised variation and inconsistency in HOT policies for people who vape, exacerbated by safety concerns and practical monitoring challenges. Numerous participants reported the lack of regional or national guidance and, consequently, the need for casebycase decisionmaking in the face of potential considerable risk.

Conclusion Our survey demonstrated national variation in prescribing practice to people who vape. HOS team members, including those who do currently prescribe HOT to people who vape have concerns over this practice, Future work should focus on local or national guidelines to support HOS team members in addition to examining specific risk profiles in HOT for people who vape.

P228 NICOTINE DEPENDENCE AMONG RESPIRATORY THERAPY STUDENTS IN SAUDI ARABIA: FINDINGS FROM A NATIONAL CROSS-SECTIONAL SURVEY

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10.1136/thorax-2026-BTSabstracts.388

Introduction Nicotine dependence among university students remains a public health concern, and psychological factors such as self-esteem may influence dependence severity. Evidence among respiratory therapy students in Saudi Arabia is limited. Hence, this study aimed to assess the relationship between nicotine dependence and self-esteem among respiratory therapy students in Saudi Arabia.

Methods This cross-sectional study was conducted between March 13 and September 16, 2025 and included 1,041 respiratory therapy students. Inclusion criteria were enrollment from second academic year through internship year; first-year students were excluded. Data were collected via an anonymous online survey assessing smoking history, nicotine dependence using the Fagerström Test for Nicotine Dependence (FTND), and self-esteem using the Rosenberg Self-Esteem Scale (RSES). Negative binomial regression was used to identify factors associated with nicotine dependence.

Abstract P228 Table 1 Multivariable negative binomial regression predicting nicotine dependence, measured by the continuous fagerström test for nicotine dependence (FTND) score among respiratory therapy students in Saudi Arabia, a cross-sectional study conducted from March to September 2025 (N = 1,041)

Predictor	Adjusted IRR	95% CI	p-value
Gender (reference: Female)			
Male	1.018	0.959–1.081	0.557
Smoking history (reference: < 1 year)			
1–5 years	1.103	1.026–1.186	0.008
6–10 years	1.148	1.041–1.267	0.006
>10 years	1.305	1.068–1.596	0.009
Monthly income (reference: < 1,000 SAR)			
1,000–3,000 SAR	1.014	0.943–1.091	0.701
>3,000 SAR	0.961	0.878–1.052	0.389
University sector (reference: Governmental)			
Private	1.022	0.955–1.095	0.525
Cumulative GPA (reference: 2.00–2.99)			
3.00–3.99	0.882	0.765–1.017	0.084
4.00–5.00	0.882	0.765–1.018	0.085
Academic level (reference: First year)			
Second year	1.117	0.927–1.347	0.245
Third year	1.115	0.924–1.344	0.256
Fourth year	0.946	0.781–1.147	0.573
Bridging	1.071	0.661–1.735	0.780
Intern	1.026	0.829–1.270	0.814
Absent days (reference: No absences)			
1–2 days	0.957	0.859–1.067	0.430
3–4 days	0.912	0.811–1.026	0.126
5–6 days	0.974	0.830–1.142	0.742
>6 days	0.798	0.563–1.129	0.202
Academic warnings (reference: No warnings)			
1 warning	1.028	0.929–1.136	0.598
2 warnings	1.059	0.950–1.180	0.300
3 warnings	1.148	0.969–1.360	0.111
>3 warnings	1.226	0.787–1.909	0.368
Self-esteem (Rosenberg Self-Esteem Scale total score)	0.972	0.952–0.993	0.011

Abbreviations: IRR, incidence rate ratio; CI, confidence interval; FTND, fagerström test for nicotine dependence; RSES, Rosenberg self-esteem scale; SAR, Saudi Riyal

Results The median FTND score was 5 (interquartile range [IQR]: 3–6). Smoking duration was significantly associated with higher nicotine dependence in a dose–response pattern. Compared with students who smoked for less than one year, those with 1–5 years (adjusted incidence rate ratio [IRR] = 1.10, 95% confidence interval [CI]: 1.03–1.19; p = 0.008), 6–10 years (IRR = 1.15, 95% CI: 1.04–1.27; p = 0.006), and more than 10 years of smoking (IRR = 1.31, 95% CI: 1.07–1.60; p = 0.009) had higher dependence scores. Higher self-esteem was independently associated with lower nicotine dependence, with each one-point increase in RSES score linked to a 3% reduction in FTND score (IRR = 0.97, 95% CI: 0.95–0.99; p = 0.011). No demographic or academic variables were significantly associated with nicotine dependence.

Conclusion Nicotine dependence among respiratory therapy students is strongly related to smoking duration and self-esteem. Further longitudinal and interventional studies are needed to clarify the temporal relationship between self-esteem and nicotine dependence and to determine whether psychosocial support could complement tobacco control efforts in university settings.

Keywords Nicotine dependence; Self-esteem; Respiratory therapy students; Saudi Arabia; Smoking

P229 THE IMPACT OF VARENICLINE IN COMBINATION WITH NICOTINE REPLACEMENT THERAPY AND SPECIALIST SUPPORT ON MORBIDITY AND HEALTHCARE UTILISATION FOR INPATIENTS WITH TOBACCO DEPENDENCY

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10.1136/thorax-2026-BTSabstracts.389

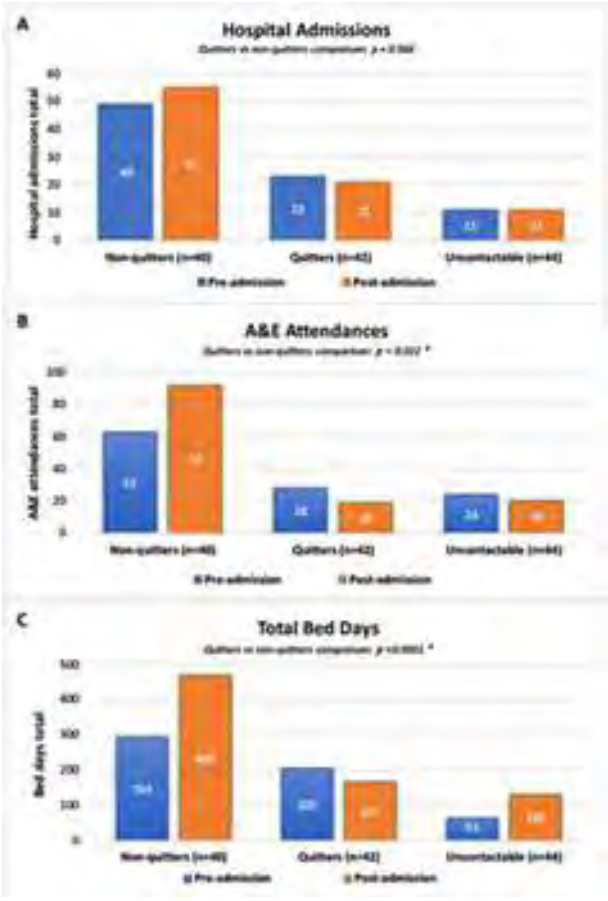
Introduction and Objectives Tobacco dependence (TD) is a leading cause of morbidity and healthcare utilisation. Varenicline, an effective treatment for TD, was unavailable in the UK between 2021 and late 2024. This study evaluated the impact of reintroducing varenicline for inpatients, prescribed alongside nicotine replacement therapy (NRT) and Tobacco Dependency Treatment (TDT) support, on quit rates and healthcare utilisation in an inner-city hospital with a 19% inpatient TD prevalence.

Methods We reviewed the notes of all inpatients prescribed varenicline between December 2024 and May 2025. Self-reported quit rates were assessed at three months. Hospital admissions, total bed-days and A&E attendances were compared for one year before and after the inpatient admission where varenicline was prescribed, excluding patients who died.

Results 126 inpatients were prescribed varenicline; 108 (86%) with combination NRT. Mean tobacco exposure was 51 pack-years. 56/126 (44%) had raised carboxyhaemoglobin on admission. 54/126 (43%) had COPD with mean (SD) FEV1 1.3 (0.68) L; 46(0.19)% predicted FEV1 (n=44).

At 3-month review, four patients had died. 49% of contactable patients (40/82) were self-reported ex-smokers. Nine patients (7%) died within one year.

The number of bed-days decreased amongst those who quit smoking post-varenicline, falling from 205 days to 167 days, while non-quitters had an increase from 293 to 469 days - a significant between-group difference ($p<0.001$). Similarly, A&E attendances declined amongst those who quit from 28 to 19 visits per year, whereas they increased amongst non-quitters from 63 to 92 visits per year ($p<0.02$). Hospital admissions decreased slightly in those who quit, from 23 to 21 per year, whilst increasing from 49 to 55 for non-quitters, however this was not statistically significant (figure 1).



Abstract P229 Figure 1 Bar charts showing (A) hospital admissions (B) A&E attendances and (C) total bed days in the 12 months before and after inpatient stay where varenicline was prescribed for quitters, non- quitters and uncontactable patients at 3 months (excluding patients who died). P-values calculated using chi-squared analysis. * indicates statistical significance

Conclusions Varenicline, in combination with NRT and TDT support, is an effective treatment for inpatients with TD. We found that quitting smoking using varenicline reduced inpatient bed-days and A&E attendances over the subsequent year, with a trend towards a reduction in hospital admissions, when compared with those who did not quit.

Varenicline can have a significant impact on morbidity and healthcare utilisation and should be routinely offered to inpatients alongside combination NRT and TDT input as treatment of TD.

Improving every breath: pulmonary rehabilitation advances

P230 EFFECTIVENESS OF PULMONARY REHABILITATION ACROSS INDIVIDUAL CHARACTERISTICS: FINDINGS FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME

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10.1136/thorax-2026-BTSabstracts.390

Introduction Pulmonary rehabilitation (PR) is a highly evidenced intervention for individuals living with COPD. Individual characteristics influence referral to PR, but their associations with PR outcomes are unknown in real-life settings.

Aim To explore the effectiveness of PR across individual characteristics using a National Respiratory Audit Programme (NRAP) dataset for England and Wales.

Methods 52,669 individuals with COPD who were assessed for PR from March 2019 - September 2023 and entered into the NRAP database, completed a PR programme. Outcomes investigated were the incremental shuttle walk test (ISWT), six-minute walk test (6MWT), endurance shuttle walk test (ESWT), COPD assessment test (CAT) and Chronic Respiratory Questionnaire (CRQ). Linear mixed effects models fitted with a random intercept for PR service and adjusted for the initial assessment score were used to predict PR outcomes across individual

Abstrcat P230 Table 1 Predicted changes in pulmonary rehabilitation outcome measures in service users with COPD that completed pulmonary rehabilitation by characteristic. The results for all characteristics (other than the characteristic specified) are averaged. 6WMT: six-minute walk test; ISWT: incremental shuttle walk test; ESWT: endurance shuttle walk test; CAT: COPD assessment test; CRQ: Chronic respiratory questionnaire: MCID: minimal clinically important difference

	Predicted changes (95% confidence intervals)				
	6MWT (metres) (n=12,628)	ISWT (metres) (n=19,829)	ESWT (seconds) (n=3,141)	CAT (points) (n=34,295)	CRQ (points) (n=15,728)
MCID	30 metres	35 metres	174 seconds	-2.0 points	+2.0 points
Gender					
Male	44 (38, 50)	58 (52, 63)	228 (171, 283)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.7)
Female	44 (38, 50)	56 (51, 62)	221 (167, 275)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.7)
Other	44 (38, 50)	63 (50, 76)	203 (39, 445)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.7)
Age (years)					
<60	51 (43, 60)	62 (53, 70)	224 (120, 328)	-2.9 (-2.6, -3.3)	2.9 (2.6, 3.3)
60-64	50 (41, 58)	61 (52, 68)	217 (116, 318)	-2.9 (-2.5, -3.2)	2.9 (2.5, 3.2)
65-69	47 (39, 55)	60 (52, 68)	223 (120, 326)	-3.0 (-2.7, -3.3)	2.9 (2.6, 3.2)
70-74	44 (36, 51)	59 (51, 67)	220 (117, 321)	-2.6 (-2.3, -2.8)	2.5 (2.2, 2.8)
75-79	36 (29, 44)	57 (50, 65)	210 (113, 307)	-2.4 (-2.1, -2.7)	2.2 (1.9, 2.5)
≥80	34 (26, 42)	56 (49, 63)	210 (112, 308)	-2.2 (-1.9, -2.5)	1.9 (1.6, 2.2)
Ethnicity					
White	44 (37, 50)	58 (51, 66)	226 (129, 323)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
Asian	44 (37, 50)	59 (51, 67)	226 (118, 336)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
Black	44 (37, 50)	61 (52, 69)	197 (61, 333)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
Mixed	44 (37, 50)	60 (52, 68)	229 (110, 348)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
Other	44 (37, 50)	57 (49, 65)	209 (64, 354)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
Medical Research Council Dyspnoea Score					
1 (least severe dyspnoea)	31 (15, 48)	36 (16, 56)	234 (183, 285)	-1.6 (-0.8, -2.4)	2.0 (1.3, 2.7)
2	42 (35, 49)	39 (32, 47)	227 (181, 272)	-2.2 (-2.0, -2.5)	2.4 (2.1, 2.6)
3	47 (41, 53)	52 (46, 59)	218 (174, 262)	-2.9 (-2.7, -3.0)	2.7 (2.5, 2.9)
4	50 (43, 56)	72 (64, 80)	210 (168, 252)	-3.2 (-3.0, -3.3)	2.8 (2.6, 3.0)
5 (most severe dyspnoea)	49 (40, 57)	90 (78, 102)	199 (156, 239)	-3.4 (-3.1, -3.8)	2.8 (2.4, 3.1)
Index of Multiple Deprivation Quintile					
1 (most deprived)	44 (37, 50)	59 (51, 66)	217 (117, 317)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
2	44 (37, 50)	59 (52, 67)	218 (117, 319)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
3	44 (37, 50)	59 (51, 67)	220 (119, 323)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
4	44 (37, 50)	59 (51, 67)	214 (116, 314)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)
5 (least deprived)	44 (37, 50)	59 (51, 67)	216 (116, 316)	-2.7 (-2.5, -2.8)	2.5 (2.3, 2.8)

characteristics. The following characteristics were included as co-variates: age, gender, ethnicity, Medical Research Council (MRC) dyspnoea grade and index of multiple deprivation (IMD) quintile. The model for each outcome measure was used to calculate estimated marginals means for the predicted change for each individual characteristic.

Results Overall, all groups of individual characteristics gained significant benefits in all outcomes after completing PR (all $p<0.01$). The minimal clinically important difference (MCID) was achieved across all characteristics in the 6MWT, ISWT and ESWT (table 1). The MCID was met by all characteristics in the CAT except MRC grade 1 and by all characteristics in the CRQ except age ≥80 years. Younger service users and higher MRC groups demonstrated greater improvements in the 6MWT, ISWT, CAT and CRQ than older individuals ($p<0.01$) or lower MRC groups ($p<0.01$). There were no significant differences in PR effectiveness across genders, ethnicities or IMD quintiles ($p>0.05$).

Conclusions Service users across all individual characteristics achieved clinically meaningful improvements following PR in all outcome measures. These findings support the need for equitable referral and access to PR for individuals living with COPD.

P231 DESIGNING THE UPTURN INTERVENTION TO SUPPORT UPTAKE OF PULMONARY REHABILITATION: A COLLABORATIVE CO-DESIGN PROCESS

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Rationale and Aims Pulmonary Rehabilitation (PR) is a gold-standard treatment for people with chronic lung conditions, combining education and structured exercise. Despite its benefits, PR referral uptake remains low among people with Chronic Obstructive Pulmonary Disease (COPD), particularly those from minoritised ethnic groups. Our NIHR-funded UPTURN study is co-designing and evaluating a behavioural intervention, delivered by Asthma & Lung UK, to support PR uptake. This paper reports the co-design process and the underlying behaviour change theory informing our Prepare for PR intervention.

Methods This qualitative co-design study used the COM-B model and Behaviour Change Wheel to develop the intervention's theory of change and intervention materials. We conducted primary data collection with COPD patients and carers of Bangladeshi and African and Caribbean heritage (n=17) through focus groups and interviews. These findings were triangulated with a systematic review and secondary analysis of an additional UK dataset of predominantly White British COPD patients (n=41). Barriers and enablers to PR uptake were analysed using COM-B and mapped to evidence-based behaviour change techniques (BCTs). This analysis informed the design brief for a multi-stage co-design process. A core team of co-designers—including patients, clinicians, user-experience and engineering designers, and researchers from UCL and Asthma & Lung UK (n=21)—developed the intervention iteratively, progressing from ideation to lo-fi and high-fi prototyping through design-led activities.

Findings Key barriers included limited understanding of PR benefits, low confidence exercising with breathlessness, emotional burden (stress and worry), and practical access challenges. Participants from minoritised ethnic groups additionally highlighted reliance on family, varying trust in healthcare, and lack of cultural relevance in aspects of PR. The resulting Prepare for PR intervention comprises a support information pack, educational and testimonial patient-facing videos, and a helpline support phone call delivered by Asthma & Lung UK. These components incorporated the mapped BCTs such as social comparison, credible sources, information about health consequences, social support, and demonstration of behaviour.

Conclusions Prepare for PR provides a theoretically grounded and culturally sensitive approach to supporting PR uptake. The co-designed components will now undergo formal evaluation to assess feasibility, acceptability, and impact on engagement across diverse COPD populations.

P232 POST EXACERBATION PULMONARY REHABILITATION: HAS THE POPULATION CHANGED?

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Introduction Post-exacerbation Pulmonary Rehabilitation (PR) is a cost-effective, evidence-based intervention offered to patients admitted to hospital. Despite strong guideline recommendations, uptake remains low due to barriers such as limited referral, patient frailty, and access issues.

Design An observational, cross sectional service evaluation of patients admitted to hospital with an exacerbation of chronic obstructive pulmonary disease (COPD) during April 2024-2025. 1315 patients were offered PR as part of the routine care bundle.

Main Clinical Findings A needs analysis was conducted using routinely collected quantitative data to identify factors which would predict those not suitable for PR following an exacerbation of COPD.

Main Outcomes Sample demographics are presented in table 1. Chi square tests showed statistically significant associations between readmission to hospital, use of oxygen therapy (ambulatory (ABOT) and long-term (LTOT)), MRC, Frailty, age and FEV_1 with referral to PR; effect sizes (Cramér's V) indicated a moderate association for MRC and Frailty (V = 0.193 & V = 0.216 respectively) as well as age (V = 0.243) and FEV_1 (V = 0.331), and small associations for readmission (V = 0.116) and oxygen use (ABOT & LTOT) (V = 0.108 & V = 0.172 respectively).

Bonferroni post hoc analysis of the multiple comparisons revealed a significant mean difference in frailty, MRC, LTOT, readmission & age ($p = 0.033$) between individuals who declined the referral and those for whom the referral was deemed not suitable. Additionally, a larger mean difference in the same variables and ABOT ($p < 0.001$) was observed between those who had a referral completed and those for whom the referral was deemed not suitable.

Abstract P232 Table 1 Demographics of 2024-2025 Post exacerbation dataset

	Whole Group (n= 1315)	Declined PR (n = 634)	Not appropriate for PR (n=241)	Referral Completed (n = 409)
Age	70.1 ± 10.6	70.3 ± 10.3*$	72.6 ± 10.4*	68.4 ± 10.7$
Gender				
Male	684 (52.1%)	318 (50.2%)	123 (51%)	227 (55.5%)
Female	628 (47.9%)	316 (49.8%)	118 (49%)	182 (44.5%)
Smoking status				
Current	488 (37.4%)	245 (38.6%)	77 (32.2%)	160 (39.2%)
Ex-smoker	804 (61.6%)	381 (60.1%)	161 (67.4%)	243 (59.6%)
Never	14 (1.1%)	8 (1.3%)	1 (0.4%)	5 (1.2%)
Clinical Frailty Score (IQR)	5 (4-6)	5 (4-6)* $	6 (5-6)*	5 (4-5) $
1	5 (0.4%)	2	2	1
2	30 (2.3%)	13	5	12
3	165 (12.7%)	67	17	75
4	265 (20.4%)	128	19	112
5	434 (33.5%)	237	59	128
6	326 (25.1%)	163	95	68
7	62 (4.8%)	22	32	7
8	3 (0.2%)	1	3	
9	7 (0.5%)		6	
MRC (IQR)	4 (3-4)	4*$	4 (4-5)*	4 (3-4) $
1	11 (0.8%)	4	5	2
2	70 (5.4%)	26	11	33
3	261 (20.1%)	109	26	122
4	640 (49.3%)	340	82	202
5	316 (24.3%)	153	114	46
FEV_1	49.6 ± 19.4	48.5 ± 18.9$	49.5 ± 21.3	51.6 ± 18.9$
No. of admissions in previous 12 months (IQR)	2 (1-4)	2 (1-4)	2 (1-4)	2 (1-3)
Readmission	206 (15.7%)	127 (20.1%)*$	30 (12.4%)*	46 (11.3%)$
Ethnic Origin				
White British	1198 (91.1%)	583 (92%)	218 (90.5%)	371 (90.7%)
Asian British	29 (2.2%)	13 (2.1%)	8 (3.3%)	6 (1.5%)
White other background	32 (2.4%)	17 (2.7%)	6 (2.5%)	9 (2.2%)
Mental Health status				
Anxiety	165 (13%)	81 (13.1%)	25 (10.9%)	57 (14.4%)
Depression	127 (10%)	55 (8.9%)	31 (13.5%)	38 (9.6%)
Severe mental impairment	38 (3%)	11 (1.8%)	8 (3.5%)	18 (4.5%)
Dementia/ cognitive impairment	8 (0.6%)	2 (0.3%)	5 (2.2%)	-
On Long term oxygen therapy (LTOT)	162 (12.4%)	83 (13.1%)*$	53 (22%)*	25 (6.1%)$
On Ambulatory Oxygen therapy (ABOT)	81 (6.2%)	43 (6.8%)	25 (10.4%)	13 (3.2%)
Referral to PR				
Not appropriate	241 (18.5%)	-	-	-
Referral completed	409 (31.4%)	-	-	-
Declined	634 (48.6%)	-	-	-
Already enrolled	20 (1.5%)	-	-	-
Reason not referred to PR				
Not interested/ refused	344 (33.2%)	343 (56%)	1 (0.4%)	
Already referred	178 (17.2%)	-	-	
Carer responsibility	3 (0.3%)	3 (0.5%)	-	
Works full time	16 (1.5%)	16 (2.6%)	-	
Transport	11 (1.1%)	11 (1.8%)	-	
Done before and knows what to do	153 (14.8%)	153 (25%)	-	
Done before and found too hard	66 (6.4%)	66 (10.8%)	-	
Clinical Frailty Score >4	183 (17.6%)	12 (2%)	171 (71.8%)	
Already enrolled	8 (0.8%)	-	-	
mMRC > 3	3 (0.3%)	-	3 (1.3%)	
Language barrier	2 (0.2%)	1 (0.2%)	1 (0.4%)	
mMRC <3 and too fit	21 (2.0%)	1 (0.2%)	20 (8.4%)	
Patient has a disability	48 (4.6%)	7 (1.1%)	41 (17.2%)	

*significant difference between baseline characteristics in the declined rehab and not appropriate; $significant difference between baseline characteristics in the declined rehab and those that were referred; $p \leq 0.05$

Conclusions Several variables were associated with patients considered not suitable for referral to PR, with the strongest associations observed for MRC, frailty and age. These along with readmission and oxygen use had significant differences between those that had a referral to PR and those not suitable. Despite changes in the patient population, PR remained suitable for most patients (79%). The higher rate of patient refusal compared with clinical unsuitability indicates that optimising referral timing and engagement may be more important than reconsidering eligibility criteria.

P233 SYSTEMATIC REVIEW OF INTERVENTIONS TO IMPROVE REFERRAL AND UPTAKE OF PULMONARY REHABILITATION IN PATIENTS WITH COPD

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Abstract P233 Table 1

Studies	Study design	Population	Diagnosis of COPD	Setting	Intervention	Control	Outcome definition
Referral and uptake							
Barker et al, 2021 (United Kingdom) (9)	RCT	AECOPD	AECOPD	Primary care	Patient co-designed education video as an adjunct to usual care	Usual care; COPD discharge bundle including standardized verbal information about PR + information leaflet.	Referral received
Soharpal et al, 2024 (United Kingdom) (8)	RCT	COPD	Confirmed diagnosis of COPD and with moderate to very severe airflow limitation	Secondary care	Intervention based on cognitive behavioural approach and self-management support	Usual care and PR education	Referral sent
Barker et al, 2021 (United Kingdom) (10)	Prospective cohort study	AECOPD	Admitted to hospital with AECOPD	Hospital	COPD discharge bundle consisting of assessment of suitability for and referral to PR, delivered by PR and non-PR practitioners	Usual care	Referral received
Referral only							
Roberts et al, 2015 (United Kingdom) (11)	CCT	COPD	Diagnosis of COPD	Primary care	Patient held COPD scorecard with 6 quality indicators, to discuss at COPD review. Helpline for queries.	Usual care including a self- management plan, rescue pack distribution and instruction part of a regional QI intervention.	Referral
Kim et al, 2025 (United States) (12)	Cohort study	AECOPD	Spirometry	Hospital	Clinical decision support embedded into clinical EHR, pathway to support referral	Pathway not accessed or used	Referral received
Uptake only							
Alsumrain et al, 2021 (United States) (13)	RCT	COPD	physician-provided diagnosis of COPD	Hospital	Peer support + self-management support from respiratory care practitioner Peer support: via 1:1 and 8 group conversations + self-management support as control	Self-management support from respiratory care practitioner, written educational guide + 1-hour session and phone support	Attended PR program
Hajizadeh et al, 2021 (United States) (14)	RCT	COPD	confirmation of COPD by a pulmonary function test (PFT) or a pulmonologist's clinical diagnosis	Hospital	PR delivered by respiratory therapist via telehealth in either patient's home or community centre	Usual care	Attended start of PR programme Attended at least one session
Zwar et al, 2012 (Australia) (15)	Cluster RCT	COPD	diagnosis of COPD; no need for spirometry confirmation	Primary care	Partnership model of care Home visits: COPD trained nurse, working with GP and other HCPS; implement evidence based individualized care plan agreed with patients	Usual care COPD guidelines provided to GPs practices.	Attended PR program
Zwar et al, 2016 (Australia) (16)	Cluster RCT	COPD	Post-bronchodilator spirometry confirmed	Primary care	Evidence-based disease management plan implemented by nurse/GP teams including: practice staff education; computerized electronic care planning templates PLUS same case-finding/diagnosis training as control	8 hrs case finding/ spirometry training/ interpretation for practice nurses with computer-based toolkit + copy of COPD- national guidelines.	Attendance at PR
Norweg et al, 2025 (United States) (17)	Feasability RCT	COPD	a diagnosis of COPD based on standard spirometry, or a diagnosis of emphysema or chronic bronchitis based on chest computed tomography	1 main outpatient PR program based at a university medical center, and pulmonary medicine and primary care clinics	Breathing intervention involving slow breathing exercises, biofeedback, motivational interviewing and home breathing exercise program	Usual care	Attended PR assessment
Harris et al, 2009 (Australia) (18)	CCT	COPD	moderate to severe COPD (GOLD criteria)	Secondary care	Patient held manual summarizing evidence about COPD and suggested questions to ask doctor	Usual care; Simple, single sheet pamphlet containing information about COPD	Enrolment in PR
Levack et al, 2023 (New Zealand) (19)	Feasability study	AECOPD	clinical diagnosis of COPD admitted to hospital with a diagnosis of AECOPD	Hospital	Facilitator discussed exploration of values, relationships, hopes/fears, and individualised assessment and goal-setting with the patient	Usual care and a pamphlet about management of common problems in COPD	Attended at least one PR session

Pulmonary rehabilitation (PR) is a cornerstone of Chronic Obstructive Pulmonary Disease (COPD) management. Despite strong guideline recommendations, referral and uptake of PR remain poor. This systematic review updates a previous review (PMID: 27547859) (Watson et al., 2023) This review aimed to identify which interventions improve PR referral and uptake in patients with COPD.

This PRISMA-compliant review was prospectively registered on PROSPERO (CRD420261309204). CENTRAL, CINAHL, Embase and MEDLINE were searched for studies published between 31 August 2021 and 1 February 2026. Studies assessing interventions targeting PR referral or uptake in patients with COPD, compared to a control group or usual care, were included. The primary outcomes were PR referral and uptake. Risk of bias was assessed using the Cochrane Risk of Bias 2 for randomised studies and ROBINS-I for non-randomised studies. Due to substantial heterogeneity, the results were synthesised narratively.

The updated search yielded six new studies, while the prior review had six studies suitable for inclusion. In total, twelve studies involving 3663 participants were included, comprising seven randomised controlled trials, two cohort studies, two controlled clinical trials, and one randomised controlled feasibility trial. Six studies Five studies evaluated referral and ten evaluated uptake, with three assessing both. Two of five interventions significantly improved referral rates: an electronic health record-embedded clinical decision support pathway (8.45% vs 1.57%, p<0.001), and a patient-held COPD scorecard (7.79% vs 4.61%, p=0.03). Three of ten interventions significantly improved uptake: tele-pulmonary rehabilitation (91.9% vs 70.3%, p=0.0046), a GP–nurse partnership model (31.1% vs 9.6%, p=0.002), and a patient-held COPD manual in a socioeconomically disadvantaged subgroup (30.0% vs 7.0%, p=0.05). A COPD discharge bundle delivered by a healthcare professional involved in PR improved both referral (60.0% vs 12.3%, p<0.001) and uptake (40.0% vs 32.0%, p<0.01). Most studies had some concerns or a high risk of bias.

System-level interventions and coordinated patient–clinician approaches appear most effective for improving PR referral and uptake. Passive educational and motivational approaches did not reliably improve outcomes. However, confidence in these findings is limited by heterogeneous outcome definitions and bias. Future research should prioritise powered RCTs with well-described population and intervention characteristics.

P234 EXPERIENCE BASED CO-DESIGN TO ADAPT PULMONARY REHABILITATION FOR SOUTH ASIAN INDIVIDUALS

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Introduction Pulmonary rehabilitation (PR) is a highly evidenced intervention for individuals living with chronic respiratory disease. However, National Respiratory Audit Programme data shows that individuals with a South Asian ethnicity are under-represented in attending PR.[1]

Aim To co-design adaptations of PR to improve accessibility for South Asian individuals in collaboration with people from these communities living with chronic respiratory disease and their family members/carers, healthcare professionals and charity representatives.

Methods Seven experience-based co-design workshops to were held to develop adaptations of PR over seven months. Initially, separate 'patient and family' and 'professional' events were held to develop each groups priorities for adapting PR. At a joint event, three shared priorities for adapting PR were agreed as whole group, before working in three smaller mixed groups to develop practical ways each priority could be achieved. A final celebration event was held to celebrate all that had been achieved.

Results All co-contributors attended at least one co-design workshop. Patient and family co-contributors consisted of seven South Asian individuals living with chronic respiratory disease and three family members/carers (median (IQR) age 57 (15); female n=5). Professional co-contributors consisted of five charity representatives and five healthcare professionals (physiotherapists n=3, nurse n=1, doctor n=1) (median (IQR) age 49 (15); female n=9). The shared priorities to adapt PR were 1) Language and communication (grab and go packs with resources for individuals where English is a second language, an infographic to support the effective use of interpreters and a communication preferences form to clarify a service users' preferred forms of communication); 2) Programme adaptations (adding a holistic activity e.g., yoga/dance and culturally adapting education sessions); 3) Supporting people and community (PR patient experts, community outreach events, representative testimonial videos and drop-in sessions).

Conclusions Experience-based co-design successfully supported the collaboration of individuals under-represented in PR to identify shared priorities and co-design adaptations of PR with key stakeholders. Future work to assess the feasibility of co-designed adapted PR is needed.

REFERENCE

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P235 CO-DESIGN OF A VERY BRIEF BEHAVIOURAL CHANGE APPROACH TO ASSIST REFERRERS DISCUSS PULMONARY REHABILITATION WITH COPD PATIENTS

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Background Pulmonary rehabilitation (PR) is an effective intervention but referral and uptake rates are low. Very Brief Advice (VBA), a widely used framework ('Ask, Advise, Act') for healthcare professionals (HCPs) to discuss smoking cessation, increases smoking cessation service referral and uptake rates.

Accordingly, adapting VBA for PR (VBA-PR) may increase referral and uptake rates.

Aim To co-design and pilot test VBA-PR to support referrers discuss PR with COPD patients.

Method Purposively sampled COPD patients (n=17), their carers/family (n=2) and HCPs (n=22) participated in semi-structured interviews via videoconference/ telephone/ in-person. Data were analysed using reflexive thematic analysis and the Theoretical Domains Framework (TDF).

Findings were shared at videoconference co-design workshops and focus groups/interviews to finalise the intervention. The process was analysed using conventional content analysis, an iterative approach used to interpret qualitative data and develop new concepts, underpinned by the TDF framework developed during participant interviews.

HCPs (n=15) pilot-tested the intervention via videoconference focus groups. Data from this preliminary pilot and acceptability testing were analysed using two behavioural change frameworks: the APEASE criteria and the Theoretical Framework of Acceptability.

Results Participant interviews identified barriers and facilitators to PR and were used to inform the TDF underpinning the proposed intervention. This process identified key intervention targets, including knowledge, skills, environmental context and resources, social influences, intentions, and beliefs about consequences.

These findings directly informed the co-design of the Very Brief Advice for Pulmonary Rehabilitation (VBA-PR) intervention, including the 'Assess, Advise, Act' framework (figure 1), alongside a 30-minute e-learning programme incorporating PR education content and film clips.

Pilot testing with HCPs confirmed the intervention's acceptability and identified areas for improvement in useability (e.g. introductory course navigation information) and educational content (e.g. refinement of knowledge-based videos, representation of diversity). Minor modifications have since been made in response to user feedback.

Conclusion We have co-designed an acceptable intervention that aims to increase PR referral and uptake rates. Following intervention modification, feasibility and acceptability will be tested.

Trial registry ISRCTN12555890.



Abstract P235 Figure 1

P236 'DESIGNING REHABILITATION FOR INDIVIDUALS WITH LONG COVID – THE 4PS OF ACCEPTABILITY'

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Introduction and Objectives There is ongoing debate surrounding the safety and effectiveness of exercise in Long Covid. This qualitative study forms part of a mixed-methods project seeking to explore the effectiveness and acceptability of exercise-based rehabilitation for individuals with Long Covid who were not hospitalised. The aim is to understand factors that influence patients' likelihood of engaging with face-to-face exercise-based rehabilitation.

Methods Semi-structured interviews were conducted with individuals living with Long Covid who either declined to take part in an exercise-based rehabilitation trial, or started the intervention but dropped out. The Theoretical Framework of Acceptability (TFA)[1] was used to frame the topic guide.

Interviews were conducted virtually via Microsoft Teams, recorded and transcribed verbatim. Initial themes derived from the interviews, were then used to inform topic guides for focus groups with individuals who had completed rehabilitation.

The focus groups, facilitated by the lead researcher (KK) and assistant moderator (GD) were conducted remotely via Microsoft Teams. The groups were recorded with consent and transcribed electronically verbatim in real time.

Reflexive thematic analysis[2] was conducted, supported by NVivo (V1.6.1) software. A patient advisory group were consulted on the initial analysis and themes to ensure the data stayed close to lived experience.

Interview and focus group findings were integrated. Transcripts were first deductively coded using the TFA, followed by inductive empirical coding. Themes were then refined through

Abstract P236 Table 1

Key Theme	Synopsis
Practicality	Time, place and cost matter. Other commitments such as work or caring responsibilities must be recognised and personalised options offered.
Personalised	Symptoms vary and exercise and education must be tailored to match this. This includes ensuring the right time within the patient experience and facilitating flexibility in delivery format.
Perception	In the context of fatigue, exercise feels counterintuitive. Those who completed the programme were keen to challenge the perception of those who were apprehensive.
Partnerships	Partnerships with professionals and peers are valued supportive networks that must be based on trust. Successful partnerships help validate symptoms but dynamics may help or hinder.

an abductive approach to generate candidate themes relevant to both patient and clinical audiences.

Results 14 semi-structured interviews were conducted with 5 rehabilitation drop-outs and 9 decliners. 2 focus groups of 5 rehabilitation completers were conducted. Themes generated from integrated analysis are outlined in table 1:

Conclusions This work highlights factors to consider when designing services to ensure they are acceptable for individuals with Long Covid. The 4Ps acceptability framework presented here can be used as a guide for service providers to enhance rehabilitation uptake.

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P237 A BEHAVIOURAL INTERVENTION FOR PATIENTS WITH LONG-COVID

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10.1136/thorax-2026-BTSabstracts.397

Background Many patients with long-COVID present with breathing patterns disorder, characterised by an inefficient control of breathing. Patients with long-COVID also experience symptoms such as dizziness and fatigue, which have been linked to dysautonomia (dysfunction of the autonomic nervous system). It is possible that dysautonomia is linked with inefficient breathing mechanics. Therefore, physiotherapy interventions which can improve breathing control may be able to improve symptoms associated with long-COVID.

Methods The aim of this study was to understand the potential of a new physiotherapy intervention, Cognitive Muscular Therapy™ (CMT), for the management of long-COVID. CMT was originally developed for chronic musculoskeletal pain. In this study, it was adapted to create an intervention for breathing re-education, which incorporated postural retraining, coaching to increase awareness of muscle tension triggers and psychological strategies to support the adoption of new breathing habits. The final intervention comprised five components: Understanding long-COVID, General relaxation, Postural deconstruction, Contextual triggers and Functional integration and was delivered over 7-8 individual physiotherapy sessions.

Participants were recruited from secondary care, via GPs and social media. To be included participants required a clinical diagnosis of long-COVID and to scored 3 or more on the breathless subscale of the Modified COVID-19 Yorkshire Rehabilitation Scale (C19-YRSm) scale. Outcomes were collected at baseline and three months after completion of the intervention. Change data were evaluated using paired t-tests.

Results A total of 17 patients were recruited and 15 provided 3-month follow-up data. Mean (SD) ages and BMI were 49 (10) years and 26.1 (7.3) Kg/m^2. All patients completed at least 7 intervention sessions. Across the participants, the mean (SD) self-evaluation of breathing score reduced from 37 (9) to 25 (13) a change of 12 points ($p<0.01$). The mean (SD) Dyspnoea-12 score reduced from 17 (7) to 10 (9), a reduction of 42% ($p<0.01$) and the mean C19-YRSm reduced by 13 points ($p<0.05$). There was no change in the Composite Autonomic Symptom Score.

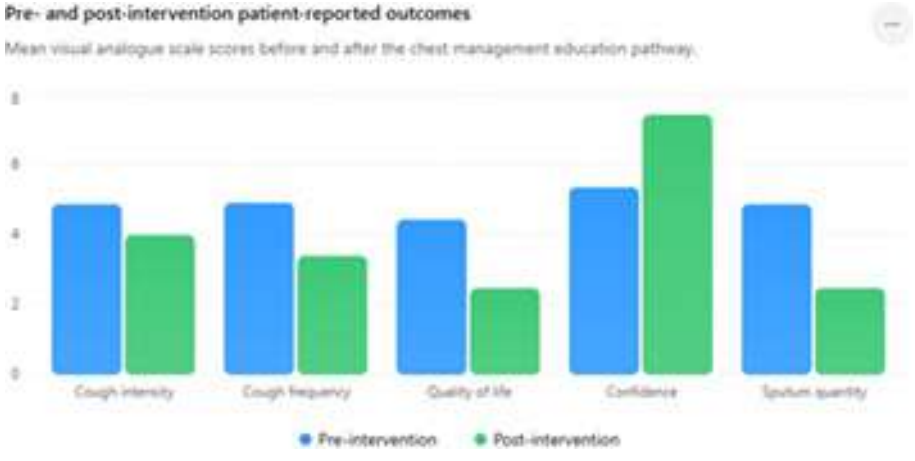
Conclusion Three months after completion of the CMT intervention, there were modest improvement in self-reported breathing. While there were no improvements in self-reported autonomic symptoms, there was an improvement in symptoms associated with long-COVID.

P238 THE BENEFITS OF A NOVEL GROUP-BASED CHEST MANAGEMENT EDUCATION PATHWAY FOR CHRONIC RESPIRATORY DISEASE: IMPROVING PATIENT OUTCOMES AND REDUCING CLINICIAN TIME

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Introduction Workforce pressures within respiratory physiotherapy services contribute to prolonged outpatient waiting



Abstract P238 Figure 1

times. A group-based chest management education pathway was introduced to improve service efficiency and optimise patient self-management.

Methods A prospective service improvement project was conducted in patients referred to a single-centre respiratory physiotherapy outpatient service between November 2025 and February 2026. Patients attended a 15 minute telephone assessment, a one hour group education session (5-6 patients/session), and a 30 minute follow up review. Education included airway clearance techniques, chest infection management, exercise benefits, and breathlessness management. Descriptive statistics summarised demographic data, with continuous variables reported as median and interquartile range (IQR). Pre and post-intervention outcomes included cough intensity, cough frequency, sputum quantity, quality of life (QoL), and confidence in cough management using visual analogue scale measures. Wilcoxon matched pairs signed-rank testing was performed due to small sample size and non-parametric paired data.

Results Sixty patients were enrolled (24 males, 36 females; median age 67 years [IQR 60–78]). Twenty-five completed follow up outcome measures. Significant improvements were observed in cough frequency ($W=40$, $p=0.008$), QoL ($W=30.5$, $p=0.009$), confidence in cough management ($W=26.5$, $p=0.003$), and self-reported sputum quantity ($W=4.5$, $p<0.001$). Reduction in cough intensity was not statistically significant ($W=46.5$, $p=0.153$). Total clinician workload, including administrative activities, cancellations and non-attendance, was 66 hours across 60 patients, equating to 66 minutes per patient compared with 105 minutes in the previous pathway, representing a 37% reduction in clinician workload.

Conclusion A structured group chest management education pathway is a feasible and time-efficient model for respiratory physiotherapy services. The intervention improved several patient-reported respiratory outcomes while substantially reducing clinician workload under real world operational conditions.

P239 MUCUS PLUGGING, EOSINOPHILIA, CHRONIC LUNG DISEASE AND MUCOLYTIC THERAPY: IDENTIFYING A PHENOTYPE

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10.1136/thorax-2026-BTSabstracts.399

Background Across chronic lung disease (CLD) apart from asthma, the relationship between eosinophilic inflammation and mucus plugging is poorly understood. In addition, mucolytic treatment response in this cohort needs to be explored further.

Aims To assess whether blood eosinophilia ($\geq 0.3 \times 10^9$/L) is associated with CT defined mucus plugging in CLD and if this phenotype has a better response to 7% hypertonic saline (HTS).

Methods This retrospective observational study included patients commenced on HTS between March 2023 and December 2025 recorded in a logbook. The highest recorded blood eosinophil count was extracted with eosinophilia defined as $\geq 0.3 \times 10^9$/L. Available chest CT scans were reviewed for mucus plugging. Symptomatic response to HTS was determined through clinic letters.

Results 55 patients were included, comprising Bronchiectasis, COPD, Asthma and overlap diseases. CT imaging was available for 52 patients.

Mucus plugging was identified in 35 patients, of which 74% (26/35) had eosinophil count $\geq 0.3 \times 10^9$/L and 26% (9/35) had counts $< 0.3 \times 10^9$/L ; OR 2.57, 95% CI 0.78–8.50; Fisher's exact $p = 0.21$

HTS treatment provided symptomatic benefit in 26 patients of which 77% (20/26) had eosinophil count $\geq 0.3 \times 10^9$/L and 23% (6/26) had counts $< 0.3 \times 10^9$/L; OR 1.67, 95% CI 0.32–8.78; Fisher's exact $p = 0.72$

Conclusion Data shows a trend towards identifying a specific phenotype in chronic lung disease who have eosinophilia and mucus plugging on CT imaging who may derive symptomatic benefit from HTS.

P240 LONG-TERM RECOVERY FOLLOWING PHYSICAL REHABILITATION FOR POST-COVID SYNDROME: A QUALITATIVE EXPLORATION OF PERSISTENT SYMPTOMS AND IMPACT OF EXERCISE

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10.1136/thorax-2026-BTSabstracts.400

Background While many studies have assessed the short-term effects of physical rehabilitation in post-COVID syndrome, limited qualitative evidence exists on its long-term impact. Post-COVID syndrome is characterised by persistent, multisystem symptoms that can significantly impair daily functioning, social participation, and quality of life.[1] Understanding how individuals experience recovery over time, particularly beyond the rehabilitation period, is essential to inform long-term management strategies.

Aim To explore experiences relating to ongoing symptoms, exercise impact, perspective, and social impact among participants with post-COVID syndrome following hospitalisation, who either completed physical rehabilitation (Rehab) or usual care (UC).

Methods Semi-structured online interviews were conducted with a convenience sample of post-COVID survivors who had either previously participated in a community-based physical rehabilitation programme or UC for post-COVID syndrome as part of the PHOSP-R study.[2] Thematic analysis was used to identify key themes related to ongoing symptoms, exercise impact, perspective, and social impact of post-COVID syndrome.

Results Fifteen participants were interviewed (Rehab group: n=8, 64-73 years; UC group: n=7, 43-86 years), 21-27 months following completion of rehabilitation or UC. Three overarching themes were identified. (1) Enduring symptoms and daily impact leads to dissatisfaction with healthcare: Participants in both groups reported ongoing breathlessness, fatigue, altered taste/smell, and cognitive difficulties. (2) Exercise improves quality of life and continuous recovery despite symptom

limitation: Participants in both groups reported engaging in exercises, including resistance training, aerobics, swimming, and walking. (3) Post COVID syndrome is misunderstood and reshapes social life: Participants in both groups reported reduced social participation, loss of identity, and feeling misunderstood.

Conclusion In post-COVID syndrome, symptoms persist despite participation in rehabilitation. Although participants in both groups engaged in physical activity, long-term follow-up alongside integrated healthcare provision and broader social support are essential to optimise recovery and quality of life.

Keywords Post-COVID Syndrome, Physical Rehabilitation, Quality of Life

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Abstract P241 Table 1 Summary of reliability, validity and diagnostic performance of the breathing pattern assessment tool (BPAT)

Measurement property	Result
Inter-rater reliability	ICC 0.905 (95% CI 0.867–0.933)
Test–retest reliability	ICC 0.869 (95% CI 0.799–0.916)
Measurement error	SEM 0.74–0.88; MDC95 2.05–2.44
Known-groups validity	χ^2=39.89, p<0.001; ε^2=0.25
Diagnostic discrimination	AUC 0.783 (95% CI 0.710–0.856)
Best-performing BPAT domain	Abdominal / Upper chest AUC 0.808
Combined BPAT + NQ model	AUC 0.832

χ^2: Kruskal–Wallis statistic; ε^2: epsilon-squared effect size (0.25 indicates a large between-group effect).
ICC = Intraclass Correlation Coefficient; **SEM** = Standard Error of Measurement; **MDC95** = Minimal Detectable
Change at the 95% confidence level; **AUC** = Area Under the Receiver Operating Characteristic Curve; **CI** = Confidence Interval.

P241 CAN OBSERVABLE BREATHING ABNORMALITIES IDENTIFY BREATHING PATTERN DISORDER? EVALUATION OF THE BREATHING PATTERN ASSESSMENT TOOL

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10.1136/thorax-2026-BTSabstracts.401

Background The Breathing Pattern Assessment Tool (BPAT) is increasingly used by physiotherapists to assess observable features of breathing pattern disorder (BrPD). However, evidence supporting its measurement properties remains limited. This study evaluated the reliability, validity and diagnostic performance of the BPAT in adults undergoing assessment for BrPD.

Methods A prospective observational study was conducted within a specialist respiratory physiotherapy service. Adults referred with suspected BrPD were recruited alongside asthma and healthy control participants. Inter-rater and test–retest reliability were assessed using intraclass correlation coefficients (ICC) with 95% confidence intervals (95%CI), standard error of measurement (SEM) and minimal detectable change (MDC95). Validity was evaluated through predefined hypothesis testing and known-groups validity analyses. Diagnostic performance was assessed using receiver operating characteristic (ROC) analysis. Exploratory analyses examined individual BPAT domains and a combined assessment model incorporating BPAT and Nijmegen Questionnaire (NQ) scores.

Results A total of 153 participants were included (36 BrPD, 60 asthma + BrPD, 28 asthma, 29 healthy controls). The BPAT demonstrated excellent inter-rater reliability (ICC=0.905, 95%CI 0.867–0.933) and good test–retest reliability (ICC=0.869, 95%CI 0.799–0.916). SEM ranged from 0.74–0.88 points and MDC95 from 2.05–2.44 points.

Validity was partially supported, with significant correlations observed with the Nijmegen Questionnaire (ρ=0.287), Self-Evaluation of Breathing Questionnaire (ρ=0.332), Dyspnoea-12 (ρ=0.328), GAD-7 (ρ=0.218) and EQ-5D-VAS (ρ=-0.251) (all p<0.05). Known-groups validity was demonstrated by significant differences in BPAT scores between the diagnostic groups (χ^2=39.89, p<0.001; ε^2=0.25). Participants with physiotherapist-diagnosed BrPD demonstrated higher BPAT scores than those without BrPD (median 5 [IQR 3–6.5] vs 2 [IQR 1–4], p<0.001).

The BPAT demonstrated acceptable discrimination for BrPD (AUC=0.783, 95%CI 0.710–0.856). Exploratory analyses identified Abdominal/Upper chest movement (AUC=0.808), air hunger (AUC=0.672) and rhythm (AUC=0.632) as the most discriminatory BPAT domains. A logistic regression model combining continuous BPAT and NQ scores improved discrimination (AUC=0.832), suggesting symptom burden and observable breathing abnormalities provide complementary diagnostic information.

Conclusions The BPAT demonstrated good-to-excellent reliability, evidence of validity and acceptable discrimination for BrPD. These findings support its use as a standardised observational assessment tool and suggest that combining observational and symptom-based assessments may enhance clinical evaluation of BrPD.

P242 BEYOND THE SCORE: PHYSIOTHERAPISTS' EXPERIENCES OF USING THE BREATHING PATTERN ASSESSMENT TOOL IN CLINICAL PRACTICE

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10.1136/thorax-2026-BTSabstracts.402

Background The Breathing Pattern Assessment Tool (BPAT) is an observational assessment tool used to evaluate breathing pattern disorder (BrPD). While quantitative studies have demonstrated promising measurement properties, less is known about how clinicians use and interpret the BPAT in practice. This study explored physiotherapists' experiences of BPAT administration, focusing on feasibility, acceptability and perceived clinical utility.

Methods A qualitative study using semi-structured interviews was undertaken with physiotherapists experienced in the assessment and management of BrPD. Participants were recruited purposively from specialist respiratory services and included clinicians with varying levels of BPAT experience. Interviews



Abstract P242 Figure 1

were conducted online, audio-recorded and transcribed verbatim. Data were analysed using reflexive thematic analysis.

Results Nine physiotherapists participated. Six themes were developed (figure 1). Clinicians described the BPAT as a useful and practical framework that provided structure to breathing assessment whilst remaining quick and feasible to use in routine practice. However, participants consistently emphasised that BPAT scores were interpreted within broader clinical reasoning processes and were not considered diagnostic on their own. Assessment of breathing pattern was viewed as highly contextual, with clinicians adapting BPAT administration according to clinical setting, patient presentation and functional triggers of breathlessness. Participants valued the BPAT for capturing observable biomechanical aspects of breathing but identified limitations relating to psychological influences, symptom perception, upper airway features and exertional breathing responses, which were not directly captured. Some BPAT domains were perceived as inherently subjective, particularly respiratory rhythm and flow, leading clinicians to develop individual scoring strategies. Despite these limitations, the BPAT was considered highly valuable for treatment planning, monitoring change over time, and supporting communication within multidisciplinary teams.

Conclusions Physiotherapists perceive the BPAT as a clinically useful and feasible assessment framework for breathing pattern evaluation. Beyond assessment, the BPAT was valued as a clinical framework that supports diagnostic reasoning, treatment planning, and communication about breathing pattern disorders. Findings suggest that the strength of the BPAT lies in its ability to combine a structured assessment framework with the flexibility required to accommodate the contextual and variable nature of breathing pattern assessment.

P243 THE PNEUMORATER CHEST-WORN SENSOR CAN CONTINUALLY MEASURE RESPIRATORY RATE AND SIGNS OF AIR HUNGER IN ADULTS WITH AND WITHOUT BREATHING PATTERN DISORDERS

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10.1136/thorax-2026-BTSabstracts.403

Introduction and Objectives Change in respiratory rate (RR) can indicate clinical deterioration or breathing pattern disorders (BrPD). However, RR is inconsistently measured in clinical practice. Prolonged RR measurement may have meaningful clinical benefits over existing methods. We aimed to compare RR averages over ten minutes using the PneumoRator device with 1-minute manual spot-checks conducted at rest. Furthermore, we aimed to assess whether the PneumoRator could accurately detect signs of air hunger (SAH).

Methods Adults from the University of Southampton were recruited. Participants sat in a chair for 10 minutes with the PneumoRator device attached to their chest. A Breathing Pattern Assessment Tool (BPAT) was completed at a randomised minute. Large-volume breaths were identified using an area-under-the-curve (AUC) threshold (>2x the average breath AUC) in MATLAB. Statistical analyses included the Wilcoxon Test comparing PneumoRator and manual RR measures, Spearman's Rho for correlation, and a Mann-Whitney U test for exploratory AUC comparison.

Results Twenty-nine participants (16 females, mean age and BMI of 21 and 24.5 respectively, 17 without BrPD and 12 with BrPD according to BPAT) provided full PneumoRator recordings for analysis. There was no significant difference in RR between PneumoRator data and observer measurements ($p=0.89$, Median difference= -0.67, 95% CI= -1.89 to 1.78). Observer and PneumoRator SAH counts reached a 66% level of agreement (19/29) with no significant difference in SAH counts ($Z = -1.39$, $p = 0.17$). There was a statistically significant moderative, positive correlation between methods ($\rho = 0.50$, $p = 0.01$). Participants with clinically observed SAH demonstrated significantly higher PneumoRator derived AUC values compared to those without ($U = 54.5$, $Z = -2.11$, $p = 0.04$).

Conclusions The PneumoRator device can continuously monitor RR and detect SAH in individuals with and without BrPD to the same accuracy as clinical standard measurement. The PneumoRator device could be used as an assessment and monitoring tool in the treatment of those with BrPD or to monitor acute deterioration in patients on ward-based settings. Further research is warranted.

P244 AN ONLINE BREATHING AND WELLBEING PROGRAMME (ENO BREATHE) FOR PEOPLE WITH LONG COVID BREATHLESSNESS: A MIXED METHODS ANALYSIS OF 3-MONTH POST-INTERVENTION FOLLOW-UP DATA

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10.1136/thorax-2026-BTSabstracts.404

Introduction Long COVID (LC) is a complex, prevalent and often debilitating condition. Clinical guidelines recommend

holistic management approaches, though a limited number of evidence-based interventions exist. ENO-Breathe is a six-week, online, breathing and wellbeing programme, integrated into NHS pathways, which uses singing techniques to improve breath control, anxiety and wellbeing in people with LC breathlessness. A previous RCT and cohort study demonstrated immediate post-intervention improvements in health-related quality of life, anxiety, and breathlessness. Longer-term impacts have not been investigated.

Methods We undertook a mixed-methods study analysing responses to a survey conducted 3-months after completion of the ENO-Breathe programme, between November 2021 and August 2022. Participants reported symptom and health changes since programme completion using multiple-choice responses; and rated the impact of the programme on aspects of health and wellbeing and on LC recovery using Likert scales (0-100 or 0-10). Reflexive thematic analysis (RTA) was conducted on free-text responses to the question 'Three months after the programme, how would you describe the impact ENO-Breathe had on you?', focusing on programme impacts and their mechanisms.

Results The survey was sent to 1179 participants and received738 responses.Following data cleaning,483 responses were included in the final analytical sample, with 474 providing a response for RTA. Participants reported improvements in breathlessness (71%), general health and wellbeing (66%), and anxiety (61%). Likert responses revealed a positiveprogrammeimpact on mental health and wellbeing (84 (70-100)) and physical health (71 (53-88)), but that participants were stillimpactedby symptoms, median 5/10 (IQR 3-7). RTA identified 4 key themes:(i)psychosocial benefits fromcommunity support,(ii)practical and effective learnt techniquesto improve self-management of symptoms, (iii)increased sense of control over breathing, anxiety, and overall health,and (iv)improvement in mental health and well-being facilitatingreablement.

Conclusions ENO-Breathe participants reported improvements in mental health and wellbeing, breathing, and anxiety 3-months post-intervention, attributing these to the techniques learnt, community support, and knowledge provided by the programme. Our study indicates sustained benefits following ENO-Breathe participation, addressing an important research gap. Future research in other settings, other causes of breathlessness, and incorporating objective measures would be valuable.

Asthma in transition

P245 DOES THE CARBON FOOTPRINT OF INHALERS IMPACT PATIENT'S INHALER CHOICE. EVIDENCE FROM THE REDUCE CARBON FOR PATIENTS RCT

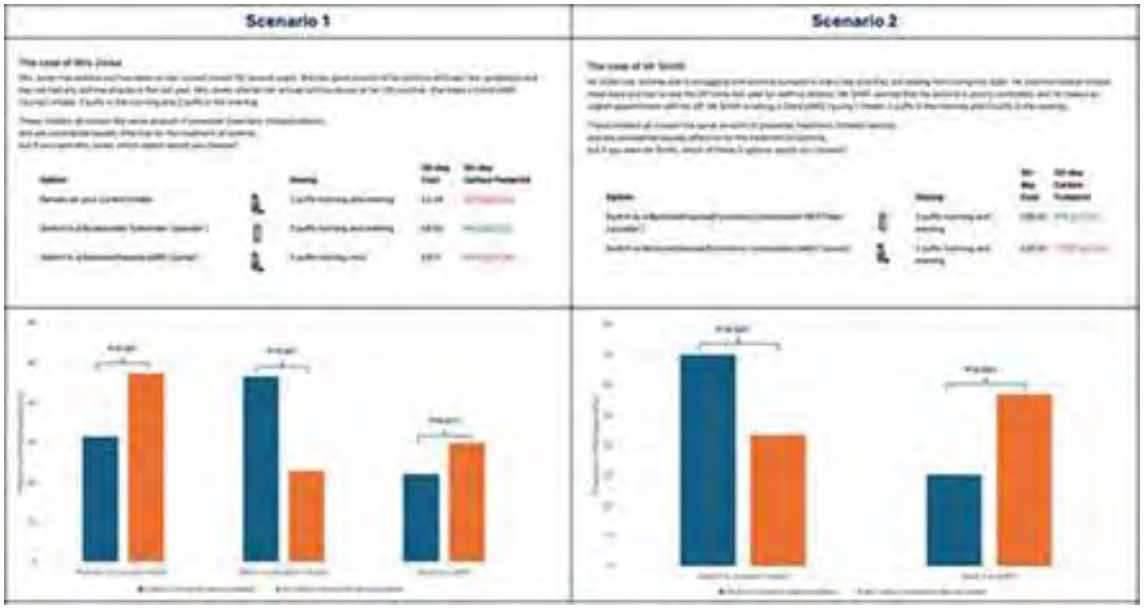
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10.1136/thorax-2026-BTSabstracts.405

Concern over the carbon footprint of some pressurised metered dose inhalers (pMDIs), relating to the global warming potential of the propellant gasses used in some of these devices, has led to guideline recommendations for 'low-carbon inhalers' where possible. However, there is limited evidence as to whether the provision of carbon data will impact a patient's inhaler choice.

We undertook a single blinded randomised-controlled trial using an online questionnaire platform which included simple case scenarios to assess participant's views on inhaler choice (ISRCTN16609010). Participants were randomised 1:1 as to whether carbon footprint data for inhalers was provided or not and further information was gathered to understand their views on how carbon data should be provided. Participants were aged ≥18years with a diagnosed respiratory condition requiring the use of at least one inhaled therapy. Differences between the study groups were analysed using chi-square testing.

9109 participants completed the study, 95% had airways disease and 83% were using an inhaler on a daily basis with the



Abstract P245 Figure 1 Case scenarios and participant responses, comparing participant responses between those who received the carbon footprint data and those who did not. Differences between the study groups were analysed using chi-square testing

majority prescribed pMDI devices. Whilst most did feel that carbon footprint data should be used when making decisions about inhaler choice, the majority were not aware of the differences in carbon footprint between inhaler devices.

When provided with carbon data participants were significantly more likely to select a 'low-carbon inhaler' prioritising this above inhaler cost or the complexity of the dosing regime. As shown in figure 1, the provision of carbon footprint data approximately doubles the likelihood of the participant choosing a low-carbon inhaler in the context of both stable and poorly controlled disease (*47% vs 23%; p=<0.001 in scenario 1 and 70% vs 43.3%; p<0.001 in scenario 2*).

84% of participants felt that carbon footprint should be considered when making decisions about which inhaler they are prescribed (where efficacy is equivalent) with most recommending this should be provided by a healthcare professional contextualised using a 'traffic-light' system.

This large study confirms that providing carbon-footprint information does impact patient decisions relating to inhaler choice and this offers an important opportunity to support guideline ambitions for the prescription of 'low-carbon inhalers'.

P246 ARE THE INHALER PRESCRIBING PREFERENCES OF HEALTHCARE PROFESSIONALS INFLUENCED BY THE CARBON FOOTPRINT - EVIDENCE FROM THE REDUCE CARBON RCT

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10.1136/thorax-2026-BTSabstracts.406

Concern over the carbon footprint of some pressurised metered dose inhalers (pMDIs), relating to the global warming potential of the propellant gasses used in some of these devices, has led to guideline recommendations for 'low-carbon inhalers' where possible. However, there is limited evidence as to whether the provision of carbon data in asthma guidelines would influence healthcare professionals (HCPs) inhaler prescribing preferences.

We undertook a single blinded randomised-controlled trial using an online questionnaire platform to assess inhaler prescribing preferences when stepping-up and stepping down treatment as well as in the context of well-controlled asthma. Participants were HCPs working within the NHS in a primary or secondary care setting. Demographics including job role, employment setting, respiratory experience and prescribing status were also collected. Participants were randomised 1:1 as to whether carbon footprint data for inhalers was provided or not. Differences between the study groups were analysed using chi-square testing. Logistic regression analysis was also used to evaluate the association between availability of carbon footprint information and the inhaler type selected (high vs. low carbon).

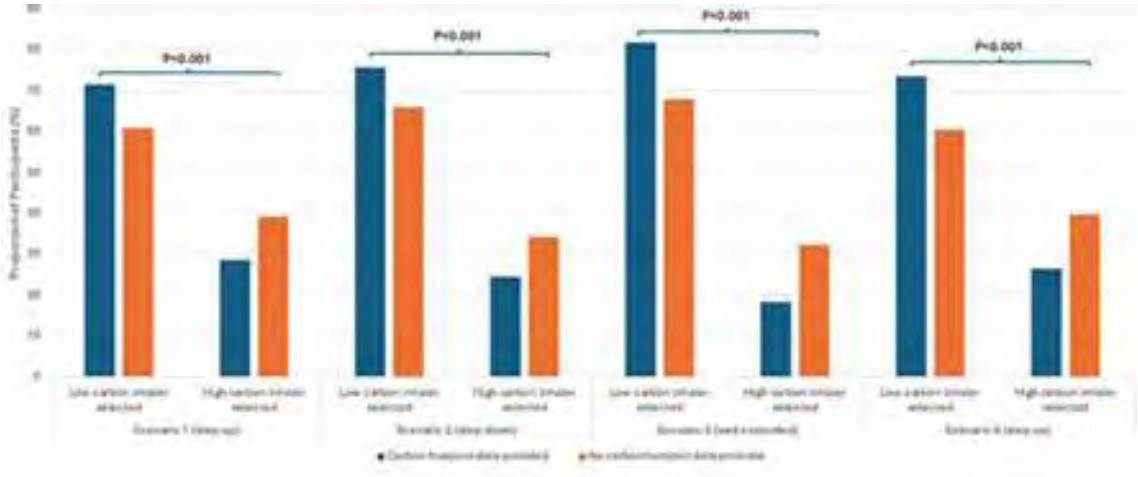
3782 participants completed the study. Participants viewed clinical evidence and patient ability as the most important factors when making decisions about inhaler choice, however, the majority ranked carbon footprint of the inhaler as more important than cost. When provided with carbon data participants were significantly more likely to select a 'low-carbon inhaler' irrespective of disease state (p<0.001) (see figure 1). When participants were provided with the carbon data, they were statistically more likely to recommend switching to a 'low-carbon inhaler' (29% vs 19%, p <0.001). Participants provided with carbon footprint data were 32% less likely to rank a high-carbon inhaler regime first (p <0.001) with the OR 1.59 greater for choosing a low-carbon inhaler when carbon data was available compared to when it was not (p <0.001).

This large study confirms that providing carbon-footprint information positively influences HCPs' decisions relating to inhaler choice. This demonstrates the importance of including carbon footprint data within asthma management guidelines to facilitate the prescription of 'low-carbon inhalers' where appropriate.

P247 SHIFTING PATTERNS OF ICS/FORMOTEROL UTILISATION FOLLOWING IMPLEMENTATION OF AIR/MART THERAPY: A NATIONAL PRESCRIBING ANALYSIS 2022–2025

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10.1136/thorax-2026-BTSabstracts.407



Abstract P246 Figure 1 Clinician preference for low carbon vs high carbon inhalers, comparing participant responses between those who received the carbon footprint data and those who did not



Abstract P247 Figure 1

Background Anti-inflammatory reliever (AIR) and Maintenance-and-Reliever Therapy (MART) strategies have transformed asthma management by combining maintenance and reliever therapy within a single ICS/formoterol inhaler. Understanding inhaler utilisation patterns may provide insight into treatment optimisation and identify patients at risk of under- or over-treatment.

Methods Pseudonymised ICS/formoterol data for England, 2022–2025, was extracted under the NHS England–PrescQIPP CIC data sharing agreement and analysed. Patients were categorised by annual inhaler use: 1–3, 4–5, 6–15 and ≥16 inhalers per year, representing AIR therapy for mild asthma, higher AIR use or underuse of regular or MART therapy, regular or MART therapy, and potential overuse respectively. Changes in prescribing distributions over time were assessed.

Results Total ICS/formoterol prescribing increased from 1.74 million inhalers in 2022 to 2.59 million in 2025. Annual inhaler utilisation changed significantly over this period (χ^2 $p<0.001$).

The proportion of patients receiving 1–3 inhalers annually increased from 25.8% to 37.0% (448,784 to 957,404 patients), consistent with increasing use of AIR therapy for mild asthma. Patients receiving 4–5 inhalers annually remained stable (14.5% to 14.7%) potentially reflecting higher AIR use or underuse of regular MART.

The 6–15 inhaler category remained the largest group (increasing from 958,277 to 1,146,434 patients), although its proportion declined from 55.1% to 44.3%, suggesting wider uptake of regular or MART.

Patients receiving ≥16 inhalers annually declined proportionally from 4.7% to 4.1%, but absolute numbers increased from 80,879 to 105,539 patients. In 2025 almost 24,000 patients in 2025 received ≥24 inhalers annually, including approximately 1,000 receiving 36–51 inhalers and more than 100 receiving ≥52 inhalers.

Conclusions ICS/formoterol utilisation shifted towards lower annual inhaler use between 2022 and 2025, suggesting increasing optimisation of AIR/MART prescribing. However, a substantial numbers continue to demonstrate excessive inhaler use despite guideline-based therapy. Limitations include the absence of linked indication and regimen data within ePACT and the possibility that lower annual use reflects treatment initiation during the study year. These findings highlight both the success of AIR/MART implementation at scale and the ongoing need to identify and support patients with persistently high reliever requirements.

P248 EXPLORING PRESCRIBING PATTERNS FOLLOWING SWITCHING BETWEEN FOSTAIR® AND LUFORBEC® IN A SCOTTISH NHS BOARD: A RETROSPECTIVE ANALYSIS OF PIS PRESCRIBING DATA

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10.1136/thorax-2026-BTSabstracts.408

Introduction Formulary-driven inhaler switches are widely used to support prescribing optimisation. In a regional NHS board, a large-scale switch from Fostair® to Luforbec® was undertaken. Although both contain beclometasone/formoterol, Luforbec® contains maleic acid, which potentially may contribute to cough and poor tolerability in some patients. Local clinicians observed patient-reported cough or symptom worsening requiring switchback. We evaluated the frequency, timing and predictors using prescribing data.

Methods A retrospective observational analysis of the outcomes of patients switched from Fostair® to Luforbec® was undertaken using patient-level prescribing data from PIS, Scotland's national database of community-dispensed prescriptions from February 2023 to February 2026.

Baseline characteristics were summarised using counts and percentages. Age was grouped (<40, 40–59, 60–79, ≥80), with sex and SIMD (Scottish Index of Multiple Deprivation) quintile recorded. Group differences were assessed using Pearson chi-squared tests. Time to switchback was analysed using Kaplan–Meier methods.

Results Overall, 8,291 patients switched from Fostair® to Luforbec. Of these, 1,661 (20.2%) switched back to Fostair® and 6,630 remained on Luforbec®. Switchback was more frequent in females than males (21.9% vs 16.8%, $p<0.001$) and increased across SIMD quintiles from 17.0% in SIMD1 to 23.0% in SIMD5 ($p<0.001$). Age also differed statistically ($p=0.022$).

Cox models assessed associations between age group, sex, SIMD quintile and GP practice with time to switchback. Male sex reduced switchback risk (adjusted hazard ratio [aHR] 0.73, 95%CI 0.65-0.81; $p<0.001$). Deprivation increased switchback risk (SIMD1 vs 5 aHR 1.55, 95%CI 1.31-1.84; $p<0.001$).

Kaplan–Meier analysis showed (figure 1), cumulative switchback of 5.7% at day 0, 16.8% at 1 year and 20.2% at 2 years.



Abstract P248 Figure 1

Excluding same-day switchbacks, cumulative switchback remained 11.8% at 1 year and 15.4% at 2 years.

Conclusion Following the Fostair® to Luforbec® switch, 20% of patients switched back to Fostair® within 2 years. Switchback was not confined to immediate reversals and remained substantial after excluding same-day switchbacks. Variation by sex, deprivation and GP practice suggests that formulary switches should be supported by patient communication, review pathways, and monitoring of persistence, switchback and patient experience. Further research should explore reasons for switchback, including the potential role of maleic acid.

P249 BUDESONIDE/GLYCOPYRRONIUM/FORMOTEROL FUMARATE DIHYDRATE EFFECTS ON LUNG FUNCTION AND ASTHMA EXACERBATIONS BY PERSISTENT AIRFLOW LIMITATION STATUS: POST HOC ANALYSES OF KALOS AND LOGOS STUDIES

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10.1136/thorax-2026-BTSabstracts.409

Background Persistent airflow limitation (PAL) in asthma is associated with poorer control, lung function (LF) decline and exacerbations. The KALOS and LOGOS studies in inadequately controlled asthma showed improved LF and exacerbation



Abstract P249 Figure 1 (A) Change from baseline in moring pre-dose trough FEV : (B) change from baseline in FEV, AUCa3, (C) severe exacerbation endpoints, and (D) percentage of responders in ACQ-7 by PAL status*

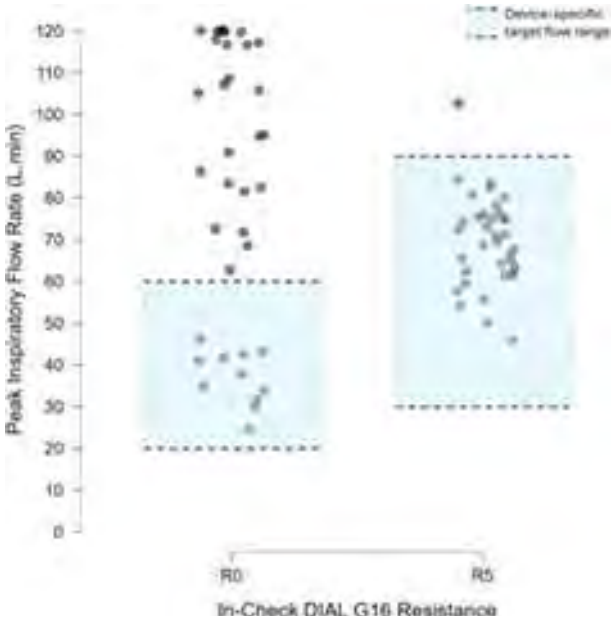
rates with budesonide/glycopyrronium/formoterol fumarate dihydrate via Aerosphere® MDI (BGF 28.8; 320/28.8/10 μg) vs combined budesonide/formoterol fumarate dihydrate ($BFF_{Combined}$) via Aerosphere® MDI (BFF_A) or Symbicort® (BFF_S).

Aims This pooled analysis evaluated BGF 28.8 or BGF 14.4 (320/14.4/10 μg) vs $BFF_{Combined}$ impact on LF and exacerbations by PAL in pooled KALOS and LOGOS studies.

Methods Participants (≥12 y; pooled N=4311) were randomised to BGF 28.8, BGF 14.4, BFF_A (320/10 μg) or BFF_S (320/9 μg) twice daily for 24–52 weeks. LF endpoints (change from baseline [BL] in trough FEV_1; FEV_1 $AUC_{0–3}$), severe exacerbation rate and percentage of responders in 7-Item Asthma Control Questionnaire (ACQ-7) were assessed by PAL status (post-albuterol FEV_1/FVC <70% at Visit 2 and 3).

Results Directionally consistent improvements in trough FEV_1 and FEV_1 $AUC_{0–3}$ were observed with BGF vs $BFF_{Combined}$ in patients with or without PAL (figure 1). BGF 28.8 reduced severe exacerbations and increased the percentage of responders in ACQ-7 vs $BFF_{Combined}$, with similar trends between PAL groups (figure 1). Similar trends were observed for BGF 14.4 vs $BFF_{Combined}$.

Conclusions BGF 28.8 showed benefits over $BFF_{Combined}$ with improved LF and reduced severe exacerbations irrespective of PAL status.



Abstract P250 Figure 1

P250 INHALER MISMATCH IN ATHLETES: INSPIRATORY FLOW AND TECHNIQUE FAVOUR DRY POWDER INHALERS OVER PRESSURISED METERED-DOSE INHALERS

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10.1136/thorax-2026-BTSabstracts.410

Introduction The efficacy of inhaled therapy is influenced by inhaler technique and appropriate inspiratory flow. Pressurised metered-dose inhalers (pMDIs) require coordination of actuation with a slow, steady inhalation, whereas dry powder inhalers (DPIs) require higher inspiratory flow rates to disperse the dose effectively. However, the most appropriate inhaler choice for athletes remains unclear. The study aimed to assess peak inspiratory flow rates (PIFR), inhaler technique, and asthma control in athletes with asthma and exercise-induced bronchoconstriction (EIB).

Method Athletes with physician-diagnosed or suspected asthma/EIB were recruited. Inspiratory flow was assessed using the In-Check DIAL G16 across three resistance conditions: participants' prescribed or familiar inhaler resistance (RX), guided low-resistance pMDI simulation (R0), and high-resistance DPI simulation (R5). Correct PIFR ('pass') was defined as 20-60 $L{\cdot}min^{-1}$ for R0 and 30-90 $L{\cdot}min^{-1}$ for R5. Mean inspiratory flow and pass/fail were compared with device-specific thresholds. Inhaler technique was assessed using a 7-point checklist during placebo pMDI or DPI use. Asthma control and lung function were assessed through ACQ, FeNO, and Spirometry.

Results Thirty-six athletes (20 male, 16 female; age 28±13 years) completed the study. Twenty-nine athletes were prescribed inhaler therapy (13 used ≥2 inhalers); 17 combination ICS/LABA, 20 SABA monotherapy, and six ICS. Uncontrolled ACQ in 10 (28%), FeNO >25 ppb in 13 (36%), and airflow obstruction in six (17%).

During unguided inhalation of familiar inhaler type (pMDI; n=24 vs. DPI; n=12), mean PIFR was 114±18 vs. 71±25 $L{\cdot}min^{-1}$, with pass rates of 4% and 92%, respectively ($\chi^2(1)=27.56$, $p<0.001$). Under guided conditions, mean PIFR was 81±34 $L{\cdot}min^{-1}$ at R0, and 70±11 $L{\cdot}min^{-1}$ at R5, with pass rates of 31% and 97%, respectively (figure 1).

No inhaler technique errors were observed in 4% of pMDI users vs. 75% of DPI users. Errors included coordination of actuation/inhalation (n=20), inadequate breath-hold (n=18), and failure to prepare/load dose (n=9).

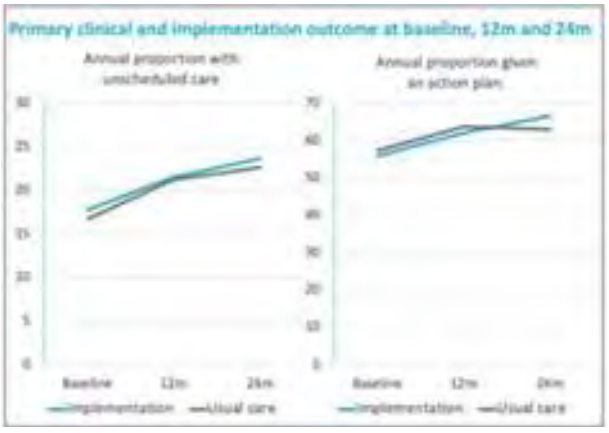
Conclusion Athletes demonstrate more effective inspiratory flow rates and fewer technique errors with DPIs compared with pMDIs during unguided and guided conditions. These findings highlight the importance of device selection and inhaler technique assessment in athletes with asthma/EIB. Furthermore, SABA monotherapy remained common despite guideline recommendations, highlighting need for prescribing review.

P251 EFFECTIVENESS OF A FACILITATED IMPLEMENTATION STRATEGY TO IMPROVE ROUTINE DELIVERY OF SUPPORTED ASTHMA SELF-MANAGEMENT IN UK PRIMARY CARE: A CLUSTER RANDOMISED IMPLEMENTATION TRIAL (IMP2ART)

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10.1136/thorax-2026-BTSabstracts.411

Introduction Since the first BTS asthma guidelines in 1990, supported self-management has been a recommended management strategy for asthma, but implementation remains poor.



Abstract P251 Figure 1

Aim To test the effectiveness of facilitated delivery of the IMP^{2}ART implementation strategy (IMP) in UK general practices compared to usual care (UC).

Methods We recruited general practices in England and Scotland during 2021-23 and randomly allocated practices to receive an implementation strategy facilitated by trained specialist nurses and targeting organisational priorities, clinicians' skills and patient needs, or to UC. We used routine data to assess between-group difference in proportion with unscheduled asthma care/year (clinical outcome) and proportion given an action plan (implementation outcome) in all patients clinically eligible (Age >=5-years, with 'active asthma') at baseline/12m/24m timepoints. Other outcomes assessed included symptom control, asthma care received, and prescribing (reliever medication, oral steroid courses).

Results We randomised 144 practices (73 IMP, 71 UC: Attrition 4 IMP; 3 UC) and included 28,488 (IMP) and 22,847 (UC) patients in the analysis. Practice/patient baseline characteristics were similar between groups and reflected known UK demographics. Overall fidelity to delivery of the strategy was good. In both groups, unscheduled care increased from a low baseline of 17.3% during the pandemic, but there was no statistically significant difference at 24m in proportion with at least one unscheduled care event/year (IMP: 23.6% vs UC 22.6%, OR 1.06 (95%CI: 0.93, 1.21) p=0.33). Action plan provision increased in line with changes in the Quality and Outcome Framework (in England), but there was no statistically significant difference in proportion given an action plan during the 2-years of the trial (IMP: 84.0% vs UC: 84.1%, OR 1.05 (95%CI: 0.76, 1.45) p=0.75). Similarly, there was no between-group differences in any of the secondary outcomes at 12 or 24-months.

Conclusion In these low-risk patients, we were unable to demonstrate any impact from the IMP^{2}ART strategy, in the context of substantial increases in unscheduled care and supported self-management delivery in both control and implementation practices over the trial period. Our experience highlights the pivotal role of national context and health systems policy.

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P252 AN ECONOMIC EVALUATION OF THE IMP^{2}ART WHOLE-SYSTEMS INTERVENTION TO SUPPORT ASTHMA SELF-MANAGEMENT IN PRIMARY CARE

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10.1136/thorax-2026-BTSabstracts.412

Background Despite substantial evidence and longstanding guideline recommendations, implementation of supported asthma self-management in primary care remains suboptimal. The IMP^{2}ART trial tested a whole-systems strategy to embed supported asthma self-management in routine primary care practice.

Aims To evaluate the costeffectiveness of the IMP^{2}ART strategy compared with usual care, acknowledging residual uncertainty arising from necessary assumptions despite comprehensive examination of implementation and control practices.

Methods The economic evaluation was conducted alongside the UK-wide cluster RCT including 144 general practices and 51,335 patients. The implementation strategy (facilitation, staff training, organisational changes, patient-oriented resources) was assessed against standard, routine care. Resource use was valued from the NHS perspective, while health outcomes were expressed in quality-adjusted life years (QALYs) over a 24-month follow-up, with both costs and QALYs discounted at an annual rate of 3.5%. Cost-effectiveness was evaluated by estimating incremental cost-effectiveness ratios (ICERs), with decision uncertainty examined through probabilistic sensitivity analyses and supplementary model-based approaches.

Results Mean costs were higher in the intervention group compared with usual care (£92.63 vs £80.39), resulting in an incremental cost of £12.24 per patient. Health outcomes were very similar between groups, with QALYs of 1.520 in the intervention arm and 1.523 under usual care (incremental difference -0.003). These findings indicate that the intervention was more costly and slightly less effective, with an ICER of -£4,134 per QALY, suggesting that usual care dominated. At conventional UK NICE willingness-to-pay thresholds, the probability that the intervention is cost-effective remained low. This was approximately 40% at £20,000 per QALY and around 50% at £30,000 per QALY. Results were robust across probabilistic sensitivity analyses and consistent with model-based findings.

Conclusions The whole-systems strategy for implementing supported asthma self-management was associated with slightly higher costs but no measurable improvement in health outcomes over 24 months, suggesting it is unlikely to be cost-effective in the short term. However, given the very small differences observed in both costs and QALYs, there remains considerable uncertainty around this conclusion. Future large-scale implementation may require more targeted or lower-cost approaches, or longer follow-up periods, to fully capture potential benefits and demonstrate value for money in routine primary care.

P253 REDUCING THE NUMBER OF HEALTHCARE INTERACTIONS TO ACHIEVE A DIAGNOSIS IN ASTHMA USING THE ASTEC SCORE VS BTS/SIGN/NICE GUIDELINES

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10.1136/thorax-2026-BTSabstracts.413

Introduction and Objective Asthma diagnosis often relies on symptoms and there is no definitive gold standard. The Astec (ASThma fEv1 composite) score integrates digital home lung, medication adherence and type 2 (T2) inflammatory markers as an alternative to current guideline pathways. A proposed diagnostic model incorporating 2 months of Astec score data followed by bronchial hyper-responsiveness (BHR) testing has been proposed as an alternative pathway for diagnosis and monitoring of disease activity. This study aimed to compare the number of healthcare interactions required to reach a diagnosis of asthma using the ASTEC score model versus the joint BTS/NICE/SIGN guideline pathway in the SMART study population.

Methods The SMART study (NCT05357274) enrolled 230 patients with a primary care diagnosis of asthma with persistent symptoms despite treatment. A definitive diagnosis of asthma and/or alternative condition was established using lung function, markers of T2 inflammation and symptoms at five timepoints. Both the BTS/NICE/SIGN guideline pathway and the ASTEC were applied retrospectively as rule- in diagnostic pathways, each incorporating one round of repeat testing prior to BHR. Once positive diagnostic criteria were met, no further testing or appointments were deemed necessary.

Results Complete data were available for 220 patients; 133 (60%) received a diagnosis of asthma and 87 (40%) did not. The BTS/NICE/SIGN pathway required a maximum of 7 healthcare interactions, comprising clinic visits and lung function appointments, compared to a maximum of 4 in the ASTEC pathway. Mean interactions to diagnosis were 4.08 and 1.84 respectively. BHR testing was required in 122 patients (55%) under BTS/NICE/SIGN guidance compared to 92 patients (42%) under the ASTEC pathway, representing a 25% reduction in BHR referral.

Conclusions Asthma diagnosis remains healthcare-intensive, with over half of patients in this cohort requiring BHR testing, a resource with no accessibility in primary and limited in secondary care. The ASTEC score model significantly reduces the number of healthcare interactions and BHR referrals required to reach a diagnosis, offering a more efficient and accessible diagnostic pathway that warrants further prospective evaluation.

P254 PHYSICAL ACTIVITY AND MORTALITY IN ASTHMA, COPD, AND OBSTRUCTIVE SPIROMETRY: AN 8-YEAR POPULATION-BASED COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.414

Introduction and Objectives Physical activity (PA) is known to associate with decreased mortality in patients with COPD, whereas evidence on the association in asthma remains limited, and no previous studies have used objectively measured PA to assess its association with mortality in asthma patients. Similarly, sedentary behaviour (SB) is associated with increased mortality in COPD, but its association in asthma remains unknown. This study aimed to evaluate the effect of objectively measured PA and SB on all-cause mortality in participants with asthma, COPD, or obstructive spirometry.

Methods Health 2011 was a Finnish population survey with a sample of 8177 adults aged ≥29 years. The data was collected via questionnaires and measurements, including spirometry (n=4100). A subsample wore an accelerometer (n=1635). The association of baseline PA or SB and baseline self-reported asthma, COPD, or measured obstruction and all-cause mortality between 2011-2019 was analysed using logistic regression.

Results We found a significant association between light PA and lower mortality in participants with asthma, COPD, and obstructive spirometry. The odds ratios (ORs, 95% confidence intervals [CIs]) for these associations were 0.75 (0.59-0.96), 0.76 (0.59-0.97), and 0.75 (0.57-0.98), compared to those without asthma, COPD, and obstructive spirometry, respectively. Regarding SB, there was an association between time spent in lying posture and higher mortality in all three groups, with ORs (95% CIs) 1.73 (1.20-2.50), 1.85 (1.28-2.67), and 1.76 (1.19-2.61), compared to those without asthma, COPD, and obstructive spirometry, respectively.

Conclusions Analogously to COPD, greater light PA was associated with lower mortality, whereas time spent lying down was associated with higher mortality among participants with asthma and obstructive spirometry. To our knowledge, no previous studies have examined these associations using objectively measured PA and SB.

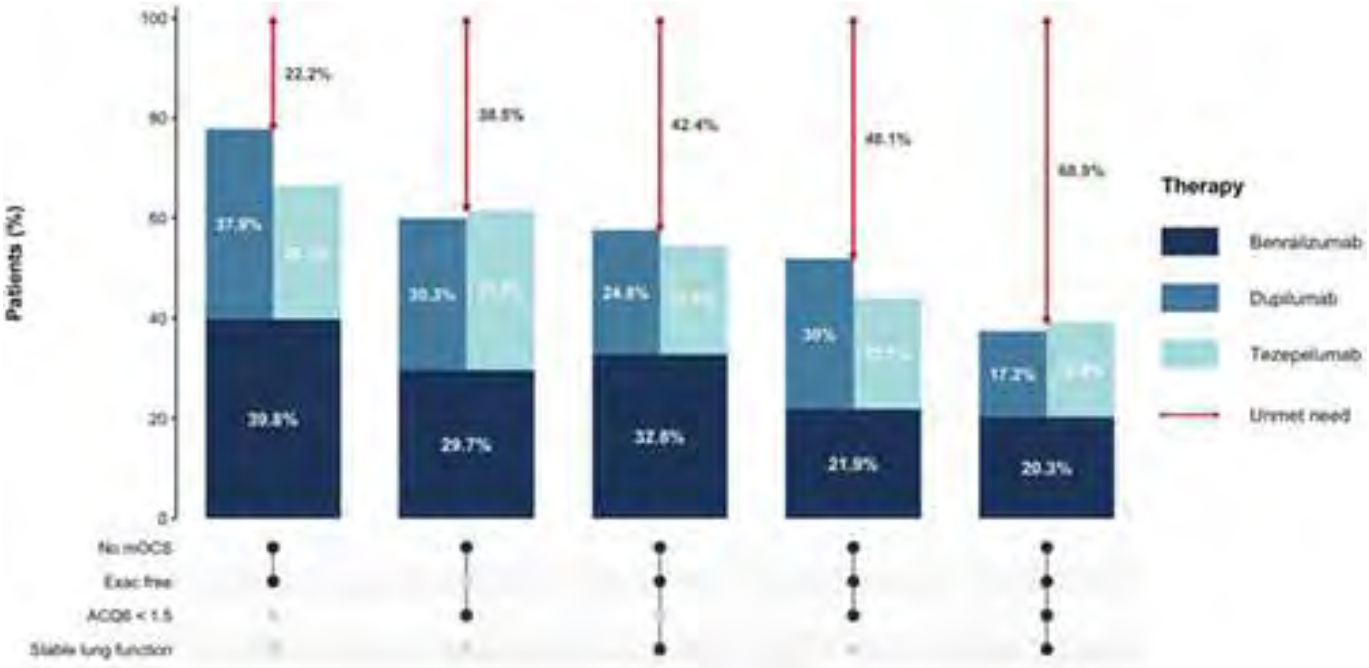
P255 IDENTIFYING THE RESIDUAL UNMET NEED IN SEVERE ASTHMA

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10.1136/thorax-2026-BTSabstracts.415

Introduction The currently available biologic therapies in severe asthma (SA) predominantly target either the IL-5/eosinophil pathway or the IL-13/T2 pathway. Clinical trials highlight similar outcomes with each therapy leading to a halving of exacerbations and clinical remission rates of 25-30%. A key unresolved question is whether patients failing one approach adequately benefit from targeting the another. An assessment of this will allow an understanding of the residual unmet in severe asthma and whether novel anti-T2 approaches are still required.

Methods We performed a new analysis of previously published biologic response datasets from our asthma centre. Patients treated with benralizumab (n=130), or switched to dupilumab (n=65) or tezepelumab (n=106) following inadequate anti-IL-5R response, were included if they had completed ≥12 months on both biologics. Mepolizumab-treated patients were excluded due to the possibility of persistent eosinophilic inflammation despite treatment. Inadequate response was defined as ongoing severe exacerbations or failure to reduce maintenance



Abstract P255 Figure 1

oral corticosteroids (mOCS) by ≥50%. Clinical remission was assessed across four domains: mOCS-free status, exacerbation-free status, ACQ6 <1.5, and stable lung function (FEV1 decline <5% from baseline). To quantify residual unmet need, response and remission rates for dupilumab and tezepelumab were calculated conditionally on the proportion of patients who had failed to achieve each threshold on benralizumab.

Results Our analysis highlights that 77.8% of patients with SA appear able to achieve the important steroid-free goal of becoming exacerbation-free and off mOCS for asthma. The residual unmet need of 22.2% rises to 48.1% when the additional requirement of symptom control (ACQ6 <1.5) is included. When the bar is raised to clinical remission, 60.9% were unable to achieve this goal (figure 1).

Conclusion Despite sequential targeting of multiple T2 pathways approximately 1 in 4 patients with SA continue to require OCS, and over 60% fail to achieve clinical remission. T2 biomarker levels suggests this partly reflects a failure to achieve biologic remission with current treatment options. Improved exacerbation phenotyping and associated steroid stewardship in patients achieving biologic remission may reduce the apparent unmet need further.

P256 ASSESSING THE ROLE OF SOCIAL DETERMINANTS OF HEALTH IN ASTHMA HOSPITALISATIONS USING THE CLINICAL PRACTICE RESEARCH DATALINK

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10.1136/thorax-2026-BTSabstracts.416

Background Socioeconomic deprivation and minority ethnicity have been shown to be associated with increased risk of adverse asthma outcomes in the literature. This phenomenon is under-researched in the United Kingdom (UK). Our study aimed to understand the impact of socioeconomic status (SES) and ethnicity on the odds of first adult asthma hospitalisation in England.

Methods A retrospective case-control study for April 2013 – March 2023 was conducted using the Clinical Practice Research Datalink (CPRD) and Hospital Episode Statistics Admitted Patient Care (HES APC) datasets. Adults aged over 18 and hospitalised for asthma between 2013 and 2023 were age and sex matched with people who had evidence of an asthma code within 1 year of the index date and no history of an adult asthma hospitalisation. Ethnicity was derived using the CPRD Aurum Ethnicity Algorithm, classified according to the categories of the 2021 Census for England, Wales and Northern Ireland. SES was classified using small area level index of multiple deprivation (IMD).

Odds of hospitalisation were derived using a conditional logistic regression model to account for matching. Odds ratios (OR) for ethnicity and SES were mutually adjusted.

Results A total of 94 392 people (19 372 cases, 75 020 controls) were included in the final study population. Sixty-five percent of the study population were female, with a mean age at index date of 42 years (standard deviation 18 years). Socioeconomic deprivation and some UK minority ethnic heritages were associated with increased odds of asthma hospitalisation (see table 1).

Conclusion Socioeconomic deprivation and certain UK minority ethnic heritages were associated with increased odds of

Abstract P256 Table 1 Index of multiple deprivation (IMD) †, UK minority ethnicity‡ and odds of asthma hospitalisation

	Odds ratio	95% LCI	95% UCI	p
IMD†				
– **IMD 1** ***(reference value, least deprived)***	1.00			
– **IMD 2**	1.10	1.01	1.19	0.022*
– **IMD 3**	1.15	1.06	1.24	<0.001*
– **IMD 4**	1.20	1.11	1.29	<0.001*
– **IMD 5**	1.28	1.19	1.39	<0.001*
– **IMD 6**	1.33	1.24	1.44	<0.001*
– **IMD 7**	1.49	1.38	1.60	<0.001*
– **IMD 8**	1.64	1.53	1.77	<0.001*
– **IMD 9**	1.77	1.64	1.90	<0.001*
– **IMD 10** ***(most deprived)***	2.07	1.93	2.23	<0.001*
UK minority ethnicity‡				
– **White: British** ***(reference value)***	1.00			
– **White: Irish**	0.92	0.74	1.16	0.500
– **White: Gypsy/ Irish Traveller**	*Censored*	*Censored*	*Censored*	*Censored*
– **White: Roma**	*Censored*	*Censored*	*Censored*	*Censored*
– **White: Other**	1.10	1.05	1.15	<0.001*
– **Mixed: White, Black Caribbean**	1.04	0.82	1.32	0.731
– **Mixed: White, Black African**	1.55	1.10	2.18	0.011*
– **Mixed: White, Asian**	0.96	0.71	1.29	0.781
– **Mixed: Other multiple ethnicity**	1.27	1.08	1.51	0.004*
– **Asian: Indian**	1.79	1.64	1.95	<0.001*
– **Asian: Pakistani**	2.05	1.88	2.24	<0.001*
– **Asian: Bangladeshi**	1.71	1.46	1.99	<0.001*
– **Asian: Chinese**	0.93	0.72	1.21	0.598
– **Asian: Other**	1.92	1.75	2.11	<0.001*
– **Black: African**	1.62	1.45	1.81	<0.001*
– **Black: Caribbean**	1.51	1.33	1.72	<0.001*
– **Black: Other**	1.71	1.49	1.97	<0.001*
– **Other: Arab**	1.77	1.13	2.78	0.013*
– **Other: Other**	1.32	0.96	1.81	0.084
– **Unknown**	0.81	0.73	0.90	<0.001*

†adjusted for ethnicity by Census 2021 categories
‡adjusted for IMD by IMD deciles

asthma hospitalisation. As evidence mounts demonstrating the effectiveness of personalised care in asthma, this study provides evidence of underserved groups who may benefit from targeted intervention.

Assessing the role of social determinants of health in asthma hospitalisations using the Clinical Practice Research Datalink.

P257 ASSESSING DEMOGRAPHIC DISPARITIES IN SEVERE ASTHMA: ACCESS TO CARE, LUNG FUNCTION, AND TREATMENT PATTERNS AT A TERTIARY RESPIRATORY CARE CENTRE

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10.1136/thorax-2026-BTSabstracts.417

Background Severe asthma (SA) imposes a substantial clinical and economic burden, with disproportionately worse outcomes reported among females and ethnic minority populations. However, the influence of demographic factors on disease severity at presentation and access to specialist care within a public healthcare system remains unclear.

Methods This retrospective cohort study included 448 patients referred to a UK tertiary respiratory centre for SA assessment. Clinical, functional, and inflammatory characteristics were compared by biological sex and ethnicity. Healthcare utilisation, pre-referral steroid exposure, lung function, and need for biologic therapy were analysed. Cohort ethnicity was compared with regional census data to assess representativeness.

Results The cohort had a mean age of 52.0 years, was predominantly female (63.8%) and Caucasian (82.8%). Male patients were older at first assessment (57.5 vs 51.0 years, p=0.01) and symptom onset (30.5 vs 20.0 years, p=0.023), with higher eosinophil counts (0.8 vs 0.63 $\times 10^9$/L, p=0.0011) and IgE levels (180 vs 118.5 IU/mL, p=0.001). Males demonstrated better lung function, including higher pre- and post-bronchodilator FEV1 and FVC ($p < 0.0001$), and had lower ACQ7 scores (2.57 vs 3.29, p<0.0001) and reduced ED attendance ($p = 0.015$) and hospital admissions (p=0.003). ANOVA showed no significant ethnic differences across most variables (p>0.05). However, Asian patients had lower pre-bronchodilator FVC (2.93 vs 3.29 L, p=0.037), higher ACQ7 scores (p<0.05), and required biologic therapy more frequently (84.2% vs 47.0%, p<0.05; Cramer's V = 0.26), suggesting greater disease severity at specialist assessment. Despite comprising 13.4% of the local population, Asian patients accounted for only 8.5% of referrals. Ethnic distribution differed significantly from the regional population ($\chi^2 = 37.16$, p<0.0001).

Conclusions Significant demographic disparities exist in SA severity and specialist referral patterns. Asian patients presented with markers of more severe disease and were more likely to require biologic therapy, yet were under-represented among tertiary referrals relative to the local population. These findings raise concern regarding delayed referral, barriers to specialist access, or other factors contributing to more advanced disease at presentation. Further investigation is warranted to identify and address potential inequities in severe asthma care pathways.

P258 SOCIOECONOMIC DEPRIVATION AND RISK OF ICU ADMISSION IN ASTHMA: A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.418

Background Socioeconomic deprivation is associated with poorer asthma outcomes and increased asthma mortality.[1] However, its relationship with life-threatening asthma requiring

intensive care unit (ICU) admission remains poorly characterised. We evaluated the association between Index of Multiple Deprivation (IMD) decile and ICU admission for refractory asthma.

Methods We conducted a retrospective cohort study of all ICU admissions for refractory asthma between 2018 and 2020, prior to the COVID-19 pandemic. Patient postcodes were mapped to IMD deciles. Emergency department (ED) attendance frequency and BTS/SIGN treatment step were analysed.

Results Sixty-three ICU admissions with refractory asthma were identified. Patients from IMD deciles 1–2 accounted for 32/63 (50.8%) admissions despite representing only 31.6% of the local population, corresponding to a 1.6-fold overrepresentation. A further 23/63 (36.5%) admissions occurred in patients from IMD deciles 3–5, compared with 32.0% of the local population, while only 8/63 (12.7%) admissions occurred in patients from IMD deciles 6–10.

The relationship between ED attendance frequency and IMD decile was less pronounced. However, differences in preventative treatment intensity were observed across deprivation groups. No patients from IMD deciles 7–9 were prescribed therapy below BTS/SIGN step 4, suggesting higher levels of treatment optimisation among less deprived populations.

Conclusions Socioeconomic deprivation was associated with disproportionate representation among ICU admissions for refractory asthma, with patients from the most deprived IMD deciles markedly overrepresented among life-threatening exacerbations. These findings suggest potential inequalities in asthma optimisation and access to specialist care. Addressing socioeconomic disparities in asthma optimisation may represent a modifiable strategy to reduce life-threatening exacerbations and critical care utilisation.

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P259 RECRUITING AND RETAINING PARTICIPANTS FROM DEPRIVED COMMUNITIES INTO AN ASTHMA DIAGNOSTIC STUDY

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10.1136/thorax-2026-BTSabstracts.419

Introduction Limited access to asthma diagnostic tests and poor disease outcomes disproportionately affect people living in deprived areas. Inclusive research mandates equitable access, participation and retention across socioeconomic groups. However, deprivation may influence engagement with diagnostic studies and contribute to under-representation of underserved populations. We investigated the impact of socioeconomic factors on study retention in a diagnostic study of people with suspected asthma.

Method The RADicA study evaluates novel diagnostics by providing rapid-access assessment for people who present to primary care with symptoms suggestive of asthma. Eligible participants were identified using an electronic alert in an electronic health system during routine GP consultations and referred to the study. Once consented, participants could withdraw at any point without giving any reasons. The study involved three to five visits over a 12-week period, including comprehensive diagnostic asthma testing and a 4-week trial of inhaled cortico-steroid. Supportive retention strategies included flexible appointment scheduling, text and telephone reminders, accessible telephone support for participant queries, and travel assistance (including paid taxi transport and parking where required). Study retention was defined as completing a minimum of a baseline and final visit. Index of Multiple Deprivation (IMD) decile was assigned based on postcode.

Results Of 493 consented participants (274 adults [≥16 years] and 219 children [3–15 years]). 84% were recruited from the most deprived areas (IMD decile ≤4), 56% were female and 28% from minority ethnic groups, with an overall retention rate of 77%. Attrition rate was higher in adults than children (26% vs 18%, p=0.034). Socioeconomic factors, including IMD, ethnicity, sex, distance to clinic, smoking status, mental health conditions, housing and employment factors, were not associated with study retention (table 1).

Conclusion We recruited a population with high levels of socioeconomic deprivation, and study retention was not influenced by socioeconomic factors. Withdrawal was less common in children than adults, potentially reflecting greater parental motivation to pursue a definitive diagnosis for their child. Simple inclusive research strategies as described here, may have influenced the high retention rate achieved, and could be applied to research and clinical practice in other deprived populations.

Abstract P259 Table 1 Baseline demographic and clinical characteristics stratified by study retention for adults (≥16 years) and children (<16 years). Continuous variables presented as median [IQR] with Wilcoxon rank-sum p-values; categorical variables presented as n (%) with chi-square. Rows with p<0.05 are highlighted

	Adults (≥16 Years)			Children (<16 Years)		
Variable	Completed N=202	Withdrawn N=72	p-value	Completed N=179	Withdrawn N=40	p-value
Female, n (%)	127 (62.9%)	47 (65.3%)	0.825	84 (46.9%)	17 (42.5%)	0.740
Age, years, median [IQR]	33.5 [25.0-42.0]	32.5 [24.0-42.8]	0.711	8.0 [6.0-10.0]	7.0 [5.8-10.0]	0.244
Distance to clinic (km), median [IQR]	8.8 [6.6–9.5]	7.7 [6.8–8.6]	0.269	7.1 [6.1–8.8]	5.7 [5.5–8.0]	0.146
Minority Ethic Groups, n (%)	51 (25.2%)	22 (30.6%)	0.409	58 (32.4%)	8 (20.0%)	0.220
IMD decile, median [IQR]	3.0 [2.0–4.0]	3.0 [2.0–4.0]	0.1704	3.0 [2.0–4.0]	3.0 [2.0–4.0]	0.0005
Ever smoked, n (%)	61 (30.2%)	19 (26.4%)	0.8553	1 (0.6%)	0 (0.0%)	1.0000
Unemployment, n (%)	14 (7.2%)	10 (15.4%)	0.086	N/A	N/A	N/A
Any mental health condition, n (%)	26 (12.9%)	15 (20.8%)	0.1516	10 (5.6%)	3 (7.5%)	0.7100

* Distance to clinic calculated as straight-line distance (km) from patient postcode centroid to clinic (M23 9QZ), geocoded via the postcodes.io bulk API and computed using the Haversine formula (Earth radius = 6,371 km).

Type 2 COPD: biomarkers, biologics and outcomes

P260 BLOOD EOSINOPHIL COUNTS AND INCIDENT ASTHMA AND COPD: PROSPECTIVE ANALYSES IN UK ADULTS

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10.1136/thorax-2026-BTSabstracts.420

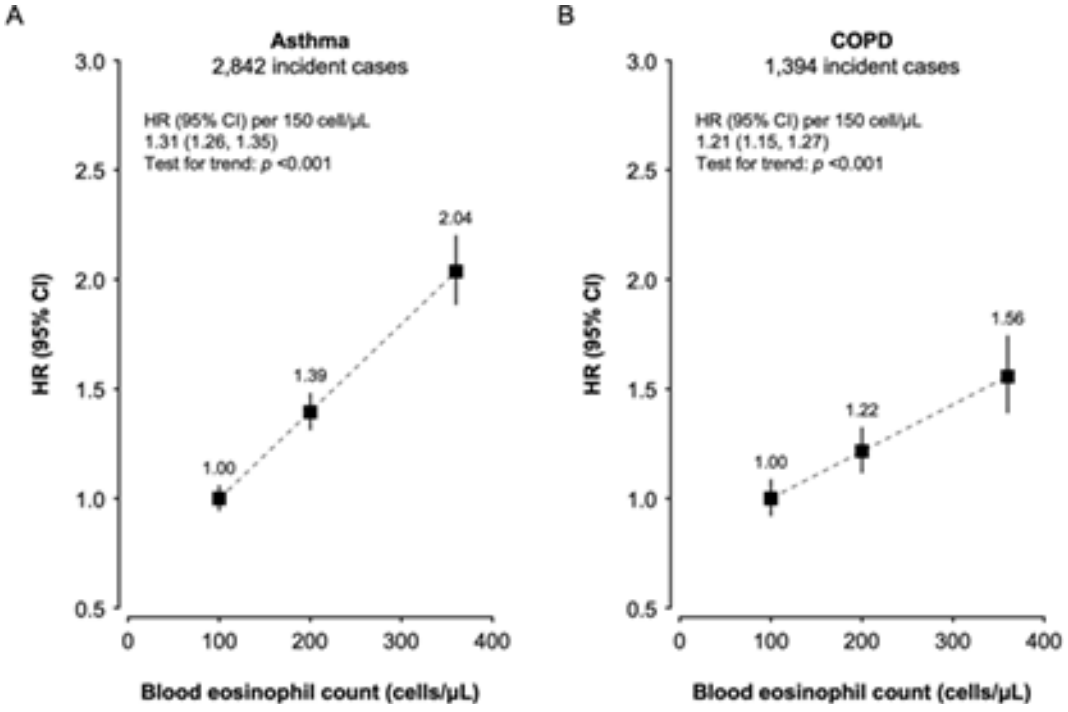
Background The blood eosinophil count (BEC) is a biomarker of type-2 inflammation in asthma and chronic obstructive pulmonary disease (COPD) with therapeutic and prognostic implications. Whether type-2 inflammation is solely a marker of existing disease or also a driver of airway disease pathogenesis, and how early it may be detected before disease onset, is uncertain.

Aim To investigate the association between BEC and incident asthma and COPD in individuals without pre-existing lung disease, and determine how far in advance of diagnosis BEC may be associated with future airway disease.

Methods A prospective cohort analysis was conducted in UK Biobank among participants without prior lung disease using BEC and spirometry assessed at baseline. Incident cases were identified from linked primary and secondary care records using diagnostic code lists derived from published sources. Multivariable Cox regression estimated adjusted hazard ratios (HR) for incident asthma and COPD by BEC. Additional analyses examined associations in participants with normal baseline spirometry and according to the time interval between BEC measurement and diagnosis. As internal validation, analyses were repeated in a broader UK Biobank population unrestricted by primary care data availability, using a distinct outcome ascertainment strategy based on secondary care and mortality records.

Findings Among 128,404 participants followed for a median 7.9 years, there were 2,824 incident cases of asthma and 1,394 cases of COPD. Compared with BEC <150 cells/uL, BEC ≥300 cells/uL was associated with a 2-fold higher risk of asthma (2.04 [1.85-2.25]) and a 56% higher risk of COPD (1.56 [1.35-1.79]) after multivariable adjustment. This relationship was approximately linear in shape, with higher BEC linked to progressively greater risks of incident disease (figure 1). Associations were unchanged in participants with normal spirometry at baseline and when BEC measurements preceded diagnosis by ≥7 years. Findings were replicated in the larger internal validation cohort (n=287,305) with longer follow-up and more incident cases, yielding concordant patterns and magnitudes of association.

Interpretation These findings support a temporal relationship in which type-2 inflammation precedes clinically recognised airway disease even before spirometric abnormalities are apparent. A single BEC may help identify individuals at increased risk of future asthma and COPD.



Abstract P260 Figure 1 Adjusted hazard ratios for the risk of incident asthma (A) and COPD (B) by blood eosinophil count HR are plotted against the median blood eosinophil value in each category. Models are stratified by age-at-risk and region, and adjusted for sex, ethnicity, region, FEV, (% predicted), body mass index, smoking status, smoking intensity, years since smoking cessation, secondhand smoke, and atopic disease. BEC, blood eosinophil count; CI, confidence interval; COPD, chronic obstructive pulmonary disease; HR, hazard ratio

P261 PREDICTORS OF BLOOD EOSINOPHIL TESTING IN MODERATE-TO-SEVERE COPD: A UK RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.421

Background GOLD recommends blood eosinophil count (BEC) to guide COPD treatment. Despite widespread triple therapy use, many with elevated BEC still exacerbate, and factors linked to BEC testing remain unclear.
Objectives To assess COPD outcomes by baseline BEC testing; to identify predictors of BEC testing among patients with moderate-to-severe COPD on triple therapy in England.
Methods Retrospective cohort study (GSK 222888) using CPRD Aurum–HES data. Adults aged ≥35 years with COPD were indexed on most recent exacerbation (Feb 2019–Feb 2022) and required ≥2 moderate exacerbation or ≥1 severe in prior year, GP registration ≥12-month pre-/≥6-month post-index, ≥1 triple therapy in 3-month pre-index. Outcomes: exacerbations, mortality and healthcare resource use (HCRU) during FU by baseline BEC testing. Multivariate logistic regression identified baseline predictors of FU BEC testing.
Results Of 35,095 patients, 46.4% had baseline BEC testing. Tested patients had fewer exacerbations (0.77 vs 0.93 events/person-year), similar mortality (0.28 vs 0.25) and greater HCRU: primary care (16.83 vs 13.42) and outpatient visits (5.59 vs 4.45). During FU, 33.8% had BEC testing. Higher odds of testing predictors: older age, higher BMI, comorbidities requiring regular blood labs, prior primary care/outpatient visits. Lower odds: frailty-related comorbidities, frequent reliever use and prior COPD hospitalisations.
Conclusion BEC testing was associated with better exacerbation control and care quality, likely reflecting treatment adjustments informed by results. Routine testing, especially in under-tested groups, may enable personalised therapy and reduce reliance on reactive COPD management.
Funding GSK (222888).
Previously submitted to ERS2026.

P262 PERSISTENT EXACERBATION AND MORTALITY IN COPD WITH TYPE 2 INFLAMMATION DESPITE TRIPLE THERAPY: A UK RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.422

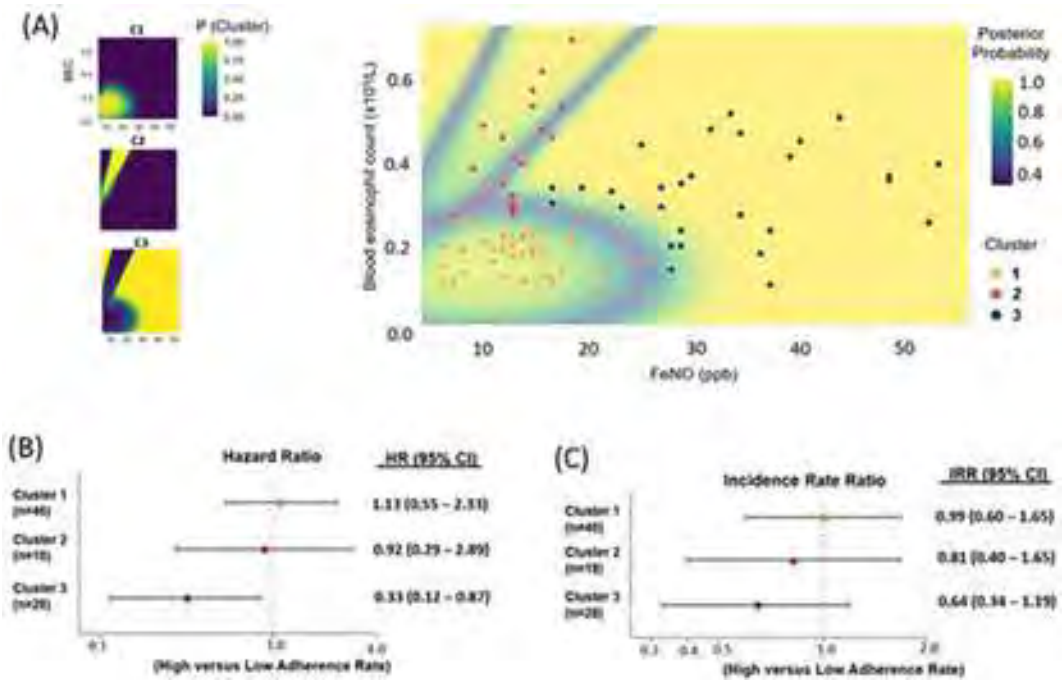
Background Blood eosinophil count (BEC) may guide treatment in COPD, per GOLD guidelines; however, real-world evidence on exacerbation and mortality in patients with type 2 (T2) inflammation is limited.
Objectives To assess exacerbation and mortality in frequently exacerbating patients with COPD with a T2 inflammatory phenotype (elevated BEC) receiving maintenance triple therapy in England.
Methods Retrospective cohort study (GSK 222888) using CPRD Aurum–HES data. Adults aged ≥35 years with COPD were indexed on their most recent exacerbation (Feb 2019–Feb 2022). Eligibility: ≥2 moderate or ≥1 severe exacerbation in prior year, GP registration ≥12 months pre- and ≥6 months post-index, ≥1 triple therapy prescription in the 3 months pre-index, and baseline BEC results. Outcomes: exacerbations and all-cause/COPD-related mortality, stratified by BEC ≥300 and ≥150 cells/μL. No minimum FU applied for mortality assessment.
Results Among 16296 patients, 35.4% had BEC ≥300 and 70.3% ≥150 cells/μL. In the ≥300 group (n=5769), 34.8% had ≥1 exacerbation during 12-month FU (rate: 0.78 events/person-year, 95% CI: 0.75–0.80). Of 7821 patients assessed for 1-year mortality, 27.2% died (all-cause) and 20.0% COPD-related deaths. In the ≥150 group (n=11,452), 35.2% had ≥1 exacerbation (rate: 0.76, 95% CI: 0.75–0.78). Of 15,370 assessed for 1-year mortality, 26.1% died (all-cause) and 19.6% COPD-related.
Conclusion One-third of frequently exacerbating patients with COPD with T2 inflammation remain at risk of subsequent exacerbation despite triple therapy. Findings highlight a persistent unmet need; biologics targeting T2 inflammation may improve outcomes in this population.
Funding GSK (222888).
Previously submitted at ERS2026.

P263 WHAT DOES FENO ADD BEYOND BLOOD EOSINOPHILS? JOINT BIOMARKER PHENOTYPING FOR ASSESSING INHALED CORTICOSTEROIDS RESPONSE IN COPD

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10.1136/thorax-2026-BTSabstracts.423

Background We previously demonstrated[1] that both fractional exhaled nitric oxide (FeNO) and blood eosinophil count (BEC) can independently identify inhaled corticosteroids (ICS) response in COPD when objectively adherent. However, FeNO and BEC are often discordant[1], capturing distinct aspects of type 2 inflammation. Whether combining these biomarkers improves identification of ICS-responsive disease remains unknown.
Aim To determine whether combined FeNO-BEC phenotyping improves prediction of ICS responsiveness beyond either biomarker alone.
Methods Patients with COPD receiving triple inhaled therapy and ≥2 exacerbations in the preceding year underwent six months of digital inhaler monitoring (NCT0581448). Participants were stratified using conventional thresholds (FeNO ≥20ppb, BEC ≥0.3×10^9/L). Adherence-associated exacerbation outcomes were assessed across joint FeNO-BEC phenotypes. Data-driven Gaussian mixture modelling was performed to evaluate whether findings were dependent on predefined biomarker thresholds.
Results Among 104 participants (Age 67±9 years; FEV_1 1.1±0.5L), concurrent FeNO and BEC elevation identified the subgroup with the greatest clinical benefit from high ICS



Abstract P263 Figure 1 Data-driven inflammatory phenotypes derived from joint FeNO and blood eosinophil count (BEC) distributions and their association with exacerbation outcomes. (A) Three inflammatory clusters identified by Gaussian mixture modelling. Cluster 1 represents a low FeNO/low BEC phenotype (n=46), Cluster 2 a low FeNO/high BEC phenotype (n=18), and Cluster 3 a high FeNO with moderate-to-high BEC phenotype (n=28). Left: posterior probability heatmaps showing the probability of cluster assignment across the FeNO-BEC space. Right: individual participant distribution by assigned cluster, with overlaid posterior probability contours and decision boundaries. Posterior probability values (0 - 1) represent the model-estimated likelihood of cluster allocation. (B) Forest plot showing adjusted hazard ratios (HRs) for time to first exacerbation comparing high versus low adherence within each cluster. (C) Forest plot showing incidence rate ratios (IRRs) for exacerbations across clusters

adherence (≥75% of prescribed doses taken), with significantly prolonged time to the first exacerbation (HR 0.26, 95% CI: 0.07-0.89, n=17). Similar direction was observed among those with either biomarker elevation: BEC-low/FeNO-high (n=12; HR 0.31, 95% CI: 0.07-1.48) and BEC-high/FeNO-low (n=19; HR 0.59, 95% CI: 0.17-2.04). No benefit was observed in participants with low levels of both biomarkers (BEC-low/FeNO-low, n=37; HR 1.26, 95% CI: 0.60-2.64). Exacerbation rate reduction followed the same pattern.

In data-driven phenotyping, High-FeNO/moderate-high BEC cluster (n=28) demonstrated the greatest reduction in the exacerbation risk (HR 0.33, 95%CI 0.12–0.87) compared to low FeNO/low BEC (n=46) and low FeNO/high BEC (n=18) clusters. Adherence-associated reductions in exacerbation rates increased with T2 biomarker elevation (see figure 1). Cluster-derived thresholds (FeNO 24 ppb; BEC 0.28×10^9/L) closely approximated conventional cut-offs, although non-linear Bayes boundaries suggested that the FeNO-BEC relationship is not fully captured by simple high-low classification. Findings were consistent across smoking strata.

Conclusion Combined FeNO-BEC phenotyping captured the greatest ICS-responsive COPD phenotype and FeNO may identify additional ICS sensitive subgroup missed by eosinophil-based thresholds alone. T2 inflammatory phenotyping in COPD may require more than one dimension of assessment.

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P264 COPD ASSESSMENT TEST SCORES IN PATIENTS MEETING NICE/MHRA DUPILUMAB ELIGIBILITY CRITERIA: A REAL WORLD ANALYSIS

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Introduction and Objectives Chronic obstructive pulmonary disease (COPD) is a heterogeneous disease characterised by diverse inflammatory pathways and variable treatment responses. Despite optimised inhaled therapy, many patients continue to experience recurrent exacerbations and significant symptom burden. Recognition of type 2 (T2) eosinophilic inflammation as a treatable trait has led to the development of targeted biologic therapies. Dupilumab is the first biologic licensed for patients with uncontrolled COPD and raised eosinophils. This study examined the relationship between COPD Assessment Test (CAT) scores and dupilumab eligibility, and assessed whether patients identified by current eligibility criteria represent a population with significant symptomatic burden.

Methods We conducted a retrospective observational study of COPD patients meeting current NICE/MHRA dupilumab eligibility criteria at a tertiary care centre. Demographic and clinical data, including eosinophil counts, exacerbation history, inhaled therapy, spirometry, smoking status were collected. CAT Scores assessed symptom burden.

Results Total of 123 patients fulfilled NICE/MHRA eligibility criteria. Mean age was 69.8 years; 64/123 (52.03%) were female and 59/123 (47.97%) were male. Smoking status was available for 118 patients, of whom 90/118 (76.3%) were ex-smokers/non-smokers and 28/118 (23.7%) were current smokers. Mean and median blood eosinophil counts were 0.56 $\times 10^9$/L and 0.50 $\times 10^9$/L, respectively (data available for 122 patients). Most patients were receiving triple therapy (121/123, 98.37%). Frequent exacerbations (≥2/year) were observed in 100/121 patients (82.64%).

Lung function data were available for 106 patients. Mean FEV_1 was 47.4% predicted. Overall, 63/106 (59.43%) demonstrated severe or very severe airflow limitation.

CAT scores were available for 79 patients. Mean CAT score was 25.6 and median score was 27. Only 3/79 (3.8%) had CAT scores <10, while 60/79 (75.9%) had scores >20, indicating high symptom burden. Scores of 21–30 were recorded in 37/79 (46.8%) and >30 in 23/79 (29.1%).

Conclusions Patients identified using NICE/MHRA dupilumab eligibility criteria represent a population with advanced COPD and substantial symptom burden. Although symptom severity is not included within the eligibility framework, over three-quarters of patients reported CAT scores consistent with a high or very high disease impact. These findings suggest that current eligibility criteria represents significant unmet clinical need.

P265 SERVICE EVALUATION OF DUPILUMAB ELIGIBILITY IN COPD PATIENTS WITH ACUTE EXACERBATIONS ACROSS WIRRAL

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10.1136/thorax-2026-BTSabstracts.425

Background Dupilumab has been recommended by NICE as an add-on maintenance treatment for patients with uncontrolled chronic obstructive pulmonary disease (COPD) with type 2 inflammation. Estimating the number of eligible patients is important for service planning and implementation of biologic therapy pathways. We evaluated the proportion of COPD patients presenting with acute exacerbations across Wirral meeting modified NICE eligibility criteria for dupilumab.

Methods A retrospective service evaluation was conducted across inpatient wards, the emergency department and the respiratory virtual ward in Wirral. Patients with COPD experiencing at least 1 acute exacerbation between February 2025 and February 2026 were identified. Clinical data, including exacerbation frequency, blood eosinophil counts, inhaled therapy and spirometry, were collected from healthcare records. Moderate and severe exacerbations were recorded separately and each counted up to four events, with higher counts categorised as ≥4. Eligibility for dupilumab was assessed using modified NICE criteria, including exacerbation burden (≥2 moderate or ≥1 severe exacerbations), blood eosinophil count ≥300 cells/μL within the preceding year and optimisation of inhaled therapy.

Results A total of 521 patients with COPD exacerbations were identified. Of these, 316 (61%) were female, with a mean age of 70 (SD 10). 228 (43.8%) met modified NICE eligibility criteria for dupilumab. The annualised moderate or severe exacerbation rate in the eligible population was 5.37 per patient-year (95% CI 5.11–5.63). Applying published dupilumab rate ratios from the BOREAS and NOTUS studies suggested a reduction of approximately 1.61–1.83 moderate/severe exacerbations per patient-year.

Conclusion Nearly half of COPD patients presenting with acute exacerbations across Wirral met modified NICE eligibility criteria for dupilumab. The eligible cohort had a higher exacerbation burden than reported in the pivotal dupilumab trial placebo arms, suggesting a high-burden real-world population in whom the absolute reduction in exacerbations may be considerable, although comparisons with trial populations should be interpreted cautiously. Applying published dupilumab rate ratios to our eligible population suggests prevention of approximately 370–420 moderate or severe exacerbations annually if eligible patients were treated. Establishing a structured pathway to identify, assess and monitor eligible patients may improve access to biologic therapy and support NICE guidance implementation.

P266 MEPOLIZUMAB REDUCES SYSTEMIC CORTICOSTEROID UTILISATION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION: POOLED RESULTS FROM PHASE III RANDOMIZED TRIALS

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10.1136/thorax-2026-BTSabstracts.426

Purpose Guidelines recommend oral corticosteroids (OCS) for chronic obstructive pulmonary disease (COPD) exacerbations. However, OCS are associated with complications highlighting an unmet need for targeted treatments to reduce OCS burden.

Methods Pooled analysis of Phase III randomized, placebo-controlled trials (METREX, METREO, MATINEE) in patients with COPD aged ≥40 years (≥1-year diagnosis), receiving inhaled triple-therapy, with prior exacerbations, GOLD 2–4 airflow limitation, and no asthma diagnosis. Blood eosinophil count (BEC) criteria were METREX (no restriction), METREO (≥150 cells/μL at screening or ≥300 cells/μL in previous year), and MATINEE (≥300 cells/μL at screening and ≥150 cells/μL in previous year). Patients received mepolizumab 100 mg subcutaneously or placebo every 4 weeks for 52–104 weeks. This analysis assessed proportion of days on OCS and overall OCS exposure. Patients were stratified by BEC ≥150 cells/μL at screening and/or ≥300 cells/μL in the previous year, or ≥300 cells/μL at screening. OCS doses were converted to prednisolone equivalents and categorised as low (<20 mg/day) or high (≥20 mg/day).

Results Of 2089 patients (1043 mepolizumab/1046 placebo), 1713 857/856) had BEC ≥150 cells/μL and 1144 (567/577) ≥300 cells/μL; 57% (457/510) and 55% (289/342) received OCS, respectively. Mean (SD) percentage days without OCS was 89.8% (20.9) vs 88.3% (21.7) in BEC ≥150 and 90.8% (19.4) vs 88.9% (21.3) in BEC ≥300 (mepolizumab vs placebo). In BEC ≥150 cells/μL, %days on OCS were similar at

low dose (5.7% vs 5.7%) but lower with mepolizumab at high dose (4.5% vs 5.9%). In BEC ≥300 cells/μL, values were 5.4% vs 6.1% (low) and 3.8% vs 5.1% (high).Mean (SD) total OCS exposure was lower with mepolizumab: 434.64mg (990.90) vs 609.95 (1392.30) mg/year in BEC ≥150 (mean difference 178.97 [95% CI 62.09, 292.19]) and 354.28 (761.53) vs 551.52 (1118.74) in BEC ≥300 (193.58 [84.01, 306.80]).

Conclusions Mepolizumab reduced total yearly exposure to OCS, in particular high-dose courses, in patients with COPD compared to placebo, regardless of screening BEC.

Clinical Implications Mepolizumab as add-on to inhaled triple therapy may reduce OCS burden in COPD, particularly complications associated with short-term high-dose courses, across a wide spectrum of screening BEC levels.

Funding GSK (117106/117113/208657).

Previously presented at ATS2026.

P267 SUSTAINED FEV1 IMPROVEMENTS WITH MEPOLIZUMAB UP TO 104 WEEKS IN PATIENTS WITH COPD AND LESS SEVERE AIRFLOW OBSTRUCTION: POST HOC ANALYSIS OF THE MATINEE PHASE III STUDY

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10.1136/thorax-2026-BTSabstracts.427

Background MATINEE assessed the efficacy of mepolizumab, a humanised monoclonal antibody specifically targeting interleukin-5, in patients with COPD up to Week (Wk) 104.

Aims Assess pre-bronchodilator FEV_1 change from baseline to Wk 104.

Methods MATINEE (NCT04133909) was a Phase III randomised, placebo-controlled, parallel-group trial enrolling a wide spectrum of patients with COPD, a history of exacerbations, and screening blood eosinophil count ≥300 cells/μL. Patients aged ≥40 years were randomised 1:1 to mepolizumab 100 mg or placebo, administered subcutaneously every 4 wks for a fixed 52-wk or variable 52–104-wk duration, alongside inhaled triple therapy. We report post hoc analyses of FEV_1 change from baseline to Wk 104, in patients treated for 52–104 wks, and by GOLD status (FEV_1 predicted: GOLD 2, 50–<80%; GOLD 3, 30–<50%; GOLD 4, <30%).

Results Of 804 randomised and treated patients, 459 (233 mepolizumab/226 placebo) participated up to 104 wks. FEV_1 (mL) improved from baseline to Wk 52 with mepolizumab and placebo (LS mean [SE] change: 31.9 [22.4] vs 38.9 [22.4]; p>0.05); by Wk 104 there was stabilisation in FEV_1 with mepolizumab vs a reduction with placebo (27.8 [25.9] vs −13.1 [27.4]; p>0.05). At Wk 104, improvements in FEV_1 with mepolizumab versus placebo were seen for GOLD 2 (n=336: 49.8 [34.5] vs –23.0 [34.9]) and GOLD 4 (n=106: 80.9 [60.1] vs −16.7 [62.3]), but with no treatment difference for GOLD 3 (n=318: −16.0 [40.3] vs −17.6 [47.0]).

Conclusions Mepolizumab was associated with lung function stabilisation up to Wk 104, particularly in patients with GOLD stage 2 and 4.

Funding GSK (208657).

Previously presented at ERS 2025.

P268 DUPILUMAB IS EFFICACIOUS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION REGARDLESS OF SMOKING STATUS

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10.1136/thorax-2026-BTSabstracts.428

Introduction Respiratory medications for COPD may be less efficacious in active smokers. In the BOREAS and NOTUS trials, dupilumab reduced exacerbations and improved lung function in patients with COPD.

Objective This pooled analysis of BOREAS and NOTUS trials evaluated the efficacy of dupilumab in current and former smokers with COPD.

Methods In Phase 3 BOREAS (NCT03930732) and NOTUS (NCT04456673) trials, patients with COPD on triple therapy, with moderate-to-severe airflow limitation, type 2 inflammation (screening eosinophils ≥300 cells/μL), and COPD symptoms received add-on dupilumab 300 mg or placebo every 2 weeks for 52 weeks. This *post hoc* analysis assessed adjusted annualised moderate or severe exacerbation rates and changes from baseline in pre- and post-bronchodilator FEV_1 by smoking status.

Results Former (dupilumab: n=662; placebo: n=654) and current smokers (dupilumab: n=276; placebo: n=282) were included. Dupilumab reduced relative risk of exacerbations vs placebo by 32% in former (relative risk vs placebo: 0.68; 95% CI: 0.57–0.81; $P<0.001$) and 31% in current smokers (relative risk: 0.69; 95% CI: 0.53–0.89; $P<0.005$). At Week 12, dupilumab improved pre-BD FEV_1 in the former (LS mean difference vs placebo: 88 mL; 95% CI: 50–126; $P<0.001$) and current smokers (64 mL; 95% CI: 19–109; $P<0.005$) (interaction P value: 0.359). Improved post-BD FEV_1 was also observed in the former (LS mean difference: 77 mL; 95% CI: 38–116; $P<0.001$) and current smokers (50 mL; 95% CI: 5–94; $P=0.028$) (interaction P value: 0.398).

Conclusions Dupilumab, compared with placebo, reduced exacerbations and improved lung function in patients with COPD and type 2 inflammation, regardless of smoking status.

P269 CLINICALLY MEANINGFUL BENEFIT OF TOZORAKIMAB OBSERVED INDEPENDENT OF BLOOD EOSINOPHIL COUNTS IN COPD: POOLED ANALYSIS OF THE OBERON/TITANIA PHASE 3 STUDIES

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10.1136/thorax-2026-BTSabstracts.429

Introduction The OBERON (NCT05166889) and TITANIA (NCT05158387) trials were replicate phase 3, randomized, double-blind, placebo-controlled studies of tozorakimab, an anti-IL-33 monoclonal antibody, in patients with COPD and a history of exacerbations. Currently available biologic therapies are approved for patients with elevated blood eosinophil count (BEC). This pooled analysis examined the relationship between screening BEC and the efficacy and safety of tozorakimab.

Methods In OBERON and TITANIA, eligible patients were current or former smokers with COPD and a history of exacerbations in the past year while on stable standard-of-care inhaled maintenance therapy. There were no selection criteria related to BEC. The primary analysis was a comparison of subcutaneous tozorakimab 300mg or placebo Q4W for 52 weeks. Prespecified pooled analyses included generalized additive models of the relationship between BEC at screening and the annualized rate of moderate/severe exacerbations in BEC subgroups.

Abstract P269 Figure 1 Generalized additive model of annualized rate of moderate/severe exacerbations by screening BEC.
BEC, blood eosinophil count; Q4W, every 4 weeks.

Results The pooled analysis set comprised 884 patients receiving tozorakimab 300mg Q4W and 866 receiving placebo. Baseline characteristics were balanced between treatment groups. Median BEC at screening was 190.0 and 177.3 cells/μL in the tozorakimab and placebo groups, respectively. BEC at screening were distributed as follows: <150 cells/μL: 38.8% and 40.4%, ≥150 cells/μL: 61.2% and 59.6%, ≥300 cells/μL: 24.8% and 24.6%, for tozorakimab and placebo, respectively. Tozorakimab reduced the annualized rate of moderate/severe exacerbations by 23% versus placebo in patients with screening BECs <150 cells/μL (rate ratio: 0.77 [95% CI: 0.62, 0.94]; p=0.0112), 34% in those with ≥150 cells/μL (0.66 [0.56, 0.78]; p<0.0001) and 43% (0.57 [0.44, 0.74]; p<0.0001) in those with ≥300 cells/μL. The estimated annualized rate of moderate/severe exacerbations was consistently lower in patients treated with tozorakimab versus placebo across the entire BEC range (figure 1). Exacerbation rate tended to increase with BEC in patients treated with placebo but not those treated with tozorakimab. The proportions of patients with adverse events were generally similar overall and across screening BEC subgroups.

Conclusion A clinically meaningful reduction in COPD exacerbations with tozorakimab was observed across all screening BEC thresholds in a broad population of current or former smokers with COPD and a history of exacerbations. Tozorakimab was well tolerated.

REFERENCE

1. No references provided in the submitted form.

P270 TREATMENT FAILURE AT 1 YEAR FOLLOWING BENRALIZUMAB DURING AN EOSINOPHILIC EXACERBATION: DATA FROM ABRA

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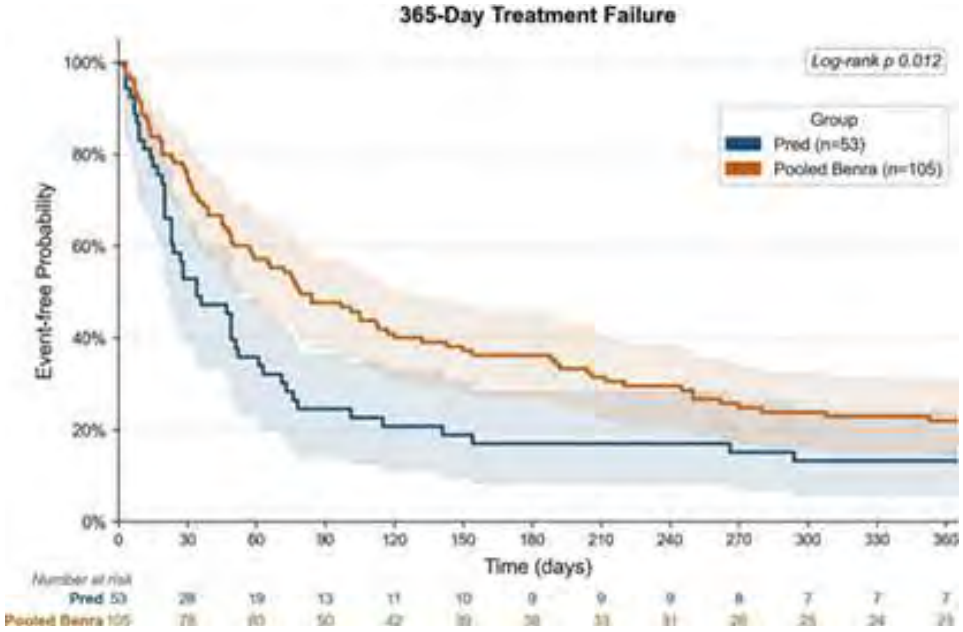
10.1136/thorax-2026-BTSabstracts.430

Introduction Exacerbations of airways disease are common and lead to significant morbidity and mortality, with up to half of patients either readmitted or dying following a hospitalised exacerbation of COPD. Improvements in outcomes are urgently needed.

ABRA was a phase 2, multi-centre, double-blind, double-dummy placebo-controlled randomised clinical trial. Patients with eosinophilic exacerbations (blood eosinophils ≥300 cells/μL) of asthma or COPD were randomised to receive: (1) benralizumab plus placebo (BENRA), (2) placebo plus prednisolone (PRED), or (3) benralizumab plus prednisolone (BENRA+PRED). Benralizumab was superior to prednisolone (standard of care) in reducing treatment failures at 90 days. Participants had further follow up at 365 days. These outcomes have not been reported.

Aim Describe treatment outcomes at 365-days between pooled BENRA and PRED groups.

Results There were 128 treatment failure events at 365 days (128/158, 81%). 82/105 (78%) occurred in pooled-BENRA group and 46/53 (87%) in PRED only group. Time to treatment failure differed significantly between treatment groups (log rank p=0.012), with 365-day time to treatment failure occurring with decreasing hazard in pooled benralizumab group (HR 0.63, 95% CI 0.44-0.91; p=0.012, figure 1).



Abstract P270 Figure 1

Conclusion After an eosinophilic exacerbation, treatment failure rates are high over 1 year. There was a sustained treatment benefit of a single treatment of benralizumab over 1 year, following an exacerbation compared to prednisolone.

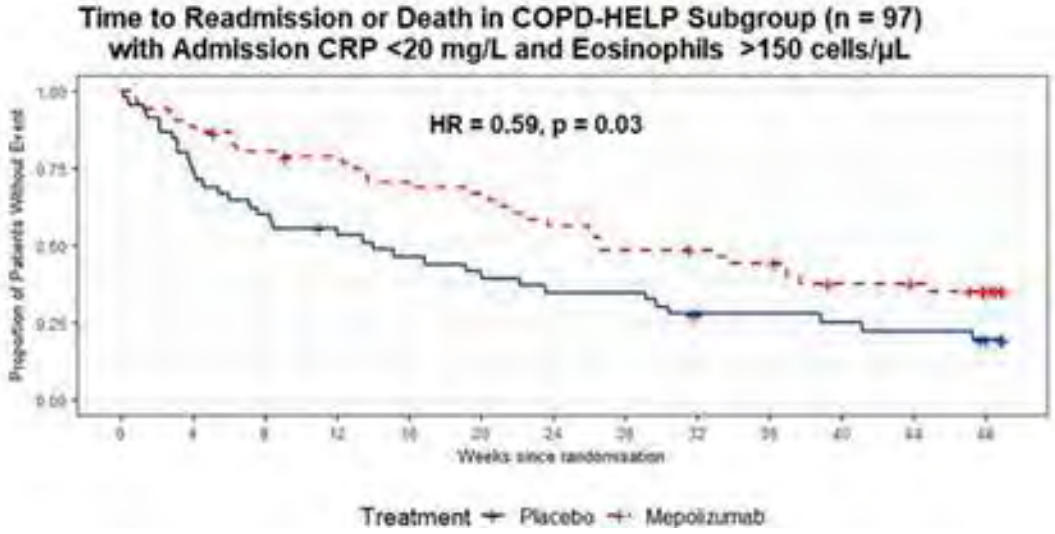
P271 CRP MODIFIES RESPONSE TO MEPOLIZUMAB FOLLOWING HOSPITALISATION FOR COPD EXACERBATION: POST-HOC ANALYSIS OF COPD-HELP

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Background Mepolizumab reduces exacerbations in eosinophilic COPD but did not improve time to readmission or death following hospitalisation in COPD-HELP (Flynn et al., NEJM Evidence 2025). We hypothesised that admission biomarkers and functional status modify treatment effect in this high-risk population.

Methods We performed post-hoc analyses of COPD-HELP, a double-blind, placebo-controlled randomised trial of 238 patients hospitalised with AECOPD and evidence of an eosinophilic phenotype (blood eosinophil count ≥300 cells/μL at least once in the prior year), randomised to mepolizumab 100 mg or placebo for 48 weeks. All participants had MRC dyspnoea score ≥2, were established on inhaled corticosteroids, and had a significant smoking history. The primary outcome was time to rehospitalisation or death. Cox models assessed interactions



Abstract P271 Figure 1

between treatment, admission eosinophils, MRC dyspnoea score, and C-reactive protein (CRP).

Results Previously reported findings for eosinophils and functional limitation were confirmed. Admission eosinophils ≥300 cells/μL showed a signal for greater mepolizumab benefit (treatment×eosinophil interaction HR 0.53; p=0.053), strengthened after excluding patients with severe functional limitation (MRC 5) (HR 0.41; p=0.019).

CRP demonstrated stronger and independent effect modification. Increasing CRP was associated with progressive attenuation of treatment benefit (treatment×CRP interaction HR 1.30; p=0.026). At lower CRP, outcomes favoured mepolizumab; with increasing CRP the treatment effect diminished, with outcomes converging and at higher levels appearing to favour placebo. Incorporating CRP with a clinically relevant eosinophil threshold (>150 cells/μL) identified a subgroup more likely to benefit, with reduced risk (HR 0.59; p=0.03 [Fig1]), and indicated that higher eosinophil levels mitigated the attenuation of treatment effect seen with increasing CRP.

Conclusions CRP was a key and independent modifier of treatment response. While eosinophil and functional findings were consistent with previous analyses, incorporating CRP alongside an established eosinophilic phenotype refined identification of patients most likely to benefit. These findings may help explain heterogeneous trial outcomes and identify patients in whom initiation of treatment in the acute phase is appropriate, while suggesting that those with higher inflammatory burden may be better reassessed once acute illness has resolved.

P272 BIOLOGIC RESPONSE IN SEVERE ASTHMA PATIENTS WITH OVERLAPPING ASTHMA AND COPD FEATURES: A REAL-WORLD COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.432

Introduction Recent licensing of biologic therapies for eosinophilic COPD has further blurred the distinction between severe asthma, asthma-COPD overlap (ACO) and type 2 (T2)-high COPD. We examined whether smoking history, COPD overlap or baseline T2 biomarkers were associated with response to biologic therapy in a real-world severe asthma cohort.

Methods We performed a retrospective cohort study of patients receiving biologic therapy for severe asthma at a tertiary respiratory centre.

Patients were classified as having ACO if they had a smoking history of >10 pack years together with objective evidence of COPD, including emphysema on CT and/or impaired gas transfer (Kco ≤ -1.64 standardised residuals). Baseline smoking history, fractional exhaled nitric oxide (FeNO) and blood eosinophil count were recorded.

Treatment response was assessed 12 months after biologic initiation. The primary outcome was achievement of oral corticosteroid (OCS)-free status.

Associations between OCS-free status and ACO, smoking history ≥10 pack years, FeNO ≥50 ppb and eosinophil count above the cohort median were assessed using Fisher's exact test.

Abstract P272 Table 1 Association between clinical phenotype, smoking history and type 2 inflammatory biomarkers with achievement of oral corticosteroid (OCS)-free status 12 months following biologic initiation. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated using Fisher's exact test. Asthma-COPD overlap (ACO) was defined as a smoking history ≥10 pack years together with objective evidence of COPD (emphysema on CT and/or impaired gas transfer). High FeNO was defined as ≥50 ppb. High eosinophil burden was defined as a blood eosinophil count above the cohort median. An OR >1 indicates increased odds of achieving OCS-free status

Predictor	OR (95% CI)
Asthma-COPD overlap	0.80 (0.26–2.27)
Smoking ≥10 pack years	1.26 (0.50–3.42)
FeNO ≥50 ppb	1.01 (0.41–2.68)
Eosinophils > median	1.23 (0.51–2.84)

Advances in translational research

Results 81 patients were included. At 12 months, 49/81 (60.5%) achieved OCS-free status.

16/81 met criteria for ACO. OCS-free status was achieved in 56.3% (9/16) of patients with ACO and 61.5% (40/65) without ACO (OR 0.80, 95% CI 0.26-2.27; p=0.78).

Similarly, no significant associations were observed between OCS-free status and smoking history ≥10 pack years (OR 1.26, 95% CI 0.50-3.42; p=0.80), FeNO ≥50 ppb (OR 1.01, 95% CI 0.41-2.68; p>0.99), or eosinophil count above the cohort median (OR 1.23, 95% CI 0.51-2.84; p=0.82).

Conclusions In this real-world severe asthma cohort, biologic-associated steroid reduction was observed across a broad spectrum of asthma and COPD phenotypes. Neither ACO, smoking history nor baseline T2 biomarker burden were significantly associated with achievement of OCS-free status at 12 months.

These findings suggest that patients selected for biologic therapy may derive similar benefit irrespective of overlapping COPD features or smoking history.

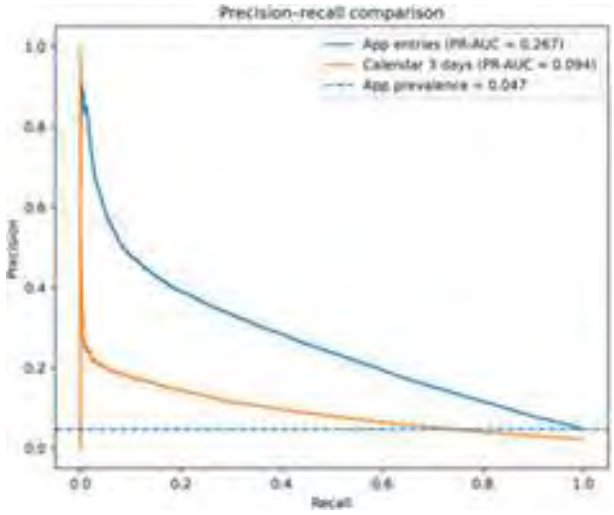
Advances in translational research

P273 COMPARING APP-ENTRY AND CALENDAR-DAY PREDICTION OF COPD EXACERBATIONS USING REAL-WORLD MYCOPD DATA

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10.1136/thorax-2026-BTSabstracts.433

Background Digital self-management platforms may help identify people with COPD at increased risk of exacerbation. However, patients do not always enter app data daily, making it unclear how best to define a clinically meaningful prediction



Abstract P273 Figure 1

window. A calendar window asks whether an exacerbation will occur within the next few days, whereas an app-entry window asks whether it will occur by the next few times the patient uses the app. We compared these approaches using myCOPD data.

Methods We analysed pseudonymised longitudinal data from myCOPD. Users with at least one exacerbation matching our operational label were included. Using a common cohort, we developed matched XGBoost models with the same app-derived predictors: current symptoms, recent symptom change, CAT-derived variables, prior exacerbation history, medication-use features and engagement-related variables. Two short-term prediction tasks were compared: exacerbation by the next 1–3 app entries, and exacerbation within the next 3 calendar days. Models were evaluated using grouped nested cross-validation at patient level. Performance was reported using sensitivity, positive predictive value (PPV), precision-recall AUC (PR-AUC) and ROC-AUC.

Results The common cohort included 3,732 users. The app-entry task generated 545,897 labelled prediction instances, each corresponding to an observed app entry, with exacerbation prevalence 4.7%. The 3-day calendar task generated 494,536 labelled instances, with prevalence 2.2%. The app-entry model performed better than the calendar-day model. Mean PR-AUC was 0.269 versus 0.096, and mean ROC-AUC was 0.828 versus 0.769. At the selected operating point, PPV was 28.4% versus 14.5%, and sensitivity was 39.9% versus 20.4%, respectively. In the app-entry approach, the median time to the next, second-next and third-next app entries was 1, 2 and 3 days, indicating that upcoming app entries often represented a near-term clinical horizon while retaining more analysable observations and exacerbation events.

Conclusions In real-world COPD app data, the prediction window influenced model performance and clinical interpretation. Predicting risk by upcoming app entries gave higher PPV and sensitivity than a fixed 3-day calendar window, while still representing a short-term warning period for many users. App-entry-based prediction may therefore be a practical way to frame digital COPD exacerbation early-warning systems when patient-reported data are entered intermittently.

P274 COMBINING PLEURAL FLUID PROTEOMICS AND MICROBIOLOGY DATA TO IDENTIFY PLEURAL INFECTION BIOLOGICAL ENDOTYPES

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10.1136/thorax-2026-BTSabstracts.434

Background Pleural infection is a major cause of morbidity and mortality. Heterogeneity exists in both microbial aetiology and host inflammatory responses. However, the association between microbiology and immune responses remains unclear.

Aim To investigate the association between microbiology and host immune response through integrated multi-omics analysis.

Methods We integrated pleural fluid proteomics and molecular microbiology (16S rRNA, n=80) with baseline clinical data and outcomes using the Multi-Omics Factor Analysis pipeline.

Results We identified 15 distinct latent factors combining microbial composition and host immune pathways. The majority of explained variance originated from the proteomic layer (approximately 65%), whereas the microbiome contributed a smaller but distinct proportion of variance (approximately 25%). Factor 5 demonstrated the strongest association with mortality (p=0.005), with higher Factor 5 values observed in the one-year mortality group.Factor 5 was not associated with RAPID score, suggesting that it captures prognostically relevant biological information beyond conventional clinical risk stratification. Moreover, proteins including POSTN, FIBG and CO3A1, together with Streptococcus pneumoniae and Enterobacteriaceae had a positive weight,while C1R, C1S, SAA1-related proteins, Streptococcus anginosus group and anaerobic organisms contributed negatively.

Conclusion Integrated multi-omics analysis identified host–pathogen factors that capture biologically meaningful heterogeneity in pleural infection. We identified a factor associated with mortality but not the RAPID score. This provides a framework for molecular endotyping of pleural infection,may enable improved risk stratification, and needs further validation.

P275 PRIOR EXACERBATION HISTORY SHAPES SHORT-TERM PREDICTION OF COPD EXACERBATIONS IN REAL-WORLD MYCOPD DATA

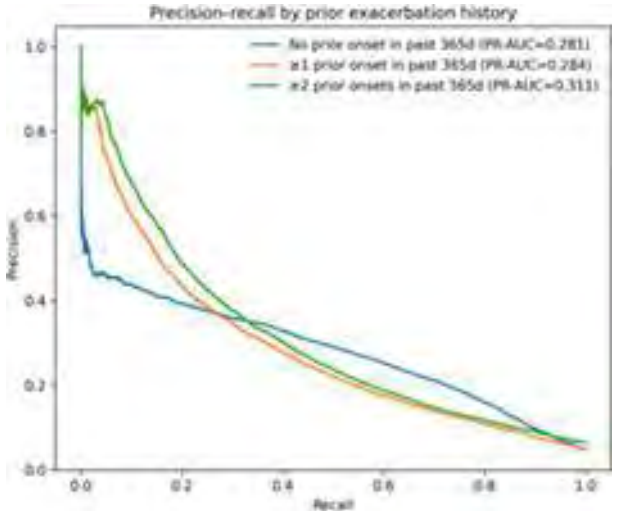
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10.1136/thorax-2026-BTSabstracts.435

Background Digital self-management platforms may help identify people with COPD at short-term risk of exacerbation. This may be useful after recent exacerbations, when patients may enter a period of repeated risk. We examined whether a digital prediction model performed differently during recurrent-risk periods, defined as app entries made after one or more exacerbation onsets in the previous year.

Methods We analysed pseudonymised longitudinal myCOPD data. Users with at least one symptom-defined exacerbation were included. We used a pragmatic label from the myCOPD self-management plan: an exacerbation onset was an app entry in the Moderate Deterioration or Severe Deterioration categories, with separate onsets at least 14 days apart. In 4,223 users, we trained an XGBoost model to predict whether an exacerbation would occur by the next 1–3 app entries. Predictors included symptoms, CAT-derived variables, medication-use, app-engagement and exacerbation-history features. Models were evaluated using grouped nested cross-validation by patient. Held-out predictions were stratified by prior exacerbation onsets in the previous 365 days: none, ≥1 and ≥2. Performance was reported using positive predictive value (PPV), sensitivity, precision-recall AUC (PR-AUC) and ROC-AUC.

Results The dataset contained 547,465 labelled app-entry observations; overall prevalence was 4.84%. Observations with no prior onset had prevalence 4.71%, mean PR-AUC 0.281, PPV 29.1% and sensitivity 50.0%; among positive observations, median time to the first future exacerbation-defining entry was 8 days [IQR 3–35]. Observations with ≥1 prior onset had prevalence 4.93%, mean PR-AUC 0.283, PPV 32.5% and sensitivity 32.0%, with median time to event 4 days [2–16]. Observations with ≥2 prior onsets had prevalence 6.25%, mean PR-AUC 0.308, PPV 33.8% and sensitivity 33.7%, with median time to event 4 days [2–15]. Pooled PR-AUC increased from 0.281 with no prior onset to 0.311 with ≥2 prior onsets.



Abstract P275 Figure 1

Conclusions Recent exacerbation history changed the clinical context and performance of short-term digital prediction. App entries after previous exacerbations were more likely to be followed by another near-term exacerbation, and positive predictions had higher PPV. Although sensitivity was lower, prior exacerbation history may help clinicians judge when early-warning alerts are more likely to reflect actionable near-term risk.

P276 PLATELET FUNCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) PATIENTS, WITH AND WITHOUT CARDIOVASCULAR CO-MORBIDITIES

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Background Chronic obstructive pulmonary disease (COPD) is characterised by airway inflammation. Current management primarily relies on inhaled therapies which undertreats systemic inflammation, potentially contributing to extrapulmonary comorbidities such as cardiovascular disease (CVD). Platelet activation in models of airway inflammation has been well-established, with evidence of platelet-dependent leukocyte recruitment to the airways. Elevated platelet counts in COPD patients further implicate their involvement in disease pathogenesis; however, there is a paucity of studies investigating their haemostatic and inflammatory functional activities in COPD.

Aim To assess platelet functional activities in COPD patients ± CVD in comparison to healthy individuals and determine whether there are differences in haemostatic and inflammatory platelet function.

Methods Blood from healthy donors (n=10) or COPD patients (n=15) was collected and used to assess platelet aggregation. Platelet-rich plasma was isolated from the blood and stimulated with a range of adenosine diphosphate (ADP) concentrations (1-100uM) to induce aggregation, measured via absorbance readouts. Plasma soluble P-selectin (sP-selectin) and platelet factor 4 (PF4) levels were measured by ELISA in healthy donors (n=9) and COPD patients (n=13).

Results A clear dose-response to ADP was observed in all groups. Platelet aggregation significantly reduced at high ADP concentrations in COPD patients taking the anti-platelet drug, clopidogrel. A trend toward reduced aggregation of ~1.3-fold was also observed in COPD patients not taking clopidogrel, in comparison to healthy controls. Platelet aggregation was slightly reduced in COPD patients with CVD (~1.8-fold). Plasma sP-selectin and PF4 levels were slightly elevated in COPD patients but this was not statistically significant.

Conclusions The overall reduction in platelet aggregation in COPD patients, and further reduction in patients with CVD, suggests that platelets have reduced haemostatic function in COPD. Therefore, platelet hyperaggregability may not be the reason for CVD comorbidities in COPD. Elevated sP-selectin and PF4 indicates potentially overactive platelets in COPD, measuring these degranulation markers in exacerbating COPD patients may provide further insight. Future studies will involve assessing platelet chemotaxis to allow for comparisons between haemostatic and inflammatory platelet function in COPD.

P277 A MULTIVARIATE SPUTUM PROTEOMIC SIGNATURE ASSOCIATED WITH AIRFLOW LIMITATION IN COPD: BASELINE FINDINGS FROM THE PHASE 2A, RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED COPD-ST2OP TRIAL

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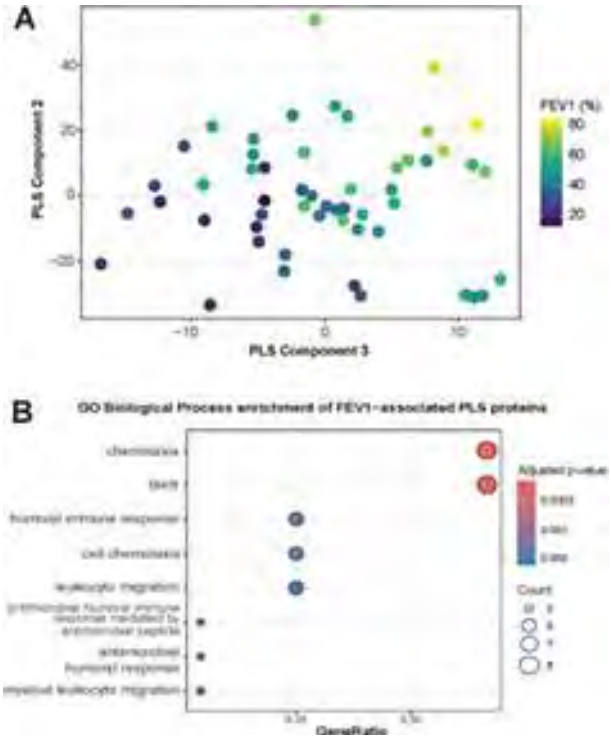
10.1136/thorax-2026-BTSabstracts.437

Background Chronic obstructive pulmonary disease (COPD) is characterised by biological heterogeneity. Sputum proteomics may identify molecular signatures associated with disease severity and underlying inflammatory mechanisms.

Methods Samples were obtained from the COPD-ST2OP trial.[1] 53 baseline sputum samples were profiled using Olink Explore platform, generating 1,472 proteins after quality control. Associations between protein abundance and forced expiratory volume in 1 second (FEV1, %predicted) were initially explored using protein-wise correlation analysis. Partial least squares (PLS) regression with leave-one-out cross-validation was then used to identify multivariate proteomic signatures associated with lung function. FEV1-associated PLS component scores, protein loadings, and exploratory Gene Ontology Biological Process (GOBP) enrichment analysis were used to characterise the dominant proteomic signal and performed on the top 25 PLS-associated proteins (24 successfully mapped to Entrez Gene identifiers) using over-representation testing with Benjamini–Hochberg correction.

Results PLS modelling identified strong multivariate proteomic structure for FEV1. Leave-one-out cross-validation selected a five-component model based on the lowest root mean square error of prediction (17.1). Component-level correlation analysis demonstrated that higher scores on PLS component 3 were associated with higher FEV1 ($\rho=0.62$; $p<0.001$), followed by component 2 ($\rho=0.49$; $p<0.001$), component 4 ($\rho=0.39$; $p=0.003$) and component 1 ($\rho=0.32$; $p=0.019$). Visualisation of the latent score space revealed a graded distribution of FEV1 values across the dominant PLS components (figure 1A). Enrichment analysis identified that chemotaxis (8/24 proteins), taxis (8/24), antimicrobial humoral immune response (5/24), humoral immune response (6/24), cell chemotaxis (6/24), and leukocyte migration (6/24) represented the strongest enriched pathways (all $p_{adj}<0.01$; figure 1B).

Conclusion Baseline sputum proteomics identified a multivariate inflammatory signature associated with airflow limitation in COPD. Although individual protein-level associations were modest, PLS modelling detected a reproducible latent proteomic structure associated with FEV1. The identified proteomic axis was characterised by inflammatory cell trafficking and humoral immune response biology, including enrichment of chemotaxis and leukocyte migration pathways. These findings support further evaluation of sputum-derived proteomic programmes as markers of airway disease severity and biological heterogeneity in COPD.



Abstract P277 Figure 1

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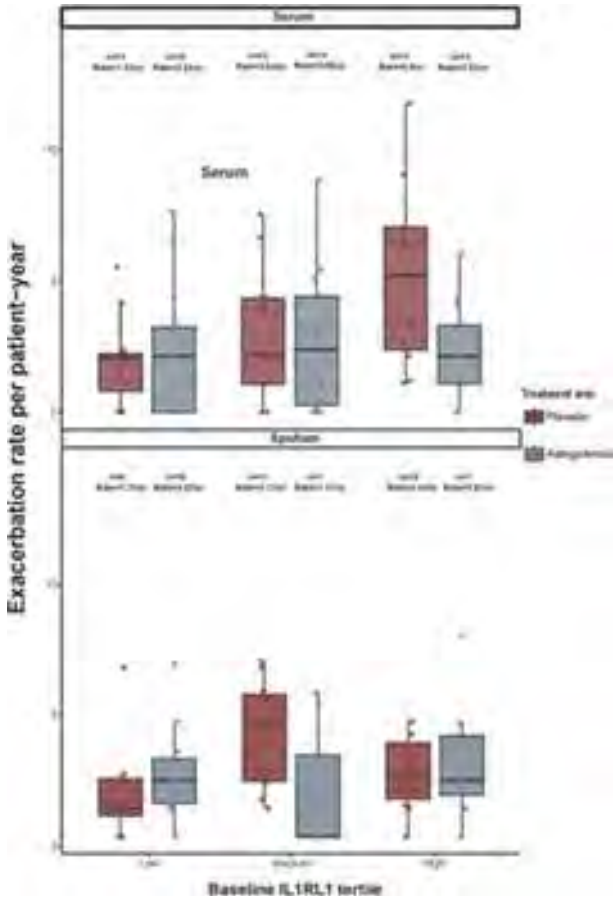
P278 BASELINE SERUM IL1RL1 IDENTIFIES A COPD SUBGROUP WITH INCREASED EXACERBATION BURDEN AND REDUCED EXACERBATION RATES FOLLOWING ASTEGOLIMAB: A POST-HOC ANALYSIS OF THE PHASE 2A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED COPD-ST2OP TRIAL

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Background Acute exacerbations of COPD contribute to disease progression and mortality. IL1RL1 is the gene that encodes ST2, which is a key component of the IL-33 signalling pathway and is the therapeutic target of Astegolimab. Whether baseline IL1RL1 identifies patients with greater exacerbation or differential response to Astegolimab remains unclear. Here we investigated associations between baseline IL1RL1 concentrations, subsequent exacerbation burden, and treatment-specific outcomes in participants enrolled in the COPD-ST2OP trial.[1]

Methods Baseline serum (n=80) and sputum (n=53) samples were analysed using the Olink Explore platform. IL1RL1 concentrations were categorised into tertiles (low/medium/high). Exacerbation rates from treatment initiation to final study visit were evaluated using negative binomial regression with follow-up duration included as an offset. Models were adjusted for age and smoking status, and included treatment-by-tertile interactions to compare Astegolimab and placebo within IL1RL1 strata.



Abstract P278 Figure 1

Results Placebo-treated participants demonstrated a marked serum IL1RL1 gradient, with crude exacerbation rates increasing from 1.93 to 4.60 exacerbations per patient-year across low to high tertiles. Participants in the highest serum IL1RL1 tertile experienced significantly higher exacerbation rates than those in the lowest tertile within the placebo arm (RR 2.78, 95%CI 1.25-6.19; p=0.009), with evidence of a positive trend across tertiles (RR per tertile increase 1.67, 95%CI 1.18-2.36; p=0.004). In contrast, exacerbation rates remained relatively stable across serum IL1RL1 tertiles in Astegolimab-treated participants (2.24, 2.66 and 2.52 exacerbations per patient-year), and no corresponding tertile gradient was observed. Within the highest serum IL1RL1 tertile, Astegolimab was associated with a 50% lower exacerbation rate compared with placebo (RR 0.50, 95%CI 0.26-0.94; p=0.032). Sputum IL1RL1 demonstrated no consistent association with exacerbation burden and did not identify a subgroup with reduced exacerbation rates following treatment (figure 1).

Conclusion Baseline serum IL1RL1 identified a subgroup characterised by higher exacerbation burden under placebo and lower exacerbation rates following Astegolimab treatment. These exploratory findings suggest that circulating IL1RL1 may capture clinically relevant IL-33/ST2 pathway activity and warrant further investigation as a biomarker for patient enrichment in future studies of IL-33/ST2 blockade.

REFERENCE

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P279 MAPPING THE PRODROME PHASE OF RHINOVIRUS-INDUCED CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) EXACERBATION

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Background The biological events preceding symptom onset and influencing severity of acute exacerbations of COPD are poorly defined. Experimental rhinovirus (RV) challenge enables serial sampling from infection through exacerbation and recovery and may help define the 'prodrome' phase, from onset of upper respiratory cold symptoms to onset of lower respiratory exacerbation, and identify biomarkers relevant to early intervention and severity prediction.

Methods 22 participants with GOLD stage 2 COPD underwent RV inoculation and serial sampling from baseline. Participants recorded daily upper (URSS) and lower (LRSS) respiratory symptom scores. Blood, sputum and bronchoscopic samples from the prodrome phase were analysed for viral load, cell counts, and inflammatory mediators.

Results 21/22 participants met criteria for clinical cold and 20/22 met exacerbation criteria. Mean URSS rose above baseline from day 2 post-inoculation and peaked at day 4, whereas mean LRSS peaked at day 10 and returned to baseline at day 21, indicating a distinct prodromal upper respiratory phase from days 2 to 10 preceding exacerbation.

Exacerbation severity, represented by either peak LRSS or LRSS area-under-the-curve to day 21 (LRSS AUC), was compared with biomarkers observed during the prodrome phase. Peak LRSS correlated with prodrome peak URSS (measured days 2-9; rho=0.550, p=0.008), peak serum CXCL10 (days 3-5; rho=0.496, p=0.018), sputum neutrophils (day 9; rho=0.450, p=0.025), peak sputum virus load (days 5-9; rho=0.448, p=0.036) and peak nasal lavage virus load (days 3-7, 9; rho=0.432, p=0.045). LRSS AUC was correlated with prodrome peak FeNO (days 3-5; rho=0.486, p=0.021), serum CRP (day 7; rho=0.478, p=0.028), peak URSS (days 2-9; rho=0.458, p=0.032) and peak serum CXCL10 (days 3-5; rho=0.436, p=0.042).

Participants with higher-than-median exacerbation severity had greater prodrome peak FeNO (50 vs 19ppb, p=0.002), serum IL-6 (5.1 vs 2.5pg/mL, p=0.028), and sputum viral load (3.25 vs 2.45 log10 copies/μL, p=0.029).

Conclusions Symptomatic upper respiratory infection appears to be a reliable warning phase for RV-induced AECOPD in GOLD stage 2 COPD. During this pre-exacerbation phase, viral load is high and relates to subsequent exacerbation severity. Greater severity is associated with impaired viral control and increased inflammation, identifying candidate biomarkers relevant to the earliest stages of RV-induced AECOPD.

P280 EOSINOPHIL DEPLETION UNMASKS DISTINCT CYTOKINE PROFILES IN EGPA AND SEVERE EOSINOPHILIC ASTHMA

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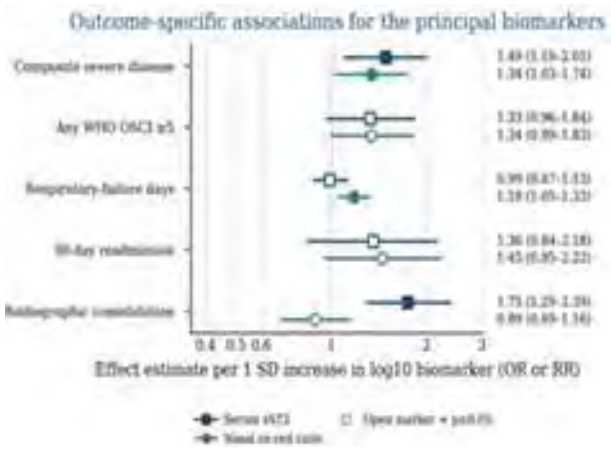
Background The extent of immune overlap and diversity between eosinophilic granulomatosis with polyangiitis (EGPA) and severe eosinophilic asthma (SEA) remains unclear. The anti-IL5R biologic benralizumab offers the first opportunity to examine this in the absence of eosinophils or the confounding effects of oral corticosteroids (OCS).
Methods Plasma cytokines from patients with EGPA (n=12) and SEA (n=13) off OCS on benralizumab was analysed using the Meso Scale Discovery platform. Unsupervised principal component analysis (PCA) and hierarchical clustering assessed global immune profile separation.
Results In the absence of OCS or other immunosuppressants, EGPA and SEA demonstrated distinct circulating cytokine profiles, with PCA and hierarchical clustering showing clear separation between groups. Despite clinical stability, EGPA was characterised by a broad multi-axis inflammatory signature including elevated T2 cytokines (IL-4, IL-13), myeloid mediators (TNF-α, IL-6, IL-8, IL-18, G-CSF), chemokines associated with leukocyte recruitment (MCP-1, MIP-1β, MIP-3α, fractalkine), and interferon signalling (IFN-γ, IFN-β). SEA demonstrated a comparatively restricted profile enriched for epithelial-derived mediators including IL-33, TSLP and IL-25 (figure 1).
Conclusions In the absence of eosinophils and confounding by broad immunosuppression, distinct inflammatory signatures persist in EGPA and SEA, reflecting a mixed T2/pro-inflammatory profile in EGPA and an epithelial-alarm signature in SEA. That EGPA retains this broad cytokine programme despite clinical remission suggests disease-specific immune activity independent of eosinophilia, with potential implications for monitoring and treatment stratification. Analysis is ongoing to assess for any association with blood immune cell phenotypes by mass cytometry.

P281 ADMISSION IL-33/ST2 PROFILING IDENTIFIES COMPARTMENT-SPECIFIC SEVERITY SIGNALS IN ADULTS HOSPITALISED WITH RESPIRATORY VIRAL INFECTIONS

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Introduction and Objectives Severe adult respiratory viral infection (RVI) is heterogeneous, and host-response remains poorly characterised. Interleukin-33 (IL-33) is an epithelial alarmin released after airway injury and modified extracellularly into oxidised/reduced forms. Serum soluble ST2 (sST2) reflects tissue-stress signalling. We tested whether admission serum sST2 and nasal oxidised:reduced IL-33 ratio were independently associated with severe disease.



Abstract P281 Figure 1 Outcome-specific associations of serum sST2 and nasal oxidised:reduced IL-33 ratio with disease severity. Adjusted forest plot showing associations between the two principal IL-33/ST2 biomarkers and clinical outcomes in adults hospitalised with respiratory viral infection. Effects are shown per 1 standard deviation increase in log10-transformed biomarker values; estimates are ORs for binary outcomes and rate ratios for respiratory-failure days. Both serum soluble ST2 (sST2) and nasal oxidised:reduced interleukin-33 (IL-33) ratio were associated with composite severe disease, supporting non-redundant systemic and upper-airway severity signals. Their secondary outcome profiles differed: sST2 was most strongly associated with radiographic consolidation, whereas the nasal oxidised:reduced IL-33 ratio was most clearly associated with respiratory- failure duration. Associations with any WHO OSCI ≥5 and 30-day readmission were weaker. Filled markers indicate p<0.05 and open markers p≥0.05; error bars show 95% CIs

Methods We analysed a nested biomarker subcohort from a prospective multicentre UK observational study of adults hospitalised with PCR-confirmed single-virus RVI and paired day 1 serum/nasal lining fluid. Viral groups were SARS-CoV-2 (n=95), influenza (n=57), rhinovirus/enterovirus (n=54), respiratory syncytial virus (RSV; n=44) and other viruses (n=32). Serum sST2, nasal oxidised IL-33 and nasal reduced IL-33 were measured; the oxidised:reduced ratio was derived. Biomarkers were log10-transformed and standardised. Composite severe disease was defined as WHO Ordinal Scale for Clinical Improvement (OSCI) ≥6, intensive care/high dependency unit admission, death within 60 days, or ≥4 hospital days with OSCI ≥5. In hospitalised adults, OSCI 4 denotes admission without oxygen, OSCI 5 oxygen therapy, OSCI 6–9 escalating ventilatory/organ support and OSCI 10 death. Between-virus differences were assessed using Kruskal–Wallis tests. Associations with severe disease were examined using multivariable logistic regression with biomarkers entered jointly, adjusted for age, sex, viral group, smoking status, COPD, asthma, diabetes and heart failure.
Results Among 282 adults, 95 (33.7%) met the endpoint, 209 (74.1%) reached OSCI ≥5 and 88 (31.2%) developed prolonged respiratory failure. Serum sST2 varied by viral group (p<0.001), highest in RSV and SARS-CoV-2 and lowest in rhinovirus/enterovirus; nasal oxidised:reduced IL-33 ratio did not vary by viral group (p=0.510).

After adjustment, serum sST2 was independently associated with composite severe disease: adjusted OR 1.49 per 1 standard deviation increase in log10-transformed biomarker (95% CI 1.10–2.01; p=0.011).

Nasal oxidised:reduced IL-33 ratio was also independently associated with composite severe disease: adjusted OR 1.37 per 1 standard deviation increase (95% CI 1.04–1.79; p=0.023).

Conclusions Serum sST2 and nasal oxidised:reduced IL-33 ratio identified independent systemic and upper-airway severity signals across hospitalised RVI, supporting paired IL-33/ST2 profiling for biologically grounded severity stratification across respiratory viruses.

P282 BEYOND THE EOSINOPHIL: BENRALIZUMAB DRIVES AIRWAY-SPECIFIC T CELL IMMUNE REMODELLING IN SEVERE EOSINOPHILIC ASTHMA

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Background The anti-IL5R biologic benralizumab depletes eosinophils. Whether additional immunological mechanisms including an impact on adaptive immunity underlie the clinical benefit of benralizumab remains poorly understood. We investigated CD4+ T cell remodelling following benralizumab initiation in adults with severe eosinophilic asthma (SEA).

Methods Adults with SEA underwent paired blood (n=11) and BAL (n=8) sampling before and after four months of benralizumab. CyTOF was used to classify CD4+ T cells into five functional subsets: Th1, Th2, Th17, Th1/Th17 and Th2/Th17 using compartment (blood/BAL)-specific chemokine receptor thresholds. Linear mixed models, differential state analysis and spearman correlation identified treatment-associated changes.

Results Despite no significant change in CD4+ T cell abundance in either compartment, benralizumab induced profound phenotypic changes exclusively in the airway. Regulatory T cells (T-reg) in BAL underwent coordinated phenotypic shift, downregulating TSLP-driven type 2-permissive characteristics (TSLPR, IL-5, IL-13) and upregulating canonical suppressive markers (TGF-β, CXCR3, CCR6) consistent with a switch from IL-10-dominant innate suppression to TGF-β-dominant adaptive suppression potentially targeting residual Th17 inflammation. Blood T-reg abundance increased post-treatment (p=0.019), suggesting systemic T-reg mobilisation without airway recruitment. CXCR3 expression increased across all six BAL memory CD4+ T cell clusters (all p<0.01) in the absence of IFN-γ upregulation, consistent with resolution-phase remodelling. Changes in Th subset composition tracked clinical outcomes: Th2/Th17 reduction correlated with FeNO improvement (rho=0.76, p=0.028) and Th17 reduction with FEV1 improvement (rho=−0.74, p=0.037). No phenotypic changes were observed in blood.

Conclusions Benralizumab induces coordinated airway-specific CD4+ T cell phenotypic shifting characterised by T-reg functional restoration and chemokine receptor reprogramming consistent with immune resolution. The compartment specificity of these findings and their association with clinical benefit suggest active immune remodelling rather than passive resolution following eosinophil depletion.

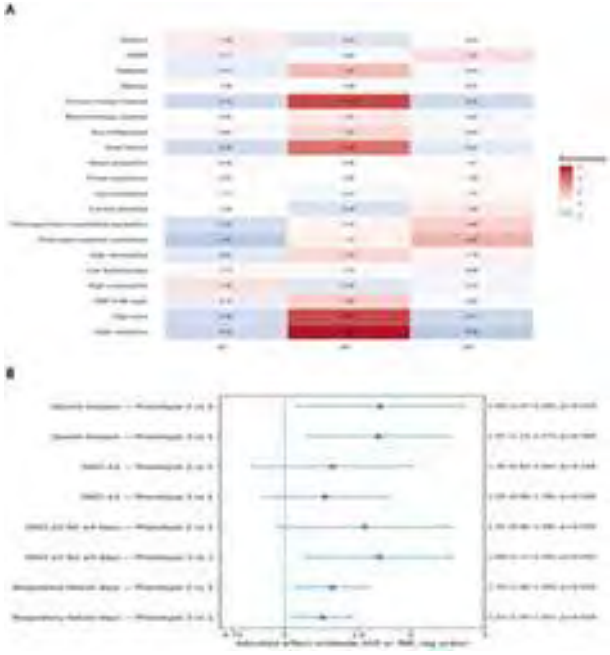
P283 HOST-DERIVED CLINICAL PHENOTYPES DELINEATE DISTINCT SEVERITY TRAJECTORIES IN ADULT RESPIRATORY VIRAL HOSPITALISATION

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10.1136/thorax-2026-BTSabstracts.443

Introduction and Objectives Adults hospitalised with respiratory viral infection (RVI) have heterogeneous outcomes within and across viral groups. We tested whether admission host phenotypes, defined by comorbidity, symptom-profile and laboratory patterns, could identify severe-disease trajectories irrespective of viral aetiology.

Methods 970 Adults in a prospective multicentre UK cohort with PCR-confirmed single-virus RVI were analysed: influenza (n=260), SARS-CoV-2 (n=252), rhinovirus/enterovirus



Abstract P283 Figure 1 Host phenotype structure and adjusted clinical outcome associations in adult respiratory viral hospitalisation. Panel A shows phenotype-defining enrichment across the three latent hostphenotypes. Enrichment was defined as the prevalence of each feature within a phenotype divided by the prevalence of that feature in the complete-case phenotype-derivation cohort. Symptom duration refers to duration before hospital admission; short duration was ≤4 days and prolonged duration was ≥5 days. Phenotype 1 was an asthma-eosinophilic/short-duration phenotype, enriched for asthma, eosinophilia and short lower-respiratory/systemic symptom duration. Phenotype 2 was a multimorbid cardiorenal-inflammatory phenotype, enriched for chronic kidney disease, heart failure, diabetes, raised urea, raised creatinine, high C-reactive protein and neutrophilia. Phenotype 3 was a chronic obstructive pulmonary disease/prolonged respiratory-systemic phenotype, enriched for chronic obstructive pulmonary disease, current smoking, and prolonged lower-respiratory and systemic symptoms. Panel B shows adjusted associations between phenotype membership and clinical outcomes, with phenotype 1 as the reference group. Models were adjusted for age, sex and viral group. Points show adjusted ORs or incidence rate ratios; error bars show 95% CIs

(n=221), respiratory syncytial virus (RSV; n=115) and other viruses (n=122). Latent class analysis used comorbidities, smoking, lower-respiratory/systemic symptom-duration categories, defined as short (≤4 days) or prolonged (≥5 days), and categorised admission blood tests: neutrophils >10×10^9/L, lymphocytes <1×10^9/L, eosinophils ≥0.3×10^9/L, CRP ≥100 mg/L, urea >7.8 mmol/L and creatinine >110 μmol/L. Two- to six-class solutions were compared using Bayesian information criterion (BIC). Feature enrichment (E) was calculated as feature prevalence within phenotype divided by prevalence in the complete-case derivation cohort. Composite severe disease was defined as WHO Ordinal Scale for Clinical Improvement (OSCI) ≥6, death within 60 days, or ≥4 hospital days with OSCI ≥5. OSCI 4 denotes hospitalisation without oxygen, OSCI 5 oxygen therapy, OSCI 6–9 escalating ventilatory/organ support and OSCI 10 death. Multivariable logistic regression models included adjusted for age, sex, viral group and host phenotype.

Results The three-class solution had the lowest BIC. Phenotype 1, asthma–eosinophilic/short-duration (n=346, 42.4%), was enriched for asthma (E=1.32), eosinophilia (E=1.42) and short lower-respiratory/systemic symptom duration. Phenotype 2, multimorbid cardiorenal–inflammatory (n=179, 21.9%), was enriched for raised creatinine (E=4.08), raised urea (E=3.54), chronic kidney disease (E=3.53), heart failure (E=2.96), diabetes (E=1.93), high CRP (E=1.68) and neutrophilia (E=1.53). Phenotype 3, COPD/prolonged respiratory–systemic (n=291, 35.7%), was enriched for prolonged systemic symptoms (E=2.06), prolonged lower-respiratory symptoms (E=1.64), COPD (E=1.43) and current smoking (E=1.30). All phenotypes crossed viral groups. Severe disease occurred in 202/815 (24.8%): 18.2%, 33.0% and 27.6% in phenotypes 1–3. Compared with phenotype 1, phenotypes 2 and 3 were independently associated with severe disease: adjusted OR 1.69 (95% CI 1.07–2.66; p=0.025) and 1.67 (1.14–2.47; p=0.009). Phenotype 2 was associated with radiographic consolidation; phenotype 3 with prolonged respiratory failure.

Conclusions Admission host phenotypes separated severe adult RVIs into distinct clinical trajectories.

P284 INTRAPLEURAL ENZYMATIC THERAPY MODULATES NEUTROPHIL-MEDIATED INFLAMMATORY RESPONSES - THE IFFLEY STUDY

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Background Pleural infection is a severe and complicated condition with increasing worldwide incidence. Although intrapleural enzymatic therapy (IET), in which tPA and Dnase are commonly used, has shown promising clinical results, its mechanism of action remains poorly understood. Our recently published study showed patients with pleural infection exhibited elevated level of neutrophil extracellular traps (NETs) in their pleural cavity, and pleural fluid NETs are associated with disease severity. Moreover, neutrophil elastase (ELANE) is suggested to promote intrapleural fibrinolytic deficiency by degrading plasminogen (PLG).

Aim To elucidate the biological effect of IET within the pleura of pleural infection patients, we compared pleural fluid and plasma specimens prospectively collected at baseline and post-IET and investigated the effect of IET on NETs, neutrophil granule proteins and intrapleural fibrinolysis.

Results We demonstrated that the increase in the level of soluble fibrin and D-dimer, a degradation product of fibrin, following IET comes from IET-mediated cleavage of fibrinous septations in the pleura of pleural insfection patients (n=20). Although IET reduced the level of pleural fluid PAI-1 (~46% reduction, p=0.0003, n=20), no significant associations were found between PAI-1 with soluble fibrin and D-dimer. IET reduced the level of pleural fluid NETs in pleural infection patients (~50% reduction, p=0.0008, n=20), and moreover the protein level (~39% reduction, p=0.004, n=20) and activity of ELANE (~37% reduction, p=0.0016, n=20). Pearson correlation analysis of baseline pleural fluid samples from multiple independent cohorts showed a negative association between the protein level of neutrophil elastase and plasminogen (Oxford Osler cohort: r= -0.46, p= 0.03, n=20; validation cohort: r= -0.57, p<0.0001, n=73). Pharmacological inhibition of ELANE reduced plasminogen degradation (~24% reduction, p= 0.041, n=20) and improved fibrinolytic potential (~35% increase, p= 0.028, n=29). Conversely, supplementation of elastase resulted in opposite trend, confirming the contribution of ELANE to intrapleural fibrinolytic deficiency.

Conclusion In conclusion, our data provides a mechanistic insight into how IET may improve clinical outcomes in pleural infection. We find ELANE is a potential mediator of intrapleural fibrinolytic deficiency, and presents to be a conceivable therapeutic target for pleural infection.

ILD: biomarkers and beyond

M1 CLINICAL CHARACTERISTICS AND OUTCOMES OF PLEUROPARENCHYMAL FIBROELASTOSIS (PPFE): A RETROSPECTIVE COHORT STUDY

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Background Pleuroparenchymal fibroelastosis(PPFE) is a rare form of interstitial lung disease(ILD). Studies have shown that IPF patients with PPFE-like features have accelerated progression, poorer prognosis, and shorter telomeres.[1]

Methods A retrospective analysis was performed of consecutive patients with a Multidisciplinary Team consensus diagnosis of PPFE (Feb 2018 - 2026). A convenience sample of 70 consecutive IPF patients was used as a comparator cohort.

Baseline demographics, clinical outcomes, complications, pulmonary function decline at 1 year (defined by ATS criteria: absolute decline in FVC ≥5% predicted, or TLCO ≥10% predicted) and mortality were recorded. Groups were compared using Mann-Whitney U, Fisher's exact test and Kaplan Meier analysis, as appropriate.

Results Forty-three patients had an MDT diagnosis of PPFE. Compared with IPF patients, those with PPFE were younger (71 vs75 years), more frequently female (55.8 vs15.7%), and more likely to be never smokers (60.5% vs18.6%). At presentation, PPFE patients had lower FEV1 and higher TLCO ($p<0.05$), although FVC was comparable between groups. Over half of PPFE patients (53%) had co-existing ILD, most commonly definite or probable UIP (15/23, 65%). PPFE patients also had lower BMI and were more likely to have recurrent infections, pneumothorax or pneumomediastinum ($p<0.05$). A family history of ILD was more common in PPFE (16.3% vs 10.5%), although this was not statistically significant. Among patients in whom NHS R421 genetic testing was indicated and available, 88% (7/8) had a telomere biology-related gene variant (TERT n=4, RTEL1 n=2, DKC1 n=1, none n=1).

There were no significant differences in one-year lung function decline or overall survival between PPFE and IPF patients, irrespective of co-existing ILD.

Conclusions PPFE has a distinct clinical phenotype characterised by lower BMI and increased risks of infection, pneumothorax and pneumomediastinum. Clinical management should emphasise nutritional support and prompt evaluation for these complications in patients presenting with worsening breathlessness, as respiratory deterioration should not be assumed to represent disease progression alone. The high prevalence of telomere biology-related variants provides further support to the importance of telomere dysfunction in PPFE.

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M2 BIGGER LITTLE WOMEN: LOOKING INTO ANTIFIBROTIC TOLERANCE IN RELATION TO WEIGHT, DOSE, FEMALE GENDER AND ETHNICITY

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10.1136/thorax-2026-BTSabstracts.448

Introduction Nintedanib and Pirfenidone are the two antifibrotics licensed in the UK, currently. Both have multiple side effects which affect patient tolerance, the most common being gastrointestinal side effects, such as diarrhoea and nausea. Current guidance is to attempt full dose treatment from commencement of antifibrotics.[1] However, factors such as weight, age, gender, ethnicity were anecdotally felt to influence tolerance of antifibrotic medications. If full dose isn't tolerated, often doses are reduced. Studies suggest that reducing doses of pirfenidone or nintedanib may not have a significant effect on disease progression.[1]

Methods A retrospective longitudinal review of records was conducted for patients with idiopathic pulmonary fibrosis and progressive pulmonary fibrosis initiated on nintedanib and pirfenidone across five hospitals between December 2021 and December 2023. The duration from initiation to data collection varied among patients and was between 20-53 months. The cohort was analysed to determine the proportion of patients receiving no antifibrotic, full dose or reduced dose at the time of censoring.

Results Table one shows our cohort characteristics. 445 patients were included in this study. 316 participants were male. Median participant age was 74 years old. 373/445 were commenced on nintedanib. At censor, 125 participants were on full dose antifibrotic, 113 on reduced dose and 194 had discontinued antifibrotics. Of gender, BMI, ethnicity and age; only median age at commencing antifibrotics significantly differed in the group which continued full dose until censor versus those that discontinued antifibrotics (median age 72 versus 75, respective-

Abstract M2 Table 1 Baseline participant characteristics

Characteristic	Taking full dose antifibrotic at censor (n=125)	Taking reduced dose antifibrotic at censor (n=113)	Discontinued antifibrotic medication at censor (n=194)
Median age (years (IQR))	72 (64-78)	73 (68-78)	75 (69.8-80)
Male Gender	97/125	75/113	136/194
Diagnosis of IPF (as opposed to PPF)	82/125	69/113	119/194
Median starting BMI (kg/m²) (IQR)	29 (26.2-32)	27.4 (25.1-31.4)	29 (26-32)
Caucasian Ethnicity	60/125	73/113	103/194
Unknown ethnicity	64/125	36/113	87/194

ly (P<0.01)). We accept that results are limited by a lack of diversity in our cohort, with the majority of participants being caucasian and male.

Conclusions This retrospective observational study indicates that age at commencement of antifibrotics is significantly associated with treatment discontinuation. However, a larger cohort is needed to determine if ethnicity, gender or BMI have a correlation with treatment dose reduction or discontinuation in order to tailor treatment plans.

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M3 OUTCOMES FOLLOWING RESPIRATORY-RELATED HOSPITALISATION IN PATIENTS WITH FIBROTIC INTERSTITIAL LUNG DISEASE IN THE UK: ISNTERIM RESULTS FROM THE INDEX STUDY

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10.1136/thorax-2026-BTSabstracts.449

Introduction Acute exacerbations of fibrotic interstitial lung disease (AE-fILD) are associated with diagnostic and therapeutic challenges. Hospitalisation is common with fILD, with significant in-hospital mortality. There has been no UK-wide investigation of the management and outcomes for patients with fILD and respiratory hospitalisation.

Methods The INDEX study is an INSPIRE associate, trainee-led, observational, retrospective, multicentre cohort study of patients admitted to hospital with a diagnosis of a respiratory-related hospital admission and fILD, between 1/9/22 – 31/8/23. The study received HRA approval (Reference 24/PR/1425) and was funded as an investigator-initiated study by Boehringer Ingelheim Limited.

Eligible admissions were identified using ICD-10 coding at each participating site. Electronic patient reports were screened against inclusion and exclusion criteria. Case report forms captured patient demographics, diagnostic methods, clinical presentation, imaging and laboratory investigations, and subsequent management.

The primary outcome was transplant-free survival at 90 days following the date of hospital admission.

Results Twenty-two sites were recruited, with over 65 trainees supporting the study.

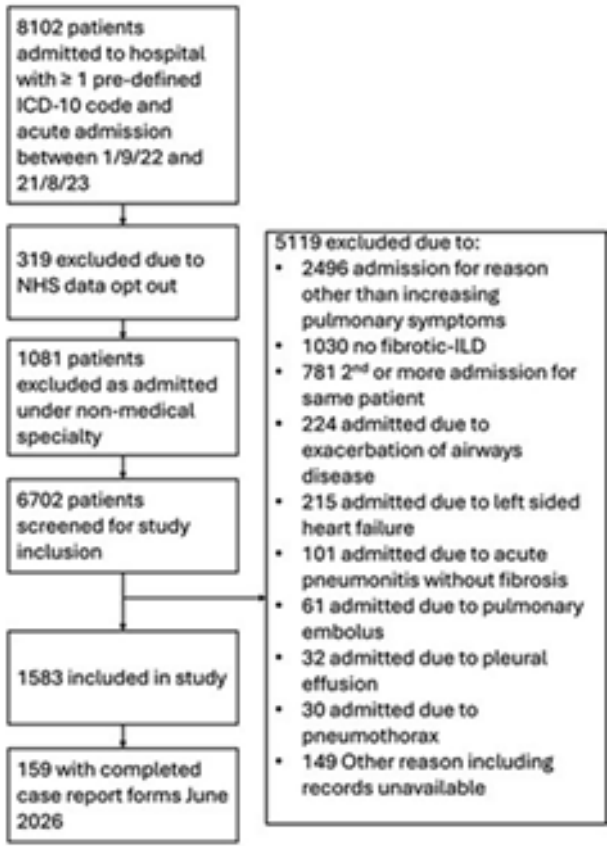
Figure 1 illustrates participant inclusion and exclusion from 16 of the 22 sites that had submitted data by June 2026.

159 cases (102 [64%] male) had completed case report forms. Median age on admission was 78 (IQR 72–84). The most common diagnoses were idiopathic pulmonary fibrosis (54, 40%), unclassifiable ILD (23, 14%) and RA-ILD (14, 9%). 27 cases (17%) had no previous diagnosis of fILD.

In-hospital, 90-day, 6-month and 12-month mortality were 21.4%, 41.5%, 50.3% and 57.9% respectively.

91/159 (57.2%) patients received oral or intravenous corticosteroids during admission.

48/159 (30.2%) had an advanced care plan in place prior to admission, 74/159 (46.6%) had documented evidence of discussion regarding treatment escalation during admission. Of those discharged, 36/125 (28.8%) had a treatment escalation or advanced care plan.



Abstract M3 Figure 1 Interim STROBE diagram for INDEX study

Conclusions Interim analysis of the INDEX study demonstrates substantial mortality following respiratory-related hospitalisation in patients with fILD, with 41.5% mortality at 90 days. Despite this, rates of documented advance care planning and treatment escalation discussions remain low. These findings highlight the need for improved advanced care planning in this high-risk population. Final study results are anticipated in November 2026.

M4 USING A QUALITATIVE CO-DESIGN APPROACH TO DEVELOP A PATIENT RESOURCE ON ARTIFICIAL INTELLIGENCE IN INTERSTITIAL LUNG DISEASE

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10.1136/thorax-2026-BTSabstracts.450

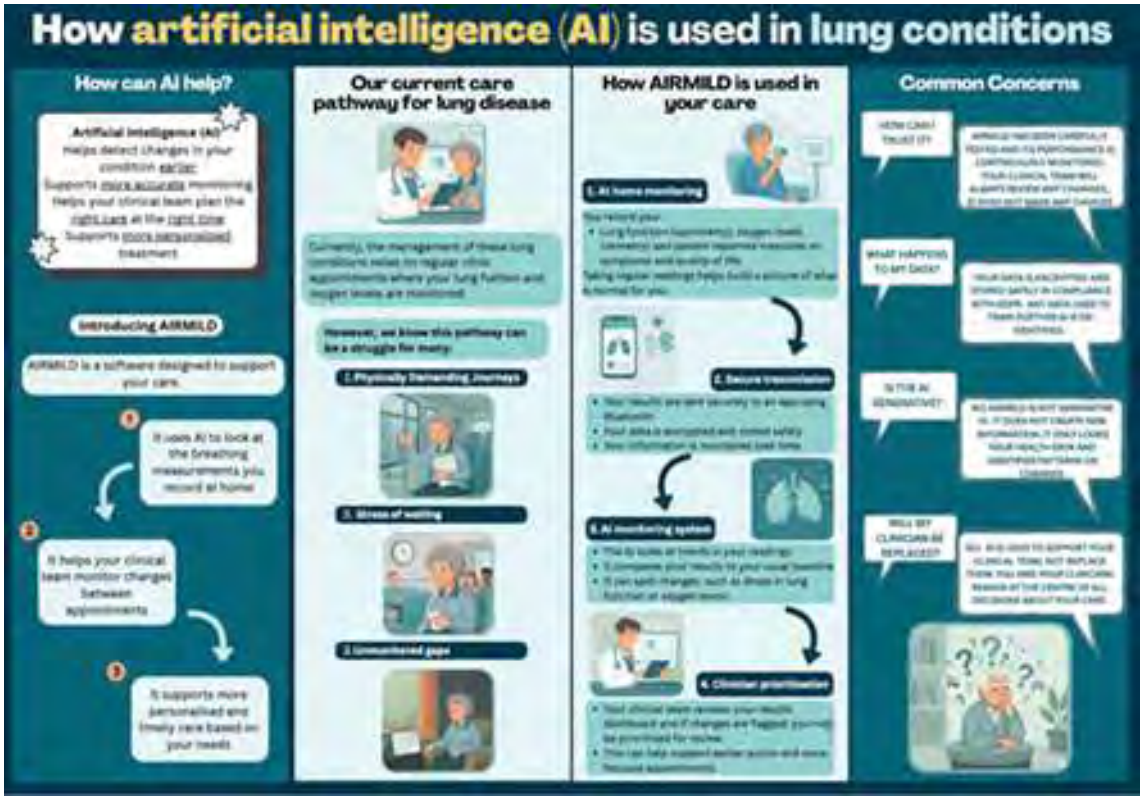
Introduction Artificial intelligence (AI)-enabled remote monitoring may support the earlier detection of clinical deterioration and delivery of more personalised care in interstitial lung disease (ILD). However, limited patient understanding of AI and concerns regarding trust, transparency and data security may affect engagement with these technologies. Patient-centred communication resources are needed to support implementation of AI-enabled technologies.

Aim To co-design a patient information resource explaining the role of AI-enhanced remote monitoring in ILD care.

Methods A qualitative, iterative co-design approach was undertaken through three Patient and Public Involvement and Engagement (PPIE) focus groups involving eleven individuals with lived experience of respiratory disease. Participants represented a range of ages, diagnoses and experience with digital health technologies. A draft patient resource was developed and refined across successive feedback cycles. Facilitated discussions explored perceptions of AI, informational needs, concerns and preferences regarding content, language and presentation. Digital engagement tools, including real-time polling and collaborative annotation, were used to capture feedback and support participation. Data were analysed thematically and findings were used to iteratively modify the resource.

Results Four key themes emerged, including trust in AI, preservation of clinician oversight, data privacy and security, and concerns regarding loss of autonomy. There was strong consensus that AI should be presented as a tool that supports clinicians rather than making independent clinical decisions. Participants preferred explanations centred on patient benefit, including earlier identification of clinical changes, more personalised care and improved continuity between appointments. Feedback informed substantial revisions to content, language, visual design and frequently asked questions, resulting in a patient-centred infographic resource explaining AI-enhanced remote monitoring in ILD (figure 1).

Conclusions Patient involvement is essential when developing educational resources for AI-enabled healthcare. Co-design enabled the creation of a patient-centred resource that directly addresses concerns regarding trust, transparency and clinician oversight. This approach may improve patient understanding and acceptance of AI-enhanced remote monitoring in ILD and could inform implementation of AI technologies across wider respiratory services.



Abstract M4 Figure 1

M5 SYSTEMIC ANTI-CANCER THERAPY-RELATED PULMONARY TOXICITY: A TERTIARY CENTRE EXPERIENCE

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10.1136/thorax-2026-BTSabstracts.451

Introduction Newer cancer therapies including immunotherapy have contributed to an increase in drug-induced lung disease (DILD) referrals to our ILD service. Collaboration between respiratory and oncology colleagues has been essential to promptly recognise and treat pulmonary toxicity. In this observational analysis, we present our centre's experience.

Methods We retrospectively reviewed electronic case records of 144 consecutive patients referred with suspected DILD to our tertiary ILD service, with 124 confirmed systemic anti-cancer therapy (SACT)-related DILD between July 2020 to March 2026.

Results Demographics: 60% male (n=124). Median age at diagnosis 68 years (IQR 58-75; range 22 - 86). Time from drug initiation to CT evidence of DILD: median 3.4 months (IQR 2.0-8.4; range 0.1 - 53.6).

Drug classes (figure 1): 79 immune checkpoint inhibitors (ICI), 11 Herceptin conjugates, 8 conventional chemotherapy agents, 7 tyrosine kinase inhibitors (TKI), 6 cyclin-dependent kinase/CDK blockers, 6 Bleomycin, 4 novel/trial agents, 2 mTOR inhibitors, and 1 poly-(ADP-ribose)-polymerase/PARP inhibitor.

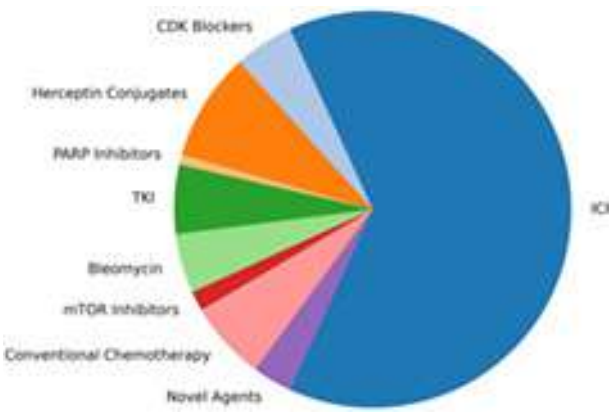
Cancer type distribution in ICI: 39.2% melanoma, 26.6% lung cancer, 7.6% mesothelioma, 7.6% breast cancer, 5.1% genitourinary cancer, 5.1% head and neck cancer, 5.1% upper gastrointestinal cancer, 2.6% skin squamous cell cancer, 1.3% lymphoma.

Of ICI toxicities, 21 (27%) occurred with dual ICI therapy, and 58 (73%) with monotherapy or chemotherapy-combination.

The number of referrals increased over time: 10 in 2020 (6 months), 15 in 2021, 17 in 2022, 22 in 2023, 26 in 2024, 30 in 2025.

107 patients (86%) were treated with steroids (Grade ≥2 toxicity). Mycophenolate mofetil (MMF) was used in 16 patients, only in ICI toxicity (13% overall, 21% of all ICI toxicities). Tocilizumab was used once, alongside MMF.

Drug-toxicity related deaths: total 16/124 (13%). ICI 7/79 (9%), CDK blockers 5/6 (83%), Herceptin conjugates 3/11 (27%), conventional chemotherapy 1/8 (13%).



Abstract M5 Figure 1

Conclusion Our data suggest that DILD from SACT represents a growing challenge for respiratory physicians. Close collaboration between respiratory and oncology physicians should aim to reduce morbidity and mortality and permit rechallenge where possible. The burden of immunotherapy related DILD reflects the evolving landscape of cancer treatment. Understanding mechanisms behind DILD is essential to counselling patients on risk and developing non-steroid based treatment regimens.

M6 NUTRITIONAL STATUS AND MALNUTRITION SCREENING IN INTERSTITIAL LUNG DISEASE: A SYSTEMATIC REVIEW

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10.1136/thorax-2026-BTSabstracts.452

Introduction and Objectives Malnutrition and nutritional risk have been reported in interstitial lung disease (ILD), with potential implications for clinical outcomes and quality of life. Despite increasing awareness of nutritional risk in chronic respiratory conditions, the assessment and management of malnutrition remain limited in routine ILD care.

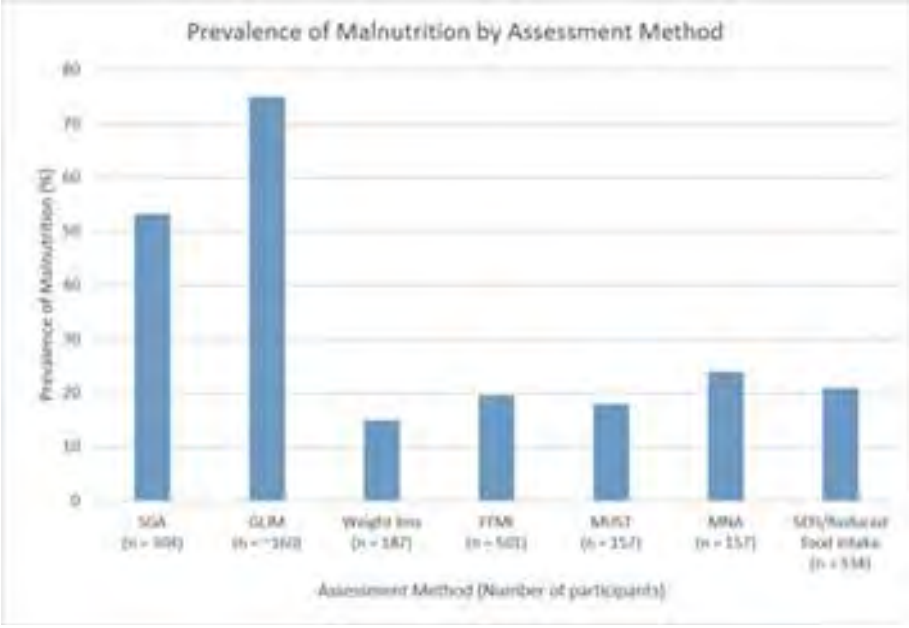
A systematic review was conducted to determine the prevalence of malnutrition and identify screening tools used to assess nutritional status in adults with ILD.

Methods The systematic review protocol was registered on PROSPERO (CRD420251013336). Eligible studies included adults (≥18 years) with clinically-diagnosed ILD of any aetiology reporting nutritional status, limited to Englishlanguage publications (01/01/2000 – 04/06/2026). Studies with major comorbidities independently affecting nutrition (e.g. cancer, HIV) and systemic sclerosisassociated ILD were excluded.

Searches covered CINAHL, AMED, MEDLINE, EMBASE, and CENTRAL, supplemented by grey literature and citation-chasing. Meta-analysis was not possible due to heterogeneity in screening tools, diagnostic criteria, and disease stage. Risk of bias was assessed using Clinical Appraisal Skills Programme (CASP) tools.

Results Ten studies (n = 839) reported malnutrition screening and prevalence across diverse ILD diagnoses and severities. Mean reported age was 61–74.4years; BMI 27.9±4.7 kg/m^2. Across all studies, 66% of participants were male. Assessment methods included Subjective Global Assessment (SGA; n=304), Global Leadership Initiative on Malnutrition (GLIM; n=∼160), weight loss (n=187), Fat-Free Mass Index (FFMI; n=501), Malnutrition Universal Screening Tool (MUST; n=157), Mini Nutritional Assessment (MNA; n=157), and Simple Evaluation of Food Intake (SEFI)/reduced intake (n=334). Reported malnutrition prevalence ranged 5–84%, reflecting variation in criteria, thresholds, and disease stage. Evidence indicated malnutrition is common in ILD and worsens with progression, yet screening remains inconsistent. Study quality and methodological variability limited comparability.

Conclusions Malnutrition in ILD is prevalent but frequently underscreened and undertreated. BMI values often fall within overweight and obese ranges, suggesting that reliance on



Abstract M6 Figure 1 SGA Subjective global assessment (4 studies); GLIM global leadership initiative on malnutrition (2 studies); weight loss (2 studies); FFMI fat free mass index (5 studies); MUST malnutrition universal screening score (2 studies); MNA mini nutritional assessment (2 studies); SEFI simple evaluation of food intake (3 studies)

anthropometric measures alone may mask malnutrition, particularly sarcopenic obesity and altered body composition, which may contribute to poorer outcomes or survival.

Standardised screening protocols and early nutritional intervention are required. ILD management would benefit from integrated nutrition pathways, but high-quality research is needed to firmly establish evidencebased guidelines.

M7 ILD SUBTYPE DISTRIBUTION IN PATIENTS OF SOUTH ASIAN VS NON-SOUTH ASIAN ETHNICITY ACROSS TWO UK ILD CENTRES

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10.1136/thorax-2026-BTSabstracts.453

Introduction and Objectives Hypersensitivity pneumonitis (HP) is reportedly the commonest interstitial lung disease (ILD) in South Asians in the Indian subcontinent.[1] To date, no studies have examined whether this is also the case in South Asian ILD patients in the UK. We therefore aimed to characterise ILD subtype distribution across two large ILD clinical services with substantial populations of South Asian ethnicity.

Methods We analysed ILD clinic incident cases from two UK centres (Centre 1: 23 months across 2023-2025; Centre 2: 22 months, 2023-2024). Patients were classified by ethnicity (South Asian (SA) versus Non-South Asian (NSA)) and ILD diagnostic subtype. Data were analysed by Chi-squared and multinomial regression analyses.

Results 1,541 patients were included (Centre 1: SA=143 (21.3%), NSA=528 (78.7%); Centre 2: SA=83 (9.5%), NSA=787 (91.5%)). ILD subtype profiles differed between SA and NSA patients, and SA and NSA profiles were similar between centres. Idiopathic ILD (IPF + iNSIP) was more common in NSA than SA patients. Rheumatoid arthritis/connective tissue disease associated-ILD (RA/CTD-ILD) and, to an even greater degree, HP were also more common in SA than NSA patients when variations in idiopathic incidence and centre effects were accounted for.

Conclusions The findings from this multi-centre study of ILD phenotypic distribution in South Asian populations in the UK are consistent with the distributions seen in the Indian sub-continent, in particular for HP and CTD-ILD cases. As the exposures implicated in HP in the Indian subcontinent are unlikely to apply to the UK, culturally specific indoor inhalational exposures within South Asian households in the UK may represent unidentified HP triggers.

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M8 FLOW-ILD: REAL-CASE VIGNETTE STUDY OF HIGH-FLOW NASAL OXYGEN AND ESCALATION DECISIONS IN ACUTE INTERSTITIAL LUNG DISEASE DETERIORATION

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10.1136/thorax-2026-BTSabstracts.454

Introduction Acute deterioration in interstitial lung disease (ILD) carries high mortality and uncertainty around escalation.[1] Using real-case vignettes, we explored variation in consultants' decisions regarding high-flow nasal oxygen (HFNO) initiation, prognostic judgement, ICU referral, invasive ventilation and treatment limitation.

Methods A single-centre exploratory survey where respiratory medicine, intensive care and acute/on-call consultants reviewed

Abstract M8 Table 1 HFNO possible includes 'yes' or 'unsure/need further information'. Invasive ventilation supported/conditional includes 'yes', 'only if reversible cause identified', or 'time-limited trial'. ROX index was calculated as (SpO_2/FiO_2)/respiratory rate, assuming FiO_2 0.8 for non-rebreathe mask and 0.6 for Venturi oxygen

Case	FRED	ROX	Outcome	Median predicted hospital survival, % (range)	Mean absolute prediction error, %	HFNO appropriate/ possible, n/12	ICU referral/ discussion, n/12	Invasive ventilation supported/ conditional, n/12
1	2	3.45	Survived	70 (20–90)	30.8	12	12	10
2	6	3.71	Died in hospital	10 (0–40)	17.5	7	1	0
3	3	5.33	Survived	55 (30–80)	44.2	12	11	8
4	4	4.03	Died by 90 days	50 (30–70)	48.3	11	12	9
5	7	3.07	Died in hospital	10 (0–20)	12.5	5	0	0

five anonymised cases of patients with ILD presenting with acute hypoxaemia. Observed hospital and 90-day mortality were used for analysis. Responses were analysed descriptively, including prediction error, intraclass correlation coefficient (ICC), associations between predicted survival and escalation decisions, and FRED/ROX contextualisation. Free-text responses underwent thematic coding.

Results Twelve clinicians completed the survey, generating 60 clinician-case assessments. Prognostic estimates and escalation decisions are summarised in table 1. Exploratory inter-rater analysis showed moderate-to-good agreement in relative prognostic ordering for hospital survival estimates, ICC(2,1)=0.79. HFNO was considered appropriate/possible in 47/60 assessments, ICU referral/discussion in 36/60, and invasive ventilation was supported/conditional in 27/60. Higher predicted hospital survival was more strongly associated with invasive ventilation support than HFNO consideration (point-biserial r=0.73 versus r=0.57). Immediate treatment-limitation discussion was recommended in 53/60 assessments.

Clinicians recognised high-risk/end-stage cases, with low predicted survival and low ICU/intubation support in high-FRED Cases 2 and 5. Variation was greater in Cases 3 and 4, where intermediate FRED and borderline ROX values reflected potentially reversible or diagnostically uncertain deterioration. Thematic coding suggested specialty-linked decision frames: respiratory clinicians emphasised ILD trajectory/reversibility, intensivists emphasised physiological rescue/conditional escalation, and acute/on-call clinicians emphasised frailty, treatment burden and advance care planning. Between-group patterns suggested lower survival estimates among respiratory clinicians, while intensivists more often supported HFNO and conditional invasive ventilation. Self-rated prognostic confidence did not correspond to lower prediction error.

Conclusions Clinicians distinguished advanced/end-stage ILD from potentially reversible deterioration, but varied in how prognosis was translated into HFNO, ICU referral and invasive ventilation decisions. HFNO was considered across a broader prognostic range than invasive ventilation, suggesting roles as bridge therapy, time-limited trial, ceiling of support or palliation. Additional recruitment is planned to further characterise specialty-specific decision frames, informing targeted education and clearer HFNO/escalation decision-support in acute ILD deterioration.

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M9 COMPARISON OF LONGITUDINAL 6-MINUTE WALK DETERIORATION, DO-GAP AND ODDS SCORE PROGRESSION FOR MORTALITY PREDICTION IN IDIOPATHIC PULMONARY FIBROSIS

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Introduction Multidimensional 6 minute walk test (6MWT) prognostic scores including distance-oxygen-gender-age-physiology (DO-GAP) index and the oxygen, distance, dyspnoea, saturation (ODDS) score improves risk stratification in idiopathic pulmonary fibrosis (IPF), but the significance of their long-term changes compared to single 6MWT measures is unclear.

Aims To compare longitudinal change in 6-minute walk test (6MWT) parameters, DO-GAP, ODDS and mortality in a real-world IPF cohort.

Methods A retrospective study of IPF patients who underwent 6MWT between 2019 and 2025. Longitudinal change from baseline to first follow-up was assessed for 6MWD, minimal SpO2% distance saturation product (DSP), DO-GAP, ODDS, and Forced vital capacity (FVC) predicted. Mortality outcomes at 1 and 2 years following the second visit were assessed. Receiver operating characteristic (ROC) analysis was performed to compare prognostic discrimination. Cox proportional analyses adjusted for age, sex, body mass index (BMI), antifibrotic therapy history, baseline FVC %, early 6MWT termination (due to significant desaturation) and time from baseline 6MWT were performed to identify independent predictors of mortality.

Results IPF cohort of 203 patients were included (80% male; mean age 74.1± 7.0 years). Median time between baseline and 1st follow-up 6MWT was 473 days (IQR 451). At baseline, DO-GAP demonstrated greatest discrimination for both 1-year and 2-year mortality (AUC 0.83 and 0.85 respectively), followed by ODDS, DSP and FVC%. Longitudinally, change in 6MWD demonstrated greatest discrimination for both 1-year and 2-year mortality (AUC 0.71 and 0.76 respectively), followed by DSP and FVC. Change in minimum SpO_2 was not predictive (see table 1). In multivariate Cox analyses adjusted for age, sex, BMI, baseline FVC %, early 6MWT termination and time from baseline 6MWT assessment, longitudinal deterioration in 6MWD, DSP, DO-GAP and ODDS independently predicted 2-year mortality. A 10 m decline in 6MWD was asso-

Abstract M9 Table 1 Prognostic discrimination of baseline and longitudinal IPF measures

	1-year (n=203)	2- years (n=150)
Baseline 6MWD	0.70 (0.63-0.78) ***	0.74 (0.66-0.81) ***
Baseline minimal SpO_2%	0.67 (0.59-0.77) ***	0.73 (0.65-0.81) ***
Baseline DSP	0.73 (0.65-0.80***	0.77 (0.70-0.85) ***
Baseline ODDS	0.75 (0.68-0.83) ***	0.79 (0.71-0.87) ***
Baseline DO-GAP	0.83 (0.77-0.90) ***	0.85 (0.78-0.91) ***
Baseline FVC %	0.75 (0.67-0.83) ***	0.70 (0.61-0.78) ***
Δ 6MWD	0.71(0.63-0.79) ***	0.76 (0.68-0.84)***
Δ Minimal SpO2% change	0.50(0.40-0.59)	0.50 (0.41-0.59)
Δ DSP	0.69(0.60-0.77) ***	0.74 (0.66-0.82) ***
Δ ODDS	0.66(0.57-0.75) ***	0.66 (0.56-0.75) ***
Δ DO-GAP	0.70 (0.60-0.80) ***	0.70 (0.61-0.79) ***
Δ FVC %	0.70(0.61-0.78) ***	0.72(0.64-0.80) ***

Δ change from visit 1 to visit 2 *** = p<0.001 ** = p<0.01 6MWD= 6-minute walk distance DSP= Distance saturation product (6MWD x Minimal SpO_2) DO-GAP= Distance-Oxygen-Gender-Age-Physiology index ODDS= oxygen distance diffusion efficiency FVC=forced vital capacity

ciated with a 6% increase in mortality risk (HR 1.06, 95%CI 1.03–1.09; p<0.001).

Conclusion At baseline, DO-GAP demonstrated the strongest mortality discrimination. Longitudinal deterioration in 6MWD, DSP, DO-GAP and ODDS independently predicted mortality. Despite its simplicity, longitudinal change in 6MWD demonstrated prognostic performance comparable to multidimensional composite scores, supporting serial 6MWT assessment as a practical tool for monitoring disease progression in IPF.

M10 COMBINED EBUS-TBNA/TBNB AND EUS-B-FNA/FNB IMPROVES TISSUE DIAGNOSIS OF GRANULOMATOUS LUNG DISEASE PRESENTING WITH ISOLATED MEDIASTINAL LYMPHADENOPATHY: A PROSPECTIVE RANDOMIZED TRIAL

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10.1136/thorax-2026-BTSabstracts.456

Introduction and Objectives Histological confirmation remains essential for the diagnosis of granulomatous diseases presenting with isolated mediastinal lymphadenopathy (IML). EBUS-TBNA/TBNB is the gold standard for mediastinal lymph node (LN) assessment. Although, in lung cancer staging, combined EBUS/EUS-b approach has shown higher diagnostic yield than EBUS alone, prospective randomized data evaluating its role in benign granulomatous lung disease (GLD) presenting with IML are lacking.

The aim of the study is to compare the diagnostic yield of combined EBUS-TBNA/TBNB with EUS-b-FNA/FNB versus EBUS-TBNA/TBNB alone in patients with undiagnosed IML.

Methods Prospective single-center randomized study of patients with mediastinal with/without hilar lymphadenopathy on imaging (CT, PET/CT) involving at least 2 lymph node stations with at least one of them is EUS-b-accessible. Patients with intraparenchymal lesion suspicious for lung cancer are excluded from the study.

Patients are randomized to EBUSalone or combined approach (during a single endoscopic session, under conscious sedation) according to two parameters of randomization. These are gender (male or female) and the short axis diameter of the smallest mediastinal targetedlymph node (10 - 15 mm or > 15 mm).

Abstract M10 Table 1

Characteristics		EBUS (n = 40)	EBUS + EUS (n = 31)	p-value
Gender, n (%)	Female	25 (62.5%)	18 (58,1%)	0.70
	Male	15 (37.5%)	13 (41,9%)	
Short axis diameter of the smallest biopsied mediastinal LN, n (%)	10 – 15 mm	27 (67.5%)	22 (71.0%)	0.75
	> 15 mm	13 (32.5%)	9 (29.0%)	
Diagnostic yield of procedure, % (n of diagnosis/n of procedures)	In total	77.5% (31/40)	90.3% (28/31)	0.21
	GLD	75.0% (24/32)	96.2% (25/26)	**0.049**
	Non-infectious GLD	74.1% (20/27)	96.0% (24/25)	0.051
	Sarcoidosis	72.0% (18/25)	96.0% (24/25)	**0.049**
	Tuberculosis	80% (4/5)	100% (1/1)	1.00
	Berylliosis and other granulomatous	100% (2/2)	-	-
	occupational diseases	85.7% (6/7)	33.3% (1/3)	0.18
	Other diagnosis	100% (1/1)	100% (2/2)	1.00
	Reactive			
Diagnostic failure, % (n of diagnostic failure/n of procedures)	In total	22.5% (9/40)	9.7% (3/31)	0.208
Major Complications, n (%)		-	-	-

We defined samples as diagnostic when a definitive diagnosis has been made. Patients with non-diagnostic sample results underwent other invasive procedures or follow-up of at least six months.

Results To date, 82 patients have been enrolled; 11 patients remain under follow-up and were excluded from the analysis. In GLD (sarcoidosis, tuberculosis, berylliosis and other occupational granulomatous diseases) diagnostic yield was significantly higher with the combined approach [96.2% (25/26 patients) vs 75.0% (24/32 patients), p=0.049]. In sarcoidosis, diagnostic yield reached 96.0% versus 72.0% (p=0.049). Overall diagnostic yield was 90.3% versus 77.5%. Diagnostic failure was reduced from 22.5% to 9.7% without major procedure-related complications (table 1).

Conclusions The addition of EUS-b-FNA/FNB to EBUS-TBNA/TBNB improves tissue diagnosis of granulomatous lung disease presenting with isolated mediastinal lymphadenopathy, particularly sarcoidosis, while reducing diagnostic failure during a single endoscopic session without major complications. These findings support the combined approach as a valuable diagnostic strategy for this group of patients.

M11 THE ROLE OF KL-6 IN PULMONARY SARCOIDOSIS

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Introduction and Aims The clinical course of sarcoidosis is highly heterogeneous, ranging from spontaneous remission to chronic, progressive phenotypes that severely impact organ function and survival. The lack of reliable biomarkers for early diagnosis, disease stratification, prediction of progression, and treatment response remains a major clinical challenge. KL-6 (Krebs von den Lungen-6) is a promising biomarker in ILD. We aim to assess the role of KL-6 as a prognostic and fibrotic biomarker in pulmonary sarcoidosis.

Methods Serum samples from 79 patients with sarcoidosis had KL-6 quantified using a commercial ELISA kit (CUSABIO, Wuhan). 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. Correlation analysis was performed for Lung function at baseline, Composite Physiological Index (CPI), baseline inflammatory markers (ESR + CRP), Neutrophil Lymphocyte Ratio (NLR), and disease duration. ROC analysis using KL-6 and physiological measurements, including the CPI, was performed to assess the ability to discriminate between fibrotic and non-fibrotic sarcoidosis.

Results The mean age of the cohort was 61.1, disease duration 10.2 years, 66% were male, and 52% received immunomodulation. KL-6 levels were stratified by fibrosis (CXR stage 0+1 v 4). KL-6 levels were significantly higher in fibrotic patients. The results were not influenced by immunomodulation. KL-6 correlated negatively with FVC% -.247 (p 0.031), DLCO% -.243 (p 0.037), and positively with Disease duration .292 (p .009) and fibrotic status .425 (p <0.001). There was no statistically significant relationship between KL-6 and ESR, CRP, NLR, or CPI. ROC analysis (figure 1) demonstrated the superior ability of KL-6 to discriminate between fibrotic and non-fibrotic disease.

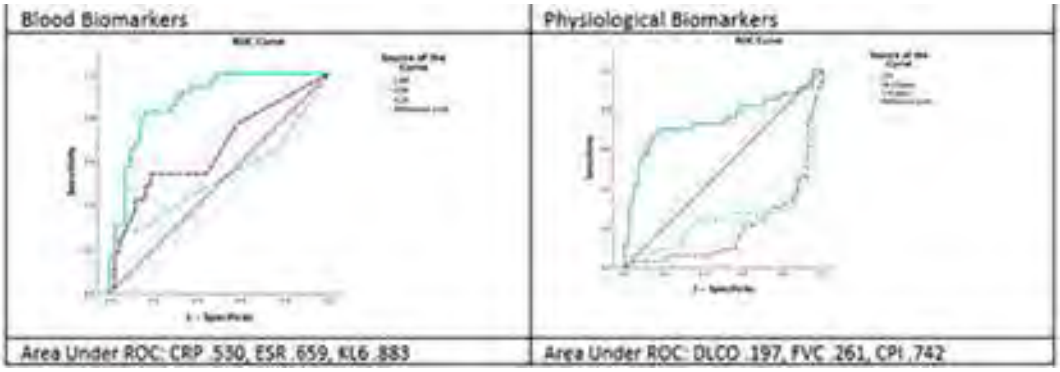
Conclusions Our results demonstrate that KL-6 has fibrotic (CXR Stage 4) and Physiological (FVC & DLCO) correlation. KL-6 is fundamentally positioned not as a tool for the initial diagnosis of sarcoidosis, but rather as a signal of ongoing alveolar epithelial damage and fibrotic remodelling. The study is limited by static measurement. Sequential monitoring of the change in KL-6 over time could be a powerful predictor of subsequent progression.

M12 ELEVATED FENO: A DIAGNOSTIC CLUE TO UNMASK CHRONIC/FIBROTIC HYPERSENSITIVITY PNEUMONITIS

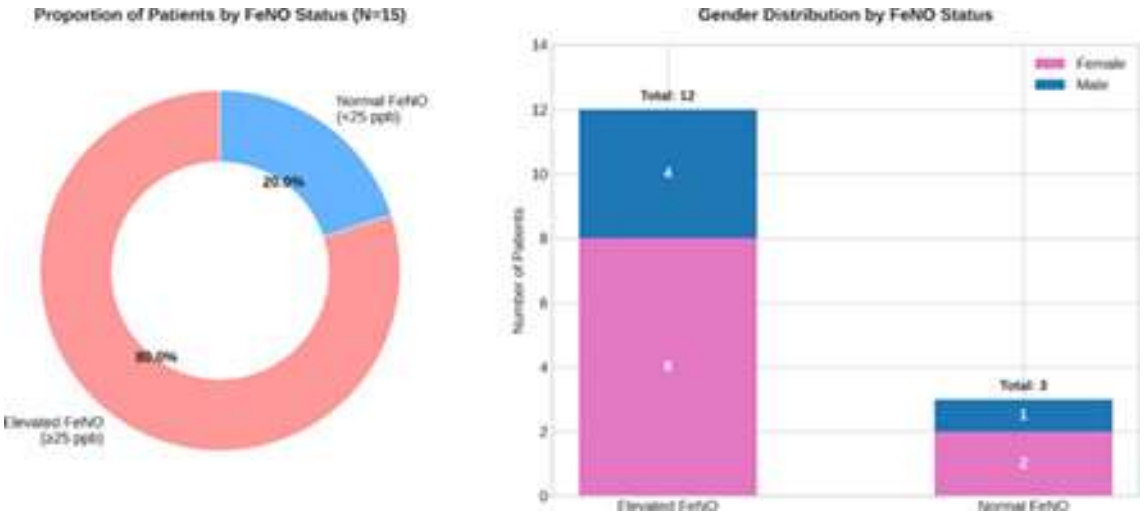
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Introduction Chronic/fibrotic hypersensitivity pneumonitis (C/F-HP) remains underdiagnosed despite its high prevalence, largely because current diagnostic pathways often require cumbersome invasive lung biopsy.[1] HP is classically driven by type III and IV hypersensitivity reactions, yet its precise pathophysiology is incompletely understood.[2] Previously, the association of allergic rhinitis (AR) with C/F-HP has been described.[3] Interestingly, treating comorbid AR (a type I response) in HP patients can alleviate respiratory symptoms, suggesting unappreciated crosstalk between type I and type III/IV inflammatory cascades.[2 3] Fractional exhaled nitric oxide (FeNO), a well-established biomarker of type II airway inflammation, might therefore simplify the diagnostic workup and also illuminate these overlooked mechanistic links.[4 5]



Abstract M11 Figure 1 ROC Analysis for blood and physiological biomarkers



Abstract M12 Figure 1

Objectives To assess whether FeNO measurements can serve as a noninvasive tool to differentiate C/F-HP from other interstitial lung diseases (ILDs).

Methods Fifteen patients with established C/F-HP (ACCP criteria) and concurrent AR with elevated serum IgE were enrolled. Patients were clinically stable on maintenance lowdose or intermittent systemic steroids. Exclusion criteria included known causes of ILD, respiratory failure, and comorbid conditions. FeNO was measured under standardized conditions per ATS/ERS recommendations.

Results Among 15 patients, 12 (80%) demonstrated persistently elevated FeNO (≥25 ppb) despite ongoing steroid therapy. This subgroup included 8 females and 4 males, aged 18–65 years. Mean FeNO in the elevated subgroup was 27 ppb (range not reported). Three patients had normal FeNO: one male had a co-infection, one female was on high-dose systemic corticosteroids, and one female had no identifiable confounder.

Conclusion Elevated FeNO persists in the majority of C/F-HP patients despite systemic steroid use. The combination of AR and elevated FeNO may serve as a potential non-invasive biomarker to differentiate C/F-HP from other ILDs. Routine upper airway assessment and FeNO measurement warrant further study to improve diagnostic accuracy, reduce need for biopsy, and guide antiinflammatory therapy. Larger prospective studies are needed to validate these findings and elucidate type I–type III/IV crosstalk.

M13 A STUDY EVALUATING THE DIAGNOSTIC UTILITY OF CHITOTRIOSIDASE VERSUS SERUM ANGIOTENSIN-CONVERTING ENZYME IN SARCOIDOSIS, INCORPORATING CLINICAL AND RADIOLOGICAL DISEASE ACTIVITY DATA

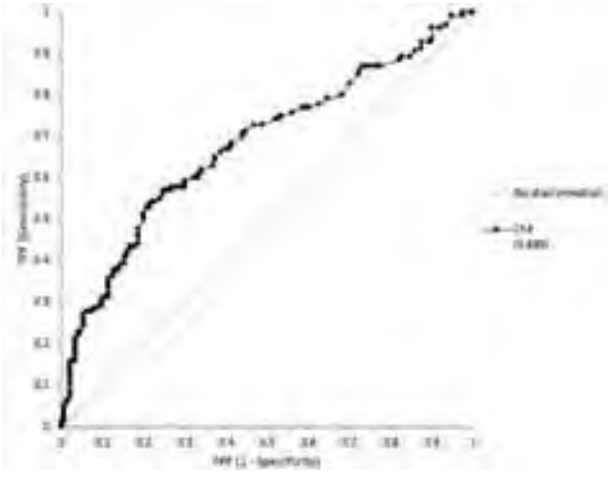
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Background Sarcoidosis is a multisystem granulomatous disease requiring reliable biomarkers for diagnosis and disease monitoring. Serum angiotensin-converting enzyme (ACE) is widely used but has well-documented diagnostic limitations. Chitotriosidase, a macrophage activation marker, has been proposed as a more sensitive alternative. Our first-cycle study (n=31) suggested chitotriosidase outperformed ACE but had limited sample size. This second study aimed to validate these findings in a substantially larger cohort, providing more robust estimates of diagnostic performance for both biomarkers across a regional NHS health board.

Methods A retrospective record analysis was conducted of patients undergoing chitotriosidase and/or serum ACE measurement across a regional NHS health board. Exclusion criteria included requests for lysosomal storage disorder investigation, duplicate requests, and cases without a confirmed sarcoidosis outcome. Positive results were defined as chitotriosidase >55 nmol/hr/ml or ACE ≥88 U/L. Diagnostic performance metrics - sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) - were calculated using standard 2×2 contingency tables.

Results 308 patients with confirmed sarcoidosis outcomes were included for chitotriosidase analysis (154 of this cohort had either clinically, histologically, and/or radiologically confirmed sarcoidosis); 154 also had evaluable ACE results. Chitotriosidase demonstrated markedly superior sensitivity compared to ACE (62.1% vs 12.8%), while ACE showed higher specifici-



Abstract M13 Figure 1 ROC curve for chitotriosidase

ty (93.4% vs 66.0%). Receiver operating characteristic (ROC) curves were generated for both biomarkers, with the area under the curve for chitotriosidase calculated at 0.68 (figure 1). Compared to cycle one, performance metrics were more modest for both biomarkers, consistent with reduced small-sample bias in this larger, more representative cohort.

Conclusion In this substantially larger second-cycle cohort, chitotriosidase demonstrated superior sensitivity to ACE for sarcoidosis diagnosis, though both metrics were more modest than the first cycle suggested. ACE retains high specificity but misses the majority of cases, limiting its utility as a standalone test. These findings support continued use of chitotriosidase as part of the diagnostic workup but highlight that neither biomarker alone is sufficient.

M14 CLINICAL AND HUMANISTIC BURDEN OF PULMONARY HYPERTENSION ASSOCIATED WITH INTERSTITIAL LUNG DISEASE (PH-ILD): SECONDARY ANALYSIS OF THE ADELPHI DISEASE SPECIFIC PROGRAMME (DSP)

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Introduction/Objectives This study provides real-world insights into patient journey, treatment patterns, and disease/caregiver burden associated with PH-ILD.

Methods Data were analysed from the Adelphi Real World PH-ILD DSP, a cross-sectional survey with retrospective data collection from specialist physicians and their patients in the US and Europe (11/2024-06/2025). Analyses were descriptive.

Results Overall, 176 physicians reported data on 515 patients. Mean (SD) patient age was 65.2 (10.5) years, 43.7% were female, and 87.0% White. Hypertension (52.8%) and dyslipidaemia (30.5%) were the most common comorbidities. Idiopathic pulmonary fibrosis (44.7%) was the predominant ILD subtype; 23.1% were diagnosed with an autoimmune disease associated with ILD. Patients had ILD for mean (SD) 2.9 (2.6) years and were WHO Functional Class I (11.1%), II (54.2%), III (29.3%), and IV (5.4%) at survey date. Dyspnoea-related symptoms, cough, and fatigue were common at first presentation and prompted suspicion of ILD. Mean (median) time from first ILD presentation to PH-ILD diagnosis was 25.3 (15.7) months. Overall, 27.6% of patients were misdiagnosed with other cardiovascular conditions or chronic obstructive pulmonary disease. Most patients (73.2%) received ILD treatment at survey date, with nintedanib prescribed most often (53.3%). Most (68.3%) received PH treatment, with sildenafil (25.3%) being the most frequently prescribed. In the US, 38.6% of patients were treated with inhaled treprostinil. In the year before survey date, 50.0% had exacerbations managed with corticosteroids (76.2%)/oxygen therapy (68.0%). At diagnosis, 35.8% of patients reported a 6-minute walk distance <250 metres versus 43.6% at most recent visit. Moderately (50%-75% of predicted value) to severely (<50% of predicted value) reduced lung function, as indicated by forced vital capacity, was experienced by 83.8% of patients at most recent test versus 77.0% at diagnosis. PH-ILD related hospitalizations occurred in 27.2% of patients in the year before survey date; 49.7% required caregiver support. For 27.3% of caregivers, care was extremely/very burdensome.

Conclusions These findings indicate a substantial clinical and caregiver burden associated with PH-ILD. Despite the association of PH with ILD, misdiagnosis poses a challenge and impacts treatment initiation. Most patients diagnosed with PH-ILD are receiving treatment, but unmet needs persist, with many still not receiving standard therapy.

M15 EFFICACY AND RADIOLOGICAL PREDICTORS OF RESPONSE TO PULSED INTRAVENOUS CYCLOPHOSPHAMIDE (IV-CYC) IN REFRACTORY ILD

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Introduction Refractory interstitial lung disease (ILD) — progressive despite first-line immunosuppression — carries significant morbidity, and real-world data on the efficacy of pulsed IV-CYC and predictors of response remain limited. We evaluated lung-function outcomes and candidate radiological predictors following pulsed IV-CYC in refractory ILD.

Methods A single-centre retrospective cohort (2011–2024) of patients receiving pulsed IV-CYC for refractory ILD. The primary endpoint was FVC% predicted stabilisation or improvement (ΔFVC% ≥ -5 percentage points, pre-treatment to first post-treatment measurement). Baseline HRCT was scored for ground-glass opacity (GGO) and a composite fibrosis score (reticulation + traction bronchiectasis + honeycombing). Subgroup and radiological analyses were prespecified; two patients received two courses and are reported per patient using the index course.

Results Thirty-six patients received pulsed IV-CYC (CTD-ILD n=15; hypersensitivity pneumonitis [HP] n=13; other ILD n=8) across 38 courses. Mean age was 62 years; 50% female; 75% (27/36) completed six cycles; median follow-up 36 months.

Paired FVC% were available in 33 patients. Overall, 24/33 (73%) stabilised or improved (mean ΔFVC +1.5 points), with DLCO broadly preserved. CTD-ILD stabilised in 12/15 (80%) (mean ΔFVC +4.2 points), with the largest gains in anti-Ro52/anti-synthetase-associated and anti-Ro/lupus disease. HP stabilised in 8/11 (73%) (mean ΔFVC +1.5 points), and other ILD in 4/7 (57%; mean ΔFVC −4.3 points).

Lower baseline composite fibrosis (<50%) was associated with greater FVC response than higher fibrosis (mean ΔFVC +6.9 vs −1.0 points; p=0.19), whereas baseline GGO burden was not.

Treatment was generally tolerated. A serious adverse event occurred in 1 patient (haemorrhagic cystitis); one further patient discontinued after four cycles owing to neutropenia. Other effects were non-serious (nausea, fatigue, transient malaise).

Conclusions Pulsed IV-CYC was associated with lung-function stabilisation in most patients with refractory ILD, with the greatest response in CTD-ILD — particularly anti-Ro52/anti-synthetase-associated disease. Lower baseline fibrotic burden was associated with greater benefit, supporting earlier, CT-guided intervention before established fibrosis develops. Concurrent immunosuppression and retrospective single-centre design limit causal inference; prospective validation is warranted.

Real world pleural medicine: improving care outcomes, training and delivery

M16 CORRELATION OF CHEST CT PATTERNS WITH PLEURAL FLUID BIOCHEMISTRY, INFLAMMATORY MARKERS AND MICROBIOLOGY IN ADULT PLEURAL INFECTION: AN EXPLORATORY STUDY

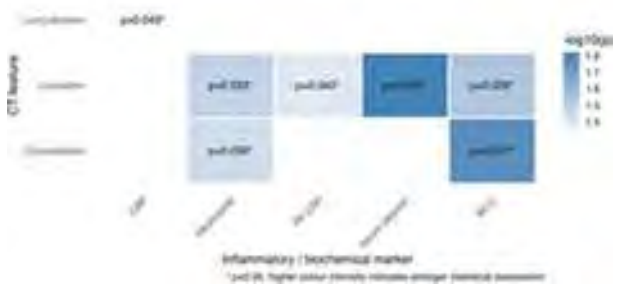
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Background and objective The correlation between chest computed tomography (CT) features, pleural fluid (PF) biochemistry, baseline inflammatory markers, and microbiology in pleural infection remains poorly defined. This exploratory study aimed to assess whether certain chest CT patterns are associated with PF biochemistry and microbiology findings in patients with pleural infection.

Method In this retrospective exploratory study, adults admitted with pleural infection to Oxford University Hospitals who underwent baseline chest CT for a clinical reason alongside PF sampling and routine blood tests were included. Chest CT scans were independently scored by two expert thoracic radiologists using a predefined proforma assessing loculation, consolidation, pleural enhancement, and lung abscess. Chest CT features were correlated with PF microbiology, biochemistry (lactate dehydrogenase [LDH], total protein, glucose) and baseline inflammatory markers (C-reactive protein [CRP], white cell count [WCC], neutrophils, and serum albumin) using exploratory statistical analyses.

Results Ten patients were included (mean age 69 years; 80% male), with a causative organism identified in 2/10 (20%) of cases. Significant associations were observed between CT features and inflammatory or biochemical markers. Lung abscess was associated with higher CRP (p=0.049). Loculation and consolidation were associated with higher WCC (p=0.029 and p=0.017, respectively) and neutrophil counts (p=0.033 and p=0.036, respectively). Loculation was associated with lower serum albumin (p=0.015) and higher PF LDH (2450 vs 980 IU/L; p=0.04), see figure 1. No other significant biochemical differences were observed across CT features.



Abstract M16 Figure 1 Heat map of significant associations between chest CT features and biological markers in pleural infection. Exploratory study, n 10

Conclusion This exploratory study provides proof of concept for a CT-based scoring system for pleural infection. It identifies associations between chest CT features and biological markers of inflammation and disease severity. Larger prospective studies are required to validate these findings and to determine whether CT-based phenotyping could contribute to risk stratification and personalised management in pleural infection.

M17 MANAGEMENT OF PRIMARY SPONTANEOUS PNEUMOTHORAX BEFORE AND AFTER IMPLEMENTATION OF 2023 BTS PLEURAL DISEASE GUIDELINES: EXPERIENCE FROM A TERTIARY CENTRE

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Introduction The British Thoracic Society (BTS) 2023 Pleural Disease Guidelines advocate conservative management for primary spontaneous pneumothorax (PSP) in patients who are minimally symptomatic and without high-risk features.[1] This contrasts with previous recommendations favouring early intervention, especially for larger pneumothoraces.[2] There are limited data evaluating the real-world adoption of these updated guidelines and their impact on PSP management.

Aim To compare management of patients presenting with a first episode of PSP before and after implementation of the 2023 BTS Pleural Disease Guidelines, focussing on initial treatment decisions and follow-up pathways.

Methods A coding search of 'pneumothorax' (ICD-10 code: J93) was performed for all patients attending a tertiary centre between 01.01.2020 and 31.08.2024. Electronic records of 1,772 patients were retrospectively reviewed to identify adults (≥18 years) presenting with a first episode of PSP. Patients with traumatic, iatrogenic, known secondary or recurrent pneumothoraces, as well as those transferred from another centre, were excluded.

Results 72 patients were identified, including 55 before and 17 after publication of the 2023 BTS pleural disease guidelines (11.07.2023). Demographic and clinical information are summarised in table 1. The number of pleural interventions performed on first presentation reduced from 55% to 24% between cohorts, with a substantial reduction in use of intercostal chest drains (40% to 18%) and needle aspiration (15% to 6%). Most patients (>87%) had appropriate follow-up arranged, though <50% were via dedicated respiratory services. The mean time to follow-up was 17 days for both patient cohorts. Advice regarding avoidance of high-risk activities (e.g. flying, diving) after pneumothorax was inconsistently documented (60% and 53% respectively).

Conclusions Management of PSP at our centre has shifted towards a more conservative approach, in line with current guidance. However, substantial variation in follow-up exists, with many patients not reviewed within recommended timeframes, or in dedicated respiratory clinics. Improved follow-up pathways could enhance patient education, identification of modifiable risk factors (e.g. smoking, cannabis, and vape use) and recurrence prevention. Development of standardised local

protocols is warranted to ensure consistent and optimised delivery of care.

Abstract M17 Table 1 Baseline demographics, clinical characteristics and management strategies

	Pre 2023 BTS guidelines	Post 2023 BTS guidelines
Clinical characteristics		
Number of patients	55	17
Sex		
- Male	43 (78%)	13 (76%)
- Female	12 (22%)	4 (24%)
- Age (years)*	30.3 (1.4)	29 (12.5)
Smoking History		
- Yes	32 (58%)	4 (23%)
- No	17 (31%)	12 (71%)
- Not recorded	6 (11%)	1 (6%)
Cannabis History		
- Yes	14 (26%)	4 (24%)
- No	15 (27%)	5 (29%)
- Not recorded	26 (47%)	8 (47%)
Vaping History		
- Yes	5 (9%)	2 (12%)
- No	7 (13%)	3 (18%)
- Not recorded	43 (78%)	12 (71%)
Time to presentation (days)*	2 (3.1)	2.8 (7)
Management		
Initial intervention		
- Aspiration	8 (15%)	1 (6%)
- Chest drain	22 (40%)	3 (18%)
- None (conservative)	25 (45%)	13 (76%)
Subsequent intervention performed		
- Aspiration	2 (4%)	1 (6%)
- Chest drain	6 (11%)	2 (12%)
- Suction via chest drain	14 (25%)	0 (0%)
- Cardiothoracic surgery	7 (13%)	0 (0%)
- None	35 (64%)	15 (88%)
Location		
- ED	15 (27%)	5 (29%)
- Respiratory ward	20 (36%)	2 (12%)
- Non-respiratory ward	8 (15%)	3 (18%)
- SDEC	12 (22%)	7 (41%)
Advice provided (e.g. flying, diving)		
- Yes	33 (60%)	9 (53%)
- No	22 (40%)	8 (47%)
Follow-up		
- Arranged	48 (87%)	15 (88%)
- Respiratory clinic	18 (38%)	8 (53%)
- SDEC	13 (27%)	3 (20%)
- Respiratory in SDEC	8 (17%)	4 (27%)
- Cardiothoracic surgery	3 (6%)	0 (0%)
- Did not attend	6 (12%)	0 (0%)
- Not arranged	7 (13%)	2 (12%)
- Time to follow-up (days)*	17 (18)	17 (34)
Recurrence of pneumothorax		
- Yes	7 (13%)	4 (23%)
- No	48 (87%)	13 (76%)
Time to recurrence (days)*	213 (116)	172 (235)

*Mean (+SD). *SDEC*, same day emergency care; *ED*, emergency department

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M18 CLINICAL OUTCOMES OF PLEURAL INFECTION IN A TERTIARY RESPIRATORY CENTRE DESPITE HIGH ADHERENCE TO BRITISH THORACIC SOCIETY GUIDELINES FOR PLEURAL DISEASE

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10.1136/thorax-2026-BTSabstracts.464

Pleural infection remains associated with substantial morbidity, mortality and healthcare utilisation. The 2023 British Thoracic Society (BTS) Pleural Disease Guidelines provide evidence-based recommendations for investigation and management. We evaluated adherence to BTS standards at a tertiary respiratory centre and assessed clinical outcomes, including length of hospital stay, mortality rate, escalation to Intrapleural Enzyme Therapy (IET), and surgery, among patients admitted with pleural infection.

Methods We conducted a retrospective audit of consecutive patients admitted with pleural infection to a tertiary respiratory ward between January and June 2025. Demographic, laboratory, chest Computed Tomography (CT), procedural, microbiological, and clinical outcomes data were collected from electronic patient records. Audit standards were based on 2023 BTS pleural disease guidelines.

Results Thirty-seven patients were included; 65% (24/37) were male. Median age was 64 (55,80), median length of hospital stay was 13.0 days (IQR 9.0–22.0). Adherence to advocated investigations was high, with pleural fluid pH recorded in 100% (37/37), chest radiography performed within 24 hours in 94.6% (35/37), thoracic ultrasound performed in 91.9% (34/37), and CT imaging in 86.5% (32).

Chest drain was placed in 86.1% (31/37) of patients. Of these, IET was required in 18.9% (7/37), while 35.1% (13/37) required surgical interventions, indicating a substantial burden of advanced disease. In-hospital mortality was 18.9% (7/37), and 30-day readmission occurred in 8.1% (3/37).

Length of stay varied according to RAPID risk category, with a median duration of 12.5 days (IQR, 9.8–18.2) among low-risk patients, 11.0 days (IQR, 9.0–17.2) and 27.0 days (IQR, 24.5–29.0) among intermediate and high-risk groups, respectively (P=0.08).

Conclusion This audit demonstrated excellent adherence to BTS guidelines for the recommended investigation and management of pleural infection. However, pleural infection continued to be associated with prolonged hospitalisation, frequent treatment escalation to IET or surgery, and substantial mortality. These findings highlight the persistent burden of the disease and the need for more effective management strategies.

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M19 WAITING TIMES TO MEDICAL THORACOSCOPY AND ITS IMPACT ON OUTCOMES: A RETROSPECTIVE DESCRIPTIVE STUDY AT A TERTIARY CARE RESPIRATORY CENTER

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10.1136/thorax-2026-BTSabstracts.465

Local anaesthetic medical thoracoscopy (MT) is a vital diagnostic and therapeutic procedure in pulmonology. We evaluated waiting times, diagnostic yield, and outcomes of MT in a tertiary service.

A retrospective cohort study was conducted on the patients who underwent Semirigid MT from 01/03/2022 to 28/02/2025 at a tertiary care center. Time from the 1st presentation to MT, and time from the referral to MT were evaluated. Procedural, histological, and follow-up outcomes were extracted from the MT registry and patient records.

77 patients underwent MT (69% male, age range 18-97); 70.1% were external referrals. Mean time from 1st presentation to MT was 64 days; mean time from referral to MT was 18 days, irrespective of internal/external.

From the date of presentation, 37.7% had MT within 1 month, and 67.5% had it within 3 months. From the date of referral, 62.3% had MT within 14 days and 77.9% had MT within 30days.

MT was diagnostic in 93.5% of patients, and 6.5% required Video-Assisted Thoracoscopic Surgery (VATS). Considering the diagnoses, 31.1% had malignant mesothelioma. 27.3% had other malignancies. 37.7% had fibro-inflammatory changes, and 3/77 (3.9%) had Tuberculosis.

Regarding effusion management, 22% underwent pleurodesis, and 13% had pleurodesis plus an Indwelling Pleural Catheter (IPC). 15.6% had delayed IPC. 45.5% improved with just MT or MT + management of the cause. MT 28-day mortality is 0%, and one-year survival is 78%.

22% (N=17) patients expired during the 1st year, and 41.2% of them had mesothelioma, 41.2% had other cancers, and 17.6% didn't have cancer.

Local anaesthetic MT had a high diagnostic yield (93.5%), excellent safety with zero 28-day mortality, and 78% Survival after one year. Referral-to-MT time averaged 17 days from referral to the procedure, but the first presentation-to-procedure time was considerable. However, this delay did not alter the outcomes of our cohort. In the future, we aim to assess the reasons for delays in referral to MT and how they affect patient outcomes.

M20 INDWELLING PLEURAL CATHETER VERSUS MEDICAL PLEURODESIS IN MALIGNANT PLEURAL EFFUSIONS: UK-BASED SINGLE-CENTRE EXPERIENCE OVER 3 YEARS

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Introduction Indwelling pleural catheter (IPC) and talcpleurodesis (TP) are established interventions for malignant pleural effusion (MPE). They improve symptom burden and quality of

Abstract M20 Table 1 Patient demographics and outcomes between TP and IPC cohorts

	TP	IPC
Primary malignancy	N=38 (%)	N=59 (%)
Non-small cell lung cancer	13 (34.2)	17 (28.8)
Small cell lung cancer	3 (7.9)	0 (0)
Lung primary with no histology	1 (2.6)	0 (0)
Mesothelioma	0 (0)	14 (23.7)
Breast	6 (15.8)	9 (15.3)
Renal	0 (0)	4 (6.8)
Upper gastrointestinal	2 (5.3)	4 (6.8)
Haematology	1 (2.6)	3 (5)
Prostate	4 (10.5)	3 (5)
Gynaecology	4 (10.5)	2 (3.4)
Head and neck	2 (5.3)	1 (1.7)
Peritoneal	1 (2.6)	0 (0)
Melanoma	0 (0)	1 (1.7)
Cancer of unknown origin	1 (2.6)	0 (0)
Mean performance status	2	1.73
Mean age (years [range])	69.7 [43-94]	72.2 [33-92]
Gender		
Male	18 (47.4)	32 (54.2)
Female	20 (52.6)	27 (45.8)
Prognostic scoring		
Mean LENT score	4.4	3.0
Mean PROMISE score	27.7	21.0
Inpatient encounters		
Procedure performed during emergency admission	38 (100%)	8 (13.6%)
Mean length of stay during emergency admission (days [range])	13.8 [3-45]	14.9 [7-42]
Procedure related readmissions	9 (23.7%)	12 (20.3%)
Failed medical pleurodesis	9 (23.7%)	
Mean days from TP to failed pleurodesis	100 days	
Further IPC insertion	4 (10.5%)	
Further intercostal drain/aspiration	4 (10.5%)	1 (1.7%)
IPC outcomes		
IPC removal		14 (23.7%)
Fibrinolysis		2 (3.4%)
Autopleurodesis		11 (18.6%)
Mean days to autopleurodesis from insertion of IPC [range]		123.3 [45-394]
Complications	n (%)	n (%)
Pleural infection	0 (0)	5 (8.5)
Pneumonia	1 (2.6)	0 (0)
Pain and discomfort	13 (34.2)	8 (13.6)
Fever	2 (5.3)	0 (0)
Re-accumulation of effusion	9 (23.7)	0 (0)
Blockage	1 (2.6)	3 (5.1)
Dislodgement	1 (2.6)	2 (3.4)

Loculations	N/A	6 (10.2)
Mental health impact	1 (2.6)	2 (3.4)
Mean survival post procedure (days [range])	106 [4-782]	133 [9-682]

life with no significant difference between the two interventions inpatient-reported global health status at 30 days.[1] We aimed to compare our real-world experience of both procedures at a district general hospital.

Methods A retrospective review was conducted of patients undergoing IPC insertion and TP for MPE between April 2022 and September 2025.

Results 38 patients received TP via chest drain; 59 underwent IPC insertion. 51 IPCs were performed electively, while all TP occurring during inpatient admissions.

Procedure-related readmission rates were similar (TP 23.7% vs. IPC 20.3%). Pleurodesis failed in 9 TP cases (23.7%). 8 further pleural procedures were performed in the TP cohort, compared to 1 in the IPC cohort. 14 patients underwent IPC removal, and 2 received fibrinolytics via IPC to aid drainage.

Most readmissions post TP were associated with re-accumulation of effusion. Community challenges in the IPC cohort were identified during further hospital encounters, including suture non-removal and issues surrounding home drainage. Complications are outlined in table 1. There was no significant difference between mean survival post intervention between the cohorts (TP 106 vs. IPC 127 days, p=0.4230).

8 IPCs were performed during emergency admissions. The difference in mean length of stay (LOS) during emergency admissions was insignificant (TP 13.8 vs. IPC 14.9 days, p=0.7907). There was no correlation between performance status and LOS.

Conclusion Both interventions were effective in patients with MPE. A benefit of IPC insertion is that the majority were performed as day procedures, whereas TP cohort required an average LOS of 2 weeks. Interestingly inserting an IPC during an emergency admission did not result in reduced LOS comparatively. Overall, readmission rates were similar between the groups.

Each procedure carries a different complication profile. Pleural infection and loculations were reported in patients with IPC, whilst more patients required further pleural procedures post TP. Management of MPE should be individualized with emphasis on informed consent and patient preference.

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M21 LOCAL MIST- FOGGIER THAN EXPECTED: A REAL WORLD VIEW ON MANAGEMENT OF PLEURAL INFECTION

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10.1136/thorax-2026-BTSabstracts.467

Introduction Pleural infections are associated with high morbidity and mortality. Intrapleural fibrinolytic therapy used in pleural infection was shown to reduce surgical referrals and reduce hospital stays in MIST2. It was subsequently incorporated into the BTS pleural disease guidelines in 2023. Further trials are currently ongoing with MIST4 comparing intrapleural fibrinolysis with thoracic surgery in management of pleural infection. We wanted to review, since the 2023 guidelines, how differing treatment approaches fared in real life outside of trial conditions.

Methods We carried out a data search of patients admitted to our unit from 2023 to 2026 to identify chest drains inserted for pleural infection. A total of 48 patients were managed with chest drain insertion. Of those 5 patients received fibrinolytic therapy, 5 patients underwent VATS procedures with the remainder managed with chest drain alone. We gathered information from electronic medical notes and laboratory results of these patients. The primary outcome of this review was resolution which was defined radiologically complete resolution. There were multiple secondary outcomes mainly focused on predictive parameters for resolution.

Results We found that those who were managed with fibrinolysis and received the full recommended course had a 100% resolution rate (2/2). Patients who received only partial treatment with fibrinolytics had very similar outcomes to those managed with chest drain alone, with only a 33% resolution rate. Patients who underwent cardiothoracic surgery had a resolution rate of 60%. We found lower pH and glucose levels to be strong predictors of worse outcomes.

Conclusion Our review highlighted the difficulties of managing patients with pleural infection using real world data. Small numbers of patients underwent surgical approaches and only a small number underwent fibrinolysis due to a variety of barriers. Although small numbers, our findings indicate that intrapleural fibrinolysis (when treatment course completed) can improve resolution rates of pleural infection. Also, pH and glucose levels are strong predictors of poor outcomes and can be useful to identify patients who may benefit from fibrinolytics. Cardiothoracic surgery also improved resolution rates however the numbers reviewed are too small to directly compare the two treatment modalities.

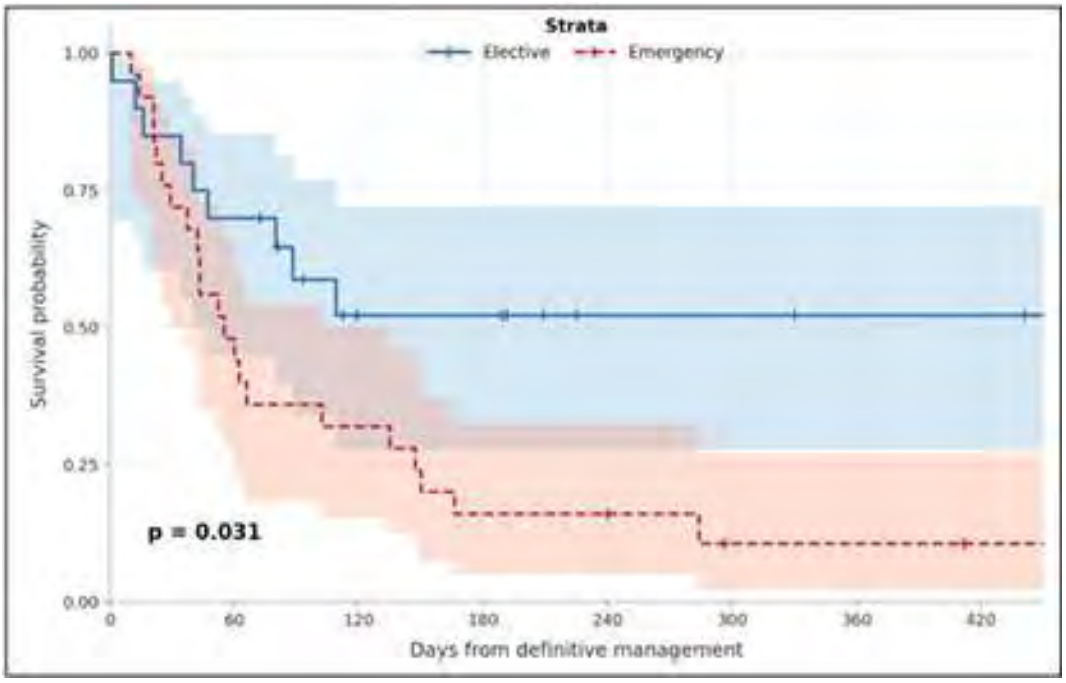
M22 DO ELECTIVE ADMISSION PATHWAYS IMPROVE OUTCOMES IN DEFINITIVE MANAGEMENT OF MALIGNANT PLEURAL EFFUSION? A RETROSPECTIVE COHORT STUDY

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10.1136/thorax-2026-BTSabstracts.468

Background Malignant pleural effusion (MPE) is a complication of malignancy with poor prognosis and high symptomatic burden, often requiring prolonged hospitalisation in patients with diminished life expectancy. While methodology of definitive management is well established, limited research compares elective versus emergency admissions for definitive management. This study evaluates the impact on overall length of hospital admission and survival between groups.

Methods A retrospective cohort study was conducted on inpatients who were considered for definitive management of MPE between March 2025 and March 2026. Patient pathways were mapped from initial presentation to definitive pleural management, then followed up at time of at time of audit (June 2026) for survival analysis. Patients were grouped into elective (planned procedures such as talc pleurodesis or placement of indwelling



Abstract M22 Figure 1 Kaplan-Meier survival curves from date of definitive management by admission pathway

pleural catheter) and emergency presentations requiring acute management with opportunistic definitive management while inpatient. The primary outcomes were total length of admission and survival time from definitive management; secondary outcomes included complication rates and time to procedure.

Results 50 patients were included, with 21 in the elective admissions group and 29 in the emergency group. Patients had a mean hospital stay of 6.2 days and 10.9 days retrospectively. Survival data was calculated in patients with completed definitive management (n=45) from date of management and Kaplan-Meier survival curves were plotted (figure 1). Mean survival following definitive pleural intervention was 254 days (95% CI 165–344 days) in the elective group and 114 days (95% CI 63–166 days) in the emergency group. Complication rates were 48% in elective and 62% in emergency groups.

Discussion Our findings suggest that elective definitive management of MPE is associated with a shorter length of admission in comparison to emergency-based treatment. Elective admission for definitive management is also associated with prolonged survival at follow-up than in emergency admission, with survival curves that separated early and remained distinct throughout follow-up, suggesting that patients entering a planned pleural management pathway may experience more favourable outcomes than those presenting acutely. These results highlight the potential importance of access to early management pathways in pleural disease, and further analysis addressing this comparison is now needed.

M23 INDWELLING PLEURAL CATHETERS FOR THE MANAGEMENT OF PERSISTENT AIR LEAKS: A RETROSPECTIVE REVIEW

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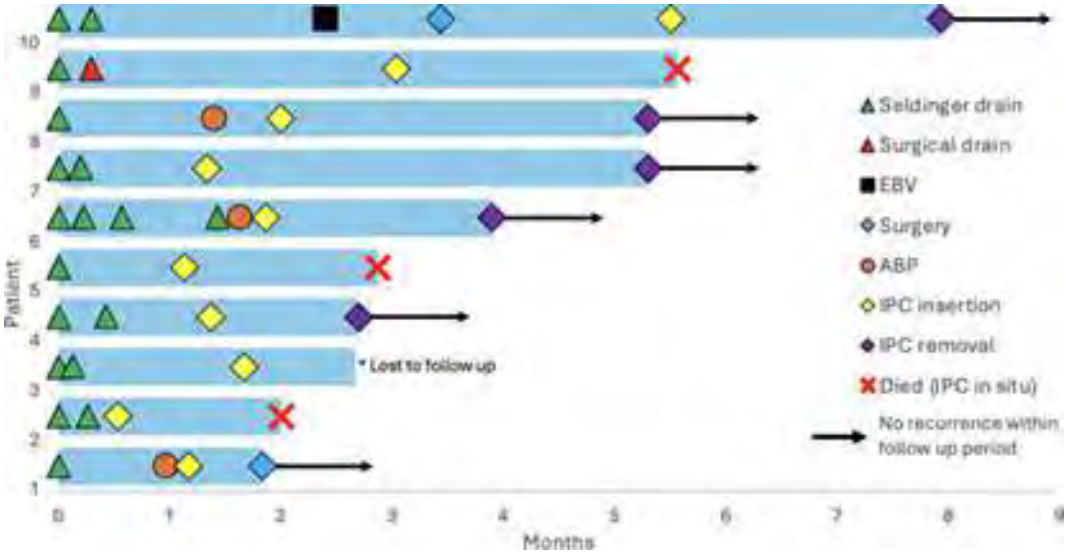
10.1136/thorax-2026-BTSabstracts.469

Introduction Pneumothorax complicated by persistent air leak (PAL) results in prolonged hospital stays and high mortality, with patients often unfit for surgical intervention. Whilst minimal data exists for the use of indwelling pleural catheters (IPC) in PAL, our centre has accumulated experience of their use.

Methods A retrospective review was performed of IPCs inserted for PAL of any aetiology between 01/01/2020 to 31/12/2025 at a tertiary institution. Data was collected from presentation to 6 months post IPC insertion.

Results Ten patients underwent IPC insertion with a median age of 71.5 years (IQR 57-79), 5/10 had a WHO PS of 3, 4/10 had a PS of 2. Eight were secondary spontaneous pneumothorax, 1 was trauma related and 1 iatrogenic. All patients were referred for surgical opinion. Management strategies included intercostal drain (ICD) insertion (10/10), negative pressure drainage (10/10), ambulatory device via ICD (5/10), autologous blood patch (3/10), thoracic surgery (2/10) and endobronchial valves (1/10). Median time to IPC insertion was 46 days (range 6–165), median inpatient days for the index admission was 56 (22–178), including 38 days prior to IPC insertion (12–165) and 8 days thereafter (1–44). All patients had ambulatory devices attached to the IPC, enabling discharge in 8/10 patients. Full resolution was seen in 5/10, with IPC removal after a median of 73 days (40-119), with no recurrence. Complications were seen in 6/10 with 4/10 requiring admission, including: empyema (4/10), enlarging pneumothorax (2/10), iatrogenic lung injury conservatively managed (1/10), minor surgical emphysema (1/10). No drain dislodgements occurred. Three patients died with IPC in situ, of which two were due to underlying disease progression and one from empyema related to the IPC.

Discussion IPCs appear to be a feasible option for management of PAL, facilitating discharge and effective palliation in patients where other strategies have failed, with low re-intervention rates and the added security of a tunnelled device to reduce drain dislodgement. High readmission and complication rates were observed in this frail cohort with prolonged drain placement. Further studies are required to determine whether these



Abstract M23 Figure 1 Swimmer plot demonstrating the timing of procedures performed for persistent air leak management, from presentation to 6 months post IPC insertion

rates differ from those associated with ambulatory management using ICDs.

M24 CELL-FREE DNA IN SUSPECTED MALIGNANT PLEURAL EFFUSION: DIAGNOSTIC AND MOLECULAR APPLICATIONS

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10.1136/thorax-2026-BTSabstracts.470

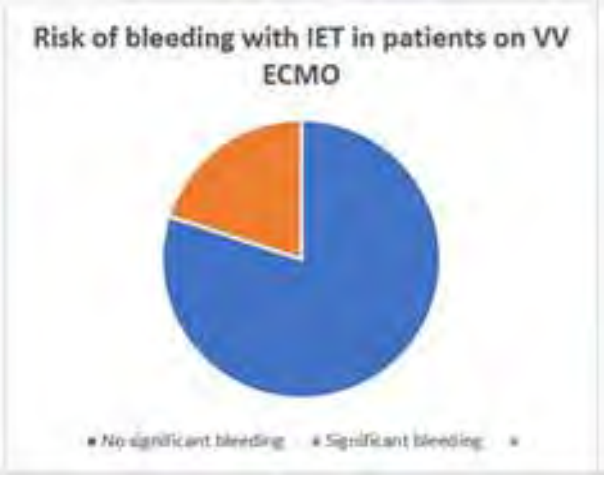
Background Cell-free DNA (cfDNA) has emerged as a minimally invasive biomarker with potential in diagnosis, molecular characterisation and monitoring of thoracic malignancies. Pleural fluid represents a biologically enriched and clinically accessible compartment; however, whether pleural cfDNA can augment existing diagnostic approaches remains unclear.
Objectives To review the evidence evaluating pleural cfDNA in thoracic malignancy.
Methods A literature search of Ovid Embase and Medline was performed. Of the 55 identified records screened, 14 met the pre-defined inclusion criteria. Substantial heterogeneity in study design, patient populations, and outcome measures, precluded meta-analysis. A secondary scoping approach was undertaken to map the evidence, synthesise findings narratively and identify methodological limitations and research priorities.
Results The review included 114 patients. Findings suggest that pleural cfDNA and cfRNA enable detection of actionable driver mutations and resistance alterations and may demonstrate higher diagnostic yield than cytology-derived cell blocks. They also show plausibility for prognostication and disease monitoring. However, most studies were small, single-centre and methodologically heterogeneous, with limited longitudinal follow-up and inconsistent comparator pathways.
Conclusions Pleural cfDNA represents a promising complementary biomarker in thoracic malignancy, but current evidence is insufficient to define clinical effectiveness or optimal pathway integration. Secondary scoping highlights the need for prospective, pathway-embedded studies to determine whether it can improve diagnostic accuracy, accelerate molecular stratification and inform disease monitoring beyond existing cytology-, tissue- and plasma-based approaches.

M25 PRACTICE OF INTRAPLEURAL FIBRINOLYTIC AND ENZYME THERAPY (IET) IN ADULT PATIENTS ON VENO-VENOUS EXTRACORPOREAL MEMBRANE OXYGENATION (VV ECMO) - A CASE SERIES

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10.1136/thorax-2026-BTSabstracts.471

Introduction Combination Intrapleural fibrinolytic and enzyme therapy (IET) is a guideline-recommended intervention for pleural infection, particularly in patients deemed unfit for surgery.[1] However systemic anticoagulation, required for VV-ECMO, poses an increased bleeding risk.[2] This study evaluates the use of IET within a tertiary ECMO centre.
Methods A retrospective cohort study was conducted, including adult VV- ECMO patients receiving IET for pleural infection between 01/01/24 to 31/12/25. Data on dosing, anticoagulation, and clinical outcomes were extracted from electronic health records. Significant bleeding was defined as causative for the need for red blood cell transfusion and / or interventional procedure, as judged by review of the clinical record.
Results Six patients (mean APACHE II score 22, mean RAPID score 3) received IET while on VV- ECMO. Most patients (n=5) received a reduced-dose regimen, most commonly 2.5 mg Alteplase and 5 mg Dornase alfa. All patients were on heparin infusions; this was maintained without interruption during the full IET course in four patients and temporarily held for alternative reasons in two. One patient had significant bleeding, managed with interventional radiology embolisation and subsequent surgery. Five patients (83%) showed radiological improvement in their pleural infection, and five patients (83%)



Abstract M25 Figure 1 Clinically significant bleeding was defined as causative for the need for red blood cell transfusion and / or interventional procedure, as judged by review of the clinical record

were successfully discharged; one death occurred due to underlying respiratory failure unrelated to IET.

Discussion This study is the first to evaluate IET for pleural infection in adult VV- ECMO patients. Whilst a small series, it shows that most patients who received IET whilst on VV-ECMO did not have significant bleeding and 83% survived to discharge. This supports IET as a therapeutic option for the treatment of pleural infection in adult VV- ECMO patients and may also aid decisions regarding IET in other cohorts where interrupting anticoagulation poses significant risks.

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M26 REVIEWING NATIONAL PLEURAL SERVICES THROUGH A SAFETY-II LENS: RECOMMENDATIONS FOR THE IDEAL PLEURAL UNIT

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10.1136/thorax-2026-BTSabstracts.472

British Thoracic Society Quality Standards for Pleural Disease[1] suggest a shift is required in how pleural services are managed nationwide. National audits have focused on serious harm incidents and patient safety concerns.[2] Learning from error is critical, however it is through studying success that true best practice is established in our ever-changing healthcare system. Safety-II thinking refers to the assessment of what goes well, rather than focusing on what goes wrong. We used this approach to evaluate pleural services across the UK.

We conducted a survey of pleural specialists across the UK in January 2026, asking them to describe the remits of their service. Specialists were contacted through the BTS Pleural Disease Advisory Group and NHS email, inviting them to complete a qualitative survey.

We gathered 42 responses. This allowed the consideration of what could constitute the 'ideal' pleural unit, with themes represented in figure 1. Undoubtedly, excellent practice occurs in several UK services. Notably, 11/42 respondents reported offering 24:7 Respiratory physician cover. 21/42 responses reported the inclusion of pleural nurse specialists, and 21/42 offer pleural clinics. Training is key, and 20/42 have regular teaching in



Abstract M26 Figure 1

chest drains for general medical teams to promote local upskilling, with 3 respondents offering specific simulation sessions. 8/42 have dedicated job plan time for thoracic ultrasound training, and 8 respondents detailed the ability to store ultrasound imaging nationally. 27 teams reported engaging in local quality improvement. Obvious disparities highlight the importance of development in this area.

The information gathered by this review of current practice not only demonstrates what excellence can look like, but serves as an aspirational blueprint for healthcare professionals aiming to enhance their pleural service and the care patients receive.

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M27 HIGHER INTERCOSTAL DRAIN DISLODGEMENT RATES OUTSIDE RESPIRATORY MEDICINE: A MULTICENTRE SERVICE EVALUATION

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Background Intercostal drain (ICD) insertion is performed by clinicians across multiple specialties; however, structured pleural training, competency assessment and procedural governance are most established within respiratory medicine through British Thoracic Society (BTS) guidance and dedicated pleural services. ICD dislodgement is a potentially avoidable complication associated with treatment failure, repeat procedures and prolonged hospitalisation. We evaluated whether ICD dislodgement rates differed according to operator specialty across three centres.

Methods A retrospective multicentre service evaluation was conducted across three tertiary UK hospitals between July and December 2025. Consecutive ICD insertions were reviewed and operators categorised as respiratory or non-respiratory clinicians. The primary outcome was ICD dislodgement, defined as documented drain slippage or complete drain fall-out. Secondary outcomes included requirement for repeat intervention.

Results Sixty-nine ICD insertions were included. Patients were predominantly male (51/69, 74%), with a median age of 64 years (range 18–101 years). Respiratory clinicians performed 47/69 (68%) procedures, while 22/69 (32%) were undertaken by non-respiratory clinicians, including the Emergency Department (14/22), Medical Assessment Unit (7/22) and Intensive Care Unit (1/22).

Overall, ICD dislodgement occurred in 9/69 (13%) cases. Among ICDs inserted by non-respiratory clinicians, dislodgement occurred in 6/22 cases (27%), compared with 3/47 cases (6.3%) following insertion by respiratory clinicians (RR 4.27, 95% CI 1.18–15.52; p=0.025).

All six dislodged drains in the non-respiratory group were 12F drains and required respiratory team review, with five requiring drain reinsertions. Reinsertions were performed for four pneumothoraces and one pleural effusion.

Among ICDs inserted by respiratory clinicians, three episodes of drain slippage occurred, none of which required reinsertion. All drain reinsertions occurred following ICD insertion outside respiratory medicine services.

Conclusions ICD dislodgement was over four-fold more common following insertion by non-respiratory clinicians than respiratory clinicians (27% vs 6.3%; RR 4.27, 95% CI 1.18–15.52; p=0.025). All cases requiring reinsertion occurred outside respiratory medicine services. These findings highlight variation in procedural outcomes across specialties and support wider adoption of standardised pleural training, competency assessment, drain fixation practices and post-procedural governance to improve patient safety and reduce avoidable repeat procedures.

M28 OUT-OF-HOURS PLEURAL PROCEDURES: A UK NATIONAL SURVEY OF GENERAL MEDICAL, EMERGENCY MEDICINE AND INTENSIVE CARE REGISTRARS

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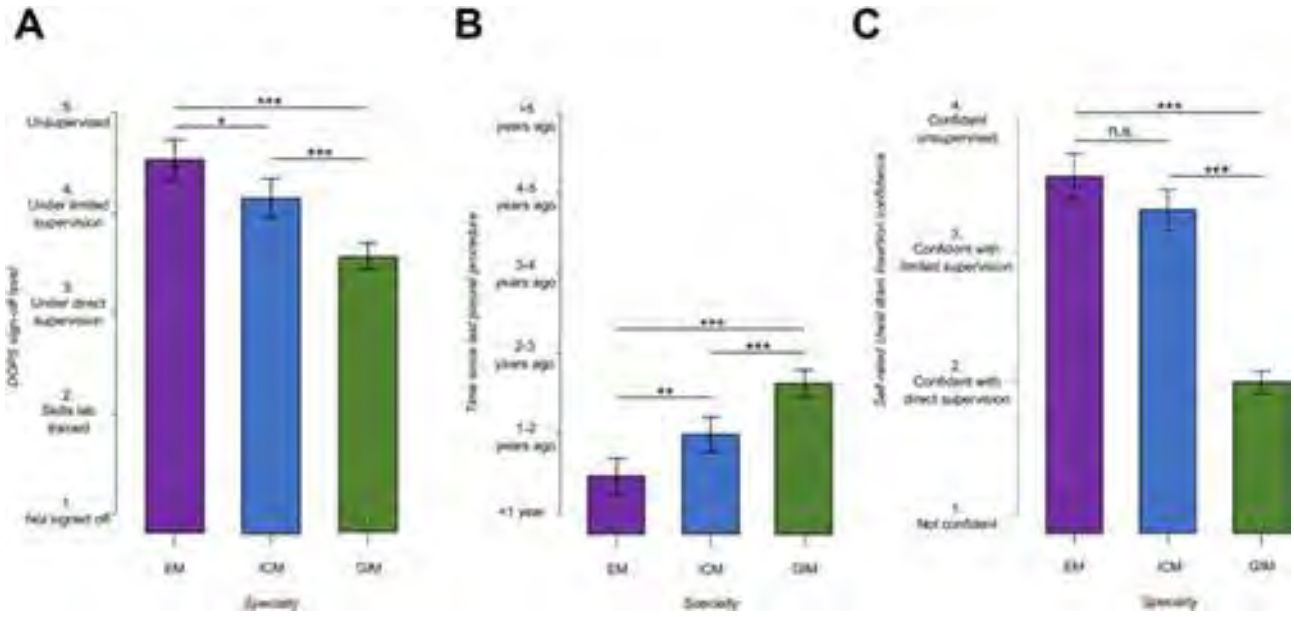
10.1136/thorax-2026-BTSabstracts.474

Introduction Pleural procedures are occasionally required out-of-hours (OOH) and performed by registrars in general internal medicine (GIM), emergency medicine (EM), and intensive care medicine (ICM). As pleural interventions are increasingly undertaken in-hours by specialist teams, there is growing concern regarding declining procedural exposure among the wider OOH workforce. Pleural procedures carry a risk of significant harm. We surveyed GIM, EM and ICM registrars to explore their roles, perceived confidence and competence in OOH pleural procedures.

Methods A UK-wide anonymised survey was disseminated to GIM, EM, and ICM registrars.

Results Responses were received from 391 non-respiratory GIM, 83 EM and 159 ICM registrars, representing all UK deaneries and training grades. Respondents reported their procedural 'sign-off' level attained in Direct Observation of Procedural Skills (DOPS) assessments. EM registrars were most often 'signed-off' as independent in chest insertion for pneumothorax (71%), followed by ICM (60%), then GIM (37%). Recent pleural procedural experience within the past year was highest among EM registrars (76%), compared with ICM (53%) and GIM (31%). EM registrars most frequently reported previous OOH pleural procedural experience (71%), followed by ICM (64%), then GIM (27%). Self-perceived confidence in independently performing OOH chest drain insertion for pneumothorax was highest among EM registrars (73%), followed by ICM (61%), then GIM (11%). Beyond specialty, chest drain confidence was associated with higher DOPS 'sign-off' level, more recent procedural experience, and prior OOH procedural experience. Multivariable linear regression showed that all of these factors, including specialty, remained independently associated with confidence. Themes in the free text included safety concerns, deskilling, evolving roles of specialty registrars, and responsibility for OOH pleural procedures.

Conclusions Pleural procedural confidence, recent experience, and DOPS 'sign-off' level varied notably across specialties,



Abstract M28 Figure 1 Pleural procedural experience and confidence among EM, ICM, and GIM registrars. (A) Distribution of DOPS sign-off levels for Seldinger chest drain insertion for pneumothorax. (B) Distribution of time since last pleural procedure. (C) Self-rated confidence in out-of-hours Seldinger chest drain insertion for pneumothorax. n=633. (*p< 0.05, ** p < 0.01, *** p < 0.001)

being higher among EM and ICM registrars and lower among GIM. This may reflect broader changes in the distribution of pleural procedural expertise across the OOH workforce, with implications for patient safety, specialty training, and the delivery of local OOH pleural procedural cover.

M29 UK PLEURAL PRACTICE REVIEW – STILL NOT GETTING IT RIGHT

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10.1136/thorax-2026-BTSabstracts.475

Pleural disease is common and increasing in incidence. Its diagnosis and management often require invasive pleural procedures. 13 years from the original National Patient Safety Agency alert on the insertion of chest drains, a British Thoracic Society (BTS) pleural services organisational audit in 2021 continued to highlight the ongoing risk of avoidable harm from pleural procedures. To improve care for patients with pleural disease, multiple national documents have outlined the minimum standards required around pleural procedures. Routine use of ultrasound for pleural procedures is now standard within respiratory departments, but real-life experience reveals large variations in infrastructural support between centres.

Aim To evaluate the current operational aspects of pleural practice in the UK.

Abstract M29 Table 1 Summarising the pleural services and infrastructural support offered by different centres within the UK

	Overall	Centre type	
	n=39	Tertiary n=16	DGH n=23
Centre type		N/A	
Tertiary	16 (41%)		
DGH	23 (59%)		
Pleural service offered by respiratory department			
Pleural aspiration	39 (100%)	16 (100%)	23 (100%)
Chest drain (seldinger)	38 (97%)	16 (100%)	22 (96%)
Chest drain (blunt dissection)	14 (36%)	7 (44%)	7 (30%)
IPC insertion	33 (85%)	16 (100%)	17 (74%)
US guided pleural biopsy	14 (36%)	10 (63%)	4 (17%)
Local anaesthetic thoracoscopy	17 (44%)	8 (50%)	9 (39%)
Ambulatory pathway for PTX	12 (31%)	7 (44%)	5 (22%)
Pleural CNS/ACP	20 (51%)	10 (63%)	10 (43%)
Dedicated space for inpatient pleural procedures	23 (59%)	10 (63%)	13 (57%)
Majority of inpatient pleural procedures performed at bedside	21 (54%)	8 (50%)	13 (57%)
Dedicated assistant for pleural procedures (outpatient) n=37	33 (89%)	15 (94%)	18 (86%)
Dedicated assistant for pleural procedures (inpatient)	6 (15%)	3 (19%)	3 (13%)

Method Respiratory registrars were invited to complete a Google Forms survey on the pleural practice and resources at their centre in March 2026. Information gathered included centre location and type, pleural services offered by respiratory department, pleural specialist nurse (CNS) presence, and space and support for pleural procedures.
Result Responses were received from 39 hospitals. 60% were from district general hospitals. Table 1 summarises the spread of pleural services offered by respiratory departments. Although 85% of centres offered IPC insertion, only 51% had pleural CNS support. 41% of centres have no dedicated space for inpatient pleural procedures, with 54% routinely performing these by the bedside. Dedicated pleural assistance, including specialist or ward nurse availability, for inpatient procedures was present in 15%. 85% of responders had to seek assistance from 'anyone they can grab' or 'no one'.
Discussion Infrastructural support to deliver safe care for pleural patients is lacking. No headway has been made compared to the BTS national procedures audit in 2010 when it comes to avoiding bedside pleural procedures. The care of inpatients, arguably the more frail and unwell population, is particularly at risk with minimal formal assistance available. The ongoing practice of performing routine pleural procedures at the bedside, without suitable assistance, has significant safety, healthcare, educational, and opportunity cost implications. Urgent action is warranted at a national level to support the development of local pleural infrastructure and ensure equitable care for all pleural patients.

M30 CONFIDENT AT THE BEDSIDE, UNSUPPORTED BY THE SYSTEM: A MULTI-CENTRE SURVEY OF AWARENESS, TRAINING AND SELF-REPORTED PRACTICE IN PLEURAL PROCEDURES ACROSS 17 EAST OF ENGLAND HOSPITALS FOLLOWING THE 2023 BTS GUIDANCE

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10.1136/thorax-2026-BTSabstracts.476

Introduction and Objectives The 2023 BTS Pleural Disease Guideline and Clinical Statement on Pleural Procedures reset standards for thoracic ultrasound, operator competency, consent and governance. Whether these have reached frontline practice is unknown, particularly outside respiratory teams and tertiary centres. We surveyed awareness, training, access and self-reported practice against key recommendations across acute hospitals in the East of England.
Methods A 29-item electronic survey was distributed to Internal Medicine Training trainees and Foundation doctors across the East of England, with higher specialty trainees and consultants engaged through site leads at each participating hospital (October 2024 to April 2026). Items captured grade, specialty, experience, formal training, thoracic ultrasound certification, guideline awareness and access, departmental governance (checklists, audit), and self-reported practice against specific BTS recommendations. Free text captured perceived barriers. Responses were summarised descriptively.
Results 85 clinicians from 17 acute hospitals responded. 56/85 (66%) were training-grade and 60/85 (71%) worked outside respiratory medicine; 42/85 (49%) had under one year of procedural experience. Self-reported bedside safety behaviours were high: stopping for symptoms 82/85 (97%), follow-up chest radiograph 82/85 (97%), pre-procedure safety checks 80/85 (94%), volume limitation 75/85 (88%) and ultrasound-guided aspiration 73/85 (86%). Foundational requirements, however, were not met: only 46/85 (54%) were familiar with the 2023 guidance and 18/85 (21%) held thoracic ultrasound certification, while 25/85 (29%) had received no formal training. Governance was weak or unknown: departmental checklists were confirmed by 25/85 (29%) and audit/feedback by 15/85 (18%), with 50/85 (59%) unsure. Between-centre variation was marked: across the seven sites with at least five respondents, guideline familiarity ranged 22–83%, ultrasound certification 0–67%, checklist provision 0–83% and audit systems 0–50%. Lack of training was the leading barrier (46/85, 54%).
Conclusions Among clinicians who perform pleural procedures across the region, confident bedside practice coexisted with low guideline awareness, minimal ultrasound certification and near-absent, highly variable governance. These are modifiable, system-level gaps. A coordinated regional response, comprising guideline dissemination, accredited ultrasound training and mandatory checklists with audit, is needed to align practice with 2023 BTS standards.

M31 THORACIC ULTRASOUND TRAINING IN THE UK: A SNAPSHOT PERSPECTIVE

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10.1136/thorax-2026-BTSabstracts.477

Introduction Widespread adoption of thoracic ultrasound (TUS) by respiratory physicians was observed following publication of the 2008 National Patient Safety Agency Report and 2010 BTS Pleural Disease Guidelines. Historically, training followed Royal College of Radiology (RCR) Training Standards. In 2020 the BTS Thoracic Ultrasound Training Standards were published. We conducted a national survey to evaluate the delivery of TUS training in the UK.
Methods An electronic survey (Google Forms) was distributed through direct communication with centres undertaking pleural procedures, and through respiratory trainee and pleural nursing networks between January and March 2026. Only those undertaking TUS training since 2020 were asked to contribute.
Results A total of 79 responses were received, which included respiratory higher specialist trainees (79.7%, n=63), pleural specialist nurses (7.6%, n=6), respiratory consultants (6.3%, n=5) and other respiratory doctors (2.5%, n=2). External courses were the primary training source (45.6%, n=36) whilst 10.1% (n=8) received no formal training; e-learning was utilised by 64.6% (n=51). Only 48.1% (n=38) had a designated ultrasound trainer. Multiple methods of reviewing images with a trainer were undertaken, with ad-hoc review of images most frequently used (38.0%, n=30). Electronic logbooks were used by 81.0% (n=64). BTS Training Standards were utilised by 77.2% (n=61), of which 72.1% (n=44) commenced the Primary Operator pathway. Progression onto a higher operator pathway was seen in 34.2% (n=27). Independent practice (Level 4) was achieved by 48.1% (n=38), whilst 17.7% (n=14) achieved Level 5 entrustment decision. DOPS were most frequently used

Abstract M31 Table 1 Summary of responses to ultrasound training survey

Respondents' role	*n*	%
Respiratory HST	63	79.7%
Pleural specialist nurse	6	7.6%
Respiratory consultant	5	6.3%
Advanced clinical practitioner	3	3.8%
Other respiratory doctor	2	2.5%
Formal training undertaken		
Dedicated thoracic ultrasound course provided externally	36	45.6%
General ultrasound course (e.g. FAMUS)	16	20.3%
Dedicated thoracic ultrasound course provided locally	16	20.3%
As part of induction programme- half day or less	3	3.8%
None	8	10.1%
Source of e-learning undertaken		
BTS	29	36.7%
Multiple sources	10	12.7%
e-learning modules associated with formal course	8	10.1%
ERS	4	5.1%
None	28	35.4%
Designated ultrasound trainer		
Yes	38	48.1%
No	41	51.9%
Ultrasound trainer aware of BTS Training Standards		
Yes	58	85.3%
No	10	14.7%
Method of reviewing images with trainer		
Ad hoc reviews with a trainer	30	38.0%
Reviewing saved images	23	29.1%
Supervised procedural lists	20	25.3%
Other/multiple answers	6	7.6%
Logbook method		
Electronic- created own	41	51.9%
Electronic- received from a colleague	15	19.0%
Paper	11	13.9%
Electronic- from JRCPTB website	8	10.1%
Other	4	5.1%
BTS Training Standards utilised		
Yes	61	77.2%
No- started training prior to the release	14	17.7%
No- not aware of them	4	5.1%
Pathway commenced		
Primary operator	44	55.7%
Emergency operator	12	15.2%
Advanced operator	4	5.1%
Not applicable	18	22.8%
No answer	1	1.3%
Entrustment level achieved		
Level 4- independent practice	38	48.1%
Level 5- able to teach/supervise	14	17.7%
Still working towards independent practice	9	11.4%
Not applicable	13	16.5%
Use of DOPS		
Only for independent practice	43	54.4%
At each entrustment level and final sign-off	32	40.5%
Not used	2	2.5%
No answer	2	2.5%
Progression along operator pathways		
None	38	48.1%
Yes- Primary then advanced operator pathway	14	17.7%
Yes- Emergency then Primary operator pathway	13	16.5%
Not applicable	14	17.7%

to demonstrate independent practice only (54.4%, n=43), whilst 40.5% (n=32) utilised DOPS at each entrustment level. Qualitative data highlighted barriers to learning including limited availability of trainers and their variable understanding of training standards, absence of dedicated training time and outdated ultrasound equipment.

Discussion The BTS Training Standards appear to be widely adopted in TUS training since 2020, with a small proportion of trainees transitioning from older training standards. There is significant variation amongst training delivery and methods of ongoing supervision. Barriers to training progression and the frequent absence of a designated TUS mentor are areas that should be addressed at a local and national level.

The future of sleep and physiological services

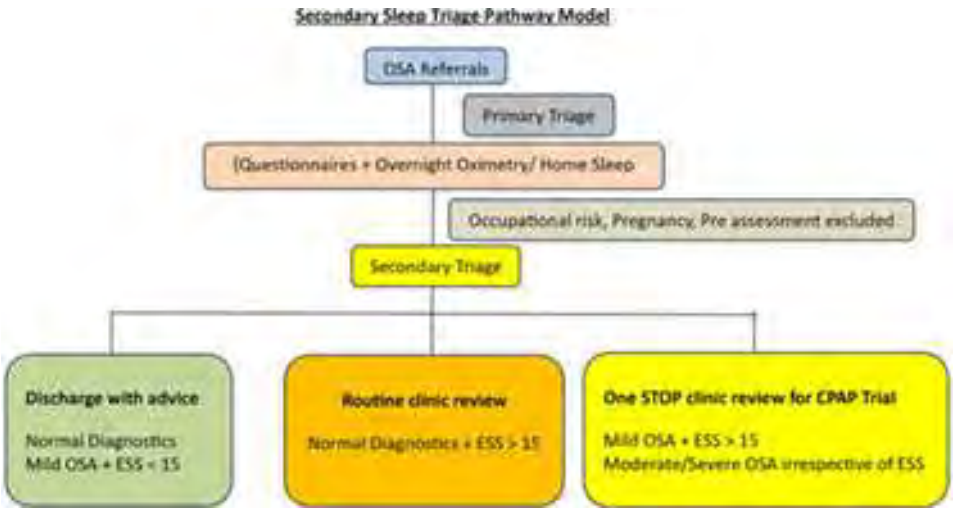
M32 A MULTIDISCIPLINARY SECONDARY TRIAGE MODEL FOR OBSTRUCTIVE SLEEP APNOEA REFERRALS: IMPROVING EFFICIENCY IN SLEEP SERVICES

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10.1136/thorax-2026-BTSabstracts.478

Background The prevalence of Obstructive sleep apnoea (OSA) and referrals to sleep services are increasing. The Elective Care Respiratory Pathways programme report highlights the urgent need for more efficient and innovative models of care to address rising demand. Reviewing all patients in outpatient clinics following traditional referral pathways increases strain on services, delaying timely access to care for those with greater need and consuming significant NHS resources. To tackle the growing burden of referrals in our trust, we implemented and evaluated a pilot 'secondary triage model'.

Method A multidisciplinary triage team (specialist nurse, sleep physiologist, consultant) was supported by a newly funded sleep co-ordinator (band 4) following a SBAR (Situation, Background, Assessment and Recommendation) assessment. A secondary triage model was devised (figure 1) and the co-or-



Abstract M32 Figure 1

dinator acted as a liaison with the triage team about referral tracking, co-ordinating pre clinic diagnostics and closing the referral loop by effective communication with the patients and primary care.

Results During the pilot phase (May - December 2025), 1621 referrals were reviewed as part of the secondary triage process. 21.70% (n= 352 referrals) were either discharged following a normal preclinic diagnostic study or with lifestyle advice (letter + snoring and sleep apnoea leaflet) for those with mild OSA and an ESS of <15. 2% (n=7) of patients were referred back from primary care for further clinical evaluation following secondary triage. Following further assessment, patients with mild OSA due to worsening ESS were initiated on CPAP trial (n=2), MRD trial (n=2), discharged following clinical assessment (n=3). Crucially, the secondary triage process created 345 additional new sleep clinic slots leading to reduced waiting times in outpatient services.

Conclusion This innovative secondary triage model, supported by a dedicated sleep co-ordinator, safely and effectively tackled rising OSA referral demands. By filtering low-risk and normal cases pre-clinically, the model aligns with the national GIRFT recommendations to free up outpatient capacity, reduce waiting times, preserves resource allocation for patients with greater clinical need and may be a scalable solution to improve care efficiency. Long term data and patient feedback will help to shape the service delivery in the region.

M33 HOME SLEEP STUDY QUALITY AND CLINICAL YIELD ACROSS DIAGNOSTIC MODALITIES IN AN NHS SLEEP SERVICE: A SERVICE EVALUATION

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10.1136/thorax-2026-BTSabstracts.479

Introduction Home-based sleep studies underpin NHS sleep diagnostics, yet real-world comparisons of technical quality and clinical yield across modalities are scarce. Respiratory polygraphy and overnight oximetry are widely used tests. We evaluated technical adequacy, signal-failure patterns and rates of clinically urgent escalation across home sleep study modalities in a large NHS sleep service.

Methods We conducted a single-centre service evaluation of consecutive home sleep studies recorded on the departmental tracking system. Respiratory polygraphy studies (January–May 2026) were compared with overnight oximetry from the same record. Technical adequacy (pass/fail) followed the local reporting protocol. Clinical escalation was defined as referral for senior review because of severe obstructive sleep apnoea or clinically significant nocturnal hypoxaemia. Pass rates and escalation rates were compared between polygraphy and oximetry using the chi-square test.

Results We analysed 391 respiratory polygraphy and 360 overnight oximetry studies. Among studies with a documented outcome, technical pass rates were similar: polygraphy 347/378 (91.8%) versus oximetry 303/331 (91.5%); p=1.00. In failed polygraphy studies, flow-signal loss occurred in 23/31 (74.2%), oximetry-channel loss in 25/31 (80.6%) and both channels in 17/31 (54.8%). A further 58/347 (16.7%) technically adequate polygraphy studies had at least one degraded channel, and reviewer documentation was absent in 37/347 (10.7%). Despite similar pass rates, overnight oximetry generated a higher rate of clinically urgent escalation than polygraphy, among technically adequate studies: 131/303 (43.2%) versus 93/347 (26.8%); p<0.001. Escalation was highest among diagnostic oximetry recorded during spontaneous breathing (102/206, 49.5%). Perioperative risk assessment accounted for 25/360 (6.9%) of the oximetry workload. Polygraphy activity rose from 77 studies in January to 95 in May.

Conclusion Respiratory polygraphy and overnight oximetry achieved similarly high technical pass rates in routine NHS practice. Oximetry generated substantially more clinically urgent escalations, particularly among spontaneously breathing diagnostic studies—most likely reflecting targeted use in higher-risk patients rather than diagnostic superiority—and supporting its role as an accessible, high-yield diagnostic pathway. Polygraphy failures clustered around flow and oximetry channel integrity, identifying patient setup, equipment checks and staff training as practical quality-improvement targets. Incomplete reviewer documentation represents a further governance and reporting priority for NHS sleep services.

M34 EFFECT OF A TELEMONITORING-ENABLED MULTIDISCIPLINARY CLINIC ON CPAP ADHERENCE IN OBSTRUCTIVE SLEEP APNOEA

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10.1136/thorax-2026-BTSabstracts.480

Introduction Poor adherence remains a major limitation to the effectiveness of continuous positive airway pressure (CPAP) therapy in obstructive sleep apnoea (OSA). Telemonitoring enables early identification and addressing of adherence problems, whilst multidisciplinary team (MDT) input provides additional support for patients with holistic management. We report the findings of a service improvement project to see whether a telemonitoring-enabled MDT clinic consisting of a respiratory physiologist, a ventilation physiotherapist and a respiratory physician added value in comparison with standard care (respiratory physiologist-only CPAP issue and follow-up service).

Methods We performed a retrospective analysis of consecutive patients initiated on CPAP between August 2023 and May 2025. Patients managed through an MDT pathway incorporating telemonitoring were compared with those receiving standard care. Baseline demographics, sleep study parameters and CPAP adherence metrics were analyzed. Multivariable logistic regression was used to identify factors independently associated with adequate adherence at 3 months (usage≥70% of days and average use≥4 hours/night), adjusting for age, body mass index (BMI), Epworth Sleepiness Scale (ESS), apnoea–hypopnoea index (AHI), oxygen desaturation index (ODI) and percentage of sleep time spent with oxygen saturation below 90% (T<90). A complementary multivariable linear regression assessed predictors of cumulative CPAP usage during the first 3 months.

Results A total of 728 patients were included. Despite having higher disease severity and BMI, patients managed through the MDT pathway demonstrated superior adherence with a trend towards a higher proportion of patients using CPAP>4 hours/night (compliance) compared with those receiving standard care (table 1). After adjustment for baseline demographic and disease-severity variables, MDT management remained independently associated with achieving adequate adherence at 3 months (adjusted OR 1.92, CI=1.326-2.782, p=0.0006). Older age was also independently associated with better adherence (p<0.0001). In the complementary continuous-outcome analysis, MDT management remained independently associated with greater CPAP utilisation during the first 3 months (additional 64.9 hours, 95% CI=20.4–109.3, p=0.004).

Conclusion An MDT model incorporating telemonitoring was independently associated with improved CPAP adherence and greater device utilisation in patients with OSA. These findings suggest that a multidisciplinary sleep service utilising telemonitoring improved CPAP adherence beyond that achieved with standard CPAP care.

Abstract M34 Table 1 Baseline Characteristics and CPAP Outcomes in MDT and non-MDT groups

Variable	MDT	Non-MDT	p
Age at setup (years)	50 (40–57)	50 (42–59)	0.236
BMI (kg/m²)	35.8 (30.7–42.4)	34.2 (29.3–40.6)	0.035
ESS	13 (9–16)	13 (9–17)	0.541
AHI (/h)	31.6 (16.5–60.5)	28.0 (14.6–55.2)	0.136
ODI (/h)	33.6 (18.9–62.3)	28.8 (15.3–50.3)	0.012
T<90 (%)	13.8 (2.8–41.8)	9.7 (1.9–35.8)	0.066
Total hours of usage / 3 months	269.1 (24.8–511.9)	157.5 (11.4–440.1)	0.012
Total number of days used / 3 months	63 (13–84)	41 (7–81)	0.006
Average usage hours/day, 3 months	4.4 (2.1–6.3)	4.0 (1.4–6.2)	0.121
Compliance (daily usage≥4 hours)	231 (59%)	172 (52%)	0.070
Adherence (≥70% of nights and daily usage≥4 hours), 3 months	179 (45%)	113 (34%)	0.002

AHI Apnoea hypopnea index, *BMI* Body mass index, *ESS* Epworth sleepiness scale, ODI oxygen desaturation index, T90 % of total sleep time spent with oxygen saturation <90%. *Note: Values presented as Median (IQR) or n (%). Comparisons between MDT and non-MDT approaches were made using non-parametric tests (Mann-Whitney-U-Test) and Fisher's Exact test of difference, with statistical significance accepted at p<0.05.*

M35 DOES WHERE YOU LIVE AFFECT EARLY CPAP USE? THREE-MONTH ADHERENCE ACROSS RURAL HEREFORDSHIRE

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10.1136/thorax-2026-BTSabstracts.481

Introduction Continuous positive airway pressure (CPAP) is first-line treatment for obstructive sleep apnoea (OSA), but benefit depends on regular nightly use. Geographic and socioeconomic barriers may affect CPAP uptake and ongoing use,[1] particularly in rural communities where travel distance, deprivation and access to follow-up vary. We evaluated whether residence within Herefordshire postcode districts was associated with early CPAP adherence and compliance.

Methods We undertook a service evaluation of adults established on CPAP with a Herefordshire outward postcode (HR1-HR9). Patients receiving non-invasive ventilation and those with COPD overlap were excluded. Device-derived usage was assessed over a one-month period starting three months after CPAP set-up. Adherence was defined as device use on at least 70% of nights, and compliance as mean use of at least 4 hours per night across all days. Residence was analysed by outward postcode district and grouped as county town (HR1, HR2 and HR4) versus surrounding rural market-town districts. Proportions were compared using chi-square testing.

Results Of 923 HR-postcode CPAP records, 730 had analysable three-month usage data. Overall adherence was 72.9% (532/730), compliance was 77.7% (567/730), and mean nightly use was 5.83 hours. Adherence did not differ significantly across HR1-HR9 (range 68.0-81.3%; p=0.77), nor between county-town and rural districts (71.8% versus 74.3%; p=0.50). Compliance was also similar across postcode districts (range 75.0-87.5%; p=0.98) and between county-town and rural districts (77.9% versus 77.4%; p=0.96). By contrast, adherence varied by diagnostic category: UARS 38.2%, mild OSA 69.1%, moderate OSA 74.9% and severe OSA 78.5% (p<0.001).

Conclusion In this rural county CPAP cohort, early device-derived adherence and compliance were high and showed no evidence of postcode-related variation. Diagnostic category, particularly UARS compared with moderate-to-severe OSA, appeared more strongly associated with CPAP use than area of residence. Linkage to deprivation, education and occupational indices is planned to explore whether more granular socioeconomic measures identify inequities not captured by outward postcode district.

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M36 SENSITIVITY OF OVERNIGHT OXIMETRY BETWEEN ETHNIC GROUPS

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Background Some centres continue to use overnight oximetry as the first-line investigation for sleep apnoea, often due to local commissioning arrangements. Oxygen saturation probes have been demonstrated to overestimate the measurement in people with darker skin tones (Martin et al 2026). A retrospective study was therefore conducted to explore the possible differences in overnight oximetry results between two ethnicities, potentially representing either end of the skin tone spectrum; to determine any differences in the quality of oximetry as a diagnostic tool.

Methods All new sleep referrals who underwent overnight oximetry between July and December 2025, and had a recorded ethnicity of either a black or white background (but not mixed) were analysed.

Results and Discussion 509 patients with the relevant ethnic backgrounds were identified, with 7 excluded as results were unavailable. Mean saturations were significantly higher in those with an ethnicity recorded as any black background, compared to those with any white background, in keeping with the previous evidence. Desaturation index was significantly lower for those with a black ethnicity. Underestimation of desaturation index would be the expected outcome from a shift in the oxyhaemoglobin dissociation curve caused by an overestimation of saturations, resulting in a move to the shallower plateau of the curve. A larger proportion of those with a black ethnicity required a second sleep study (WatchPAT), and a larger proportion of those with a black ethnicity had positive second studies. Epworth sleepiness scale was significantly higher in those with a black ethnicity. Pulse rises as a surrogate marker of breathing events also suggested an underestimate of severity in those with a black ethnicity, with a higher ratio of pulse rises to desaturation index.

Overnight oximetry overestimates mean saturations and underestimates desaturation index in those with a black ethnicity compared to those with a white ethnicity, and thus should only be used with caution as an investigation for sleep apnoea, and only with the safety net of more sensitive tests.

Abstract M36 Table 1 Values given as mean, unless otherwise stated

Ethnicity	Black Background	White Background
Number	205 (134 female)	297 (140 female)
Mean oxygen saturations	94.78	93.13 ($p < 0.001$)
Desaturation index 4% (ODI)	14.65	17.58 ($p = 0.034$)
Ratio of Pulse Rises : ODI	8.57	6.16 ($p = 0.095$)
Epworth Sleep Score (ESS)	10.80	9.84 ($p = 0.043$)
Proportion needing a more detailed sleep study	26.34%	20.88%
Proportion of more detailed study positive for sleep apnoea	33.33%	18.97%

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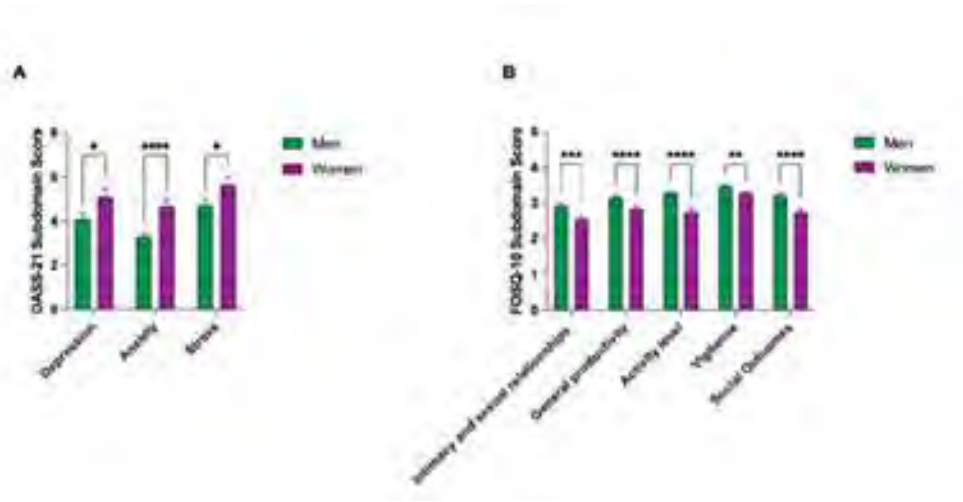
M37 DOES HYPOXIC BURDEN BETTER EXPLAIN SEX DIFFERENCES IN FUNCTIONAL IMPAIRMENTS THAN APNOEA-HYPOPNOEA INDEX IN OBSTRUCTIVE SLEEP APNOEA?

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10.1136/thorax-2026-BTSabstracts.483

Background Women with obstructive sleep apnoea (OSA) frequently report more severe functional impairments than men, despite lower apnoea-hypopnoea index (AHI) values. This raises questions about the adequacy of AHI for capturing OSA severity. Hypoxic burden (HB), a metric incorporating the depth and duration of oxygen desaturations, may be a superior measure of disease severity than AHI. We explored sex differences in functional outcomes in OSA patients and examined whether AHI or HB better explained these differences.

Methods We analysed cross-sectional data from a multicentre sleep clinic cohort (2018-2023). Adults with OSA (AHI ≥5 events/hour on polysomnography) with data on mood (Depression, Anxiety and Stress Scale; DASS-21) and daytime functioning (Functional Outcomes of Sleep Questionnaire; FOSQ-10) were included. Linear regression models examined associations between sex, AHI, HB, and functional outcomes. Interaction effects of sex and AHI/HB were explored. Multivariable models were adjusted for anthropometry, menopausal status, comorbidities and medications.



Abstract M37 Figure 1 Comparison of functional outcome subdomains between men (n=350) and women (n=168). Error bars represent SEM. (Ass) DASS-21 subdomain scores. (B) FOSQ-10 subdomain scores

Results Among 518 untreated OSA patients (67.7% men, mean age 54.0, SD 14.6 years), women had significantly higher DASS-21 (15.4 vs. 12.1, p<0.01) and lower FOSQ-10 (14.1 vs. 16.0, p<0.0001) scores, despite lower AHI (28.7 vs. 32.9, p=0.10) and HB (47.4 vs 82.7, p<0.001) values (figure 1). Neither AHI nor HB were associated with worse mood, although AHI was linked to poorer mood in women in sensitivity analyses. AHI and HB were associated with worse FOSQ-10. In multivariable models adjusting for demographics, anthropometry, comorbidities and medications, neither AHI nor HB consistently predicted mood or daytime functioning. Sex differences in mood were explained by comorbid mood disorders, insomnia and chronic pain.

Conclusions Women had more severe functional impairments despite milder respiratory indices. Given the greater influence of comorbidities on functional outcomes than respiratory metrics, comprehensive comorbidity assessment in OSA evaluation is warranted, especially in females.

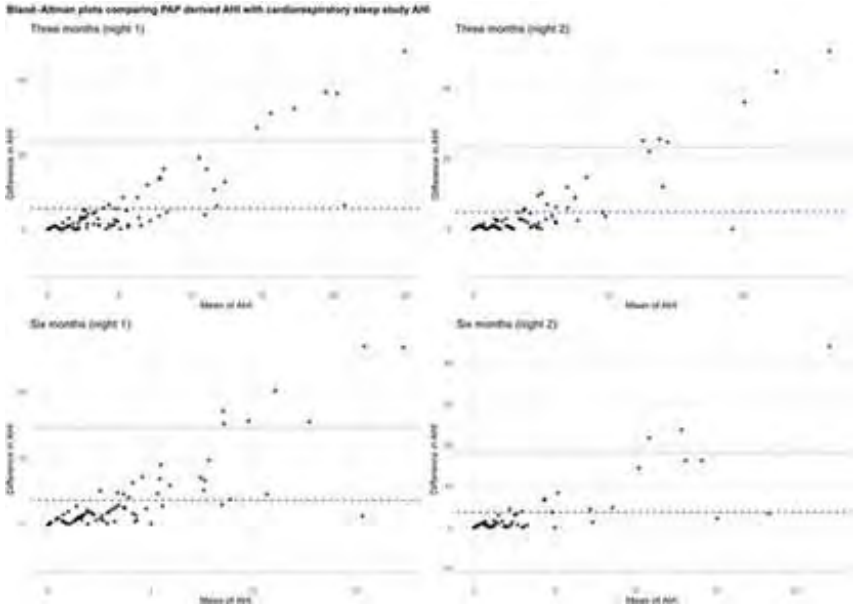
M38 RESIDUAL APNOEA HYPOPNEA INDEX MEASUREMENT FROM PAP DEVICES VERSUS HOME POLYGRAPHY: A METHOD COMPARISON STUDY USING REAL WORLD DATA

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10.1136/thorax-2026-BTSabstracts.484

Introduction Remnant Apnoea Hypopnoea Index (AHI) derived from positive airway pressure (PAP) devices is frequently used to assess treatment effectiveness in obstructive sleep apnoea (OSA). However, the accuracy of device reported AHI in realworld settings remains uncertain. This study evaluated agreement between AHI derived from PAP devices and home based limited polygraphy.

Methods Participants underwent home based polygraphy at 3 and 6 months from commencing PAP therapy. Remnant



Abstract M38 Figure 1

AHI was obtained from Prisma Smart (Löwenstein, Germany) PAP devices and Embletta MPR (Natus, USA). Agreement was assessed with Bland–Altman plots, intraclass correlation coefficients and weighted Kappa Coefficient. Sensitivity and specificity for detecting inadequate control (AHI ≥7 events/hr) was computed. Association between interface leak and AHI discrepancy was assessed with linear regression.

Results 98 participants were included. PAP derived AHI consistently underestimated remnant AHI with a median difference in PAP derived AHI and polygraphy derived AHI of 2.2 (IQR 1 to 4.1, p=0.002) at three moths and 0.5 (IQR 0.4 to 2.3, p=0.007) at 6months. Agreement was poor, with intraclass correlation coefficient was 0.02 (95% CI: –0.16 to 0.21) at 3months and 0.16 (95% CI: –0.04 to 0.35) at 6months. There was low diagnostic agreement across severity categories at both three months ($_K$0.10, 95%CI: 0 to 0.26) and six months ($_K$0.09, 95%CI: 0 to 0.28). Sensitivity for detecting inadequate control was very low at both time points (3 months 12% and 6 months 21%), despite high specificity (97% at 3 months and 95% at 6 months). Bland–Altman plots showed underestimation with wide limits of agreement (figure 1). Higher interface leak was significantly associated with greater underestimation of PAP AHI (three months: R^2 0.18, $p < 0.0001$; six months: R^2 0.14, $p < 0.0001$).

Conclusion PAP device downloads substantially underestimated residual AHI and demonstrated poor agreement with home cardiorespiratory polygraphy. These findings highlight important limitations of device derived AHI in realworld practice, particularly in the presence of interface leak. Where treatment adequacy is uncertain, confirmatory assessment with polygraphy remains necessary.

M39 FOLLOW-UP AFTER CPAP THERAPY INITIATION IN PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS) – INSIGHTS FROM A RETROSPECTIVE ANALYSIS

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10.1136/thorax-2026-BTSabstracts.485

Background Continuous positive airway pressure (CPAP) is a recognized treatment for children with obstructive sleep apnea syndrome (OSAS). After discharge, regular monitoring of therapy effectiveness is necessary, typically via clinic visits and sleep studies. However, evidence-based recommendations for structured outpatient follow-up are not available and clinical practice remains inconsistent.

Objective We aimed to develop recommendations for outpatient follow-up after CPAP initiation in children and adolescents with OSAS, based on a retrospective analysis of our patient cohort.

Methods 62 children with OSAS on CPAP therapy underwent 286 check-ups comprising sleep study and clinical review, between 2012 and 2025. Follow-up intervals were determined, as well as whether these visits led to clinical or technical therapy adjustments (pressure titration, additional O2 etc.).

Results Of 62 patients (female 34.8%), median age at CPAP initiation was 7.4 years (range 0.5 to 16.4 years); 48% of patients where classified as overweight or obese and 74.6% had comorbidities, of which the most common was Trisomy 21 (36.5%). 243 of 286 follow-up visits were evaluable. A management change was made 29.7%, most commonly at 13–15 months (43.8%) and 28–33 months (37.5–42.9%). The 19–21 month interval showed the lowest change rate (5.9%), and no changes were recorded at 34–39 months.

41 follow-up visits (19%) occurred more than 36 months after therapy initiation. The highest rate of therapy adjustment was observed in 10- to 12-year-olds (28.6%), while the fewest occurred in the 14-16 group (17.2%).

Pressure adjustments were the most common intervention (55% of visits), followed by decisions regarding continuing treatment.

Conclusion Most changes were required in the first 3 years of CPAP initiation and for the group aged 13-15 months. We recommend a structured follow-up protocol after CPAP initiation in pediatric OSAS: An initial clinical check-up with sleep study after 3 months, then every 6 months. If clinically stable after 3 years, sleep study frequency may be extended.

Data from our cohort support a streamlined follow-up approach and may serve as a basis for the development of a standardized monitoring protocol.

M40 OPTIMISING TRANSCUTANEOUS ELECTRICAL STIMULATION STRATEGIES FOR OBSTRUCTIVE SLEEP APNOEA: COMPARING CONTINUOUS AND INTERMITTENT STIMULATION USING A SKELETAL MUSCLE MODEL

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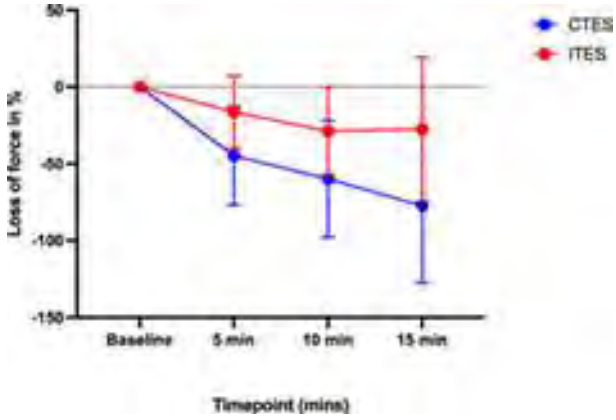
10.1136/thorax-2026-BTSabstracts.486

Background Transcutaneous electrical stimulation (TES) is a potential therapeutic approach for improving upper airway muscle function in obstructive sleep apnoea (OSA). However, sustained stimulation may induce muscle fatigue, potentially limiting long-term effectiveness. This study compared intermittent TES (ITES) and continuous TES (CTES) to determine which protocol better preserves stimulation-induced muscle force.

Methods Healthy participants underwent CTES and ITES applied to the tibialis anterior (TA) muscle as a model of upper airway dilator muscles. Stimulation-induced force was assessed during a 15-minute stimulation protocol. Maximal voluntary contraction (MVC), voluntary activation (VA), force–frequency responses, discomfort scores, and ultrasound measures of TA muscle thickness were evaluated. Data were analysed using paired and repeated-measures statistical models.

Results Fourteen participants completed both stimulation protocols (age 36.4±11.4 years; BMI 22.6±4.1 kg/m^2). At the end of the 15-minute stimulation period, ITES resulted in significantly less stimulation-induced force loss compared with CTES (−27.6% vs −77.4%; mean difference 49.8%; 95% CI 3.5–96.1; p=0.030).

MVC decreased following both CTES (mean change −19.7 N; 95% CI −35.9 to −3.4; p=0.021) and ITES (mean change −15.4 N; 95% CI −28.3 to −2.5; p=0.013). Force–frequency responses demonstrated peripheral fatigue following CTES (mean change −10.7 N; 95% CI −14.7 to −6.8; p=0.008) and ITES (mean change −11.6 N; 95% CI −15.2 to −8.0; p=0.008).



Abstract M40 Figure 1

VA remained comparable between CTES and ITES (78.4% vs 86.2%; mean difference 7.8%; 95% CI −7.0 to 22.5; p=0.294), indicating similar central activation. Discomfort scores were similar between protocols (p=0.750). TES increased TA thickness measured by ultrasound in both coronal (mean change +11.2%; 95% CI 5.8–16.6; p=0.001) and sagittal planes (mean change +8.8%; 95% CI 0.1–17.5; p=0.048).

Conclusion ITES resulted in significantly less stimulation-induced force loss than CTES during a 15-minute stimulation protocol while maintaining comparable discomfort and VA. These findings support intermittent stimulation as a potential strategy to optimise TES delivery for future translation to upper airway muscle stimulation in patients with OSA.

M41 OBESITY MANAGEMENT IN OBSTRUCTIVE SLEEP APNOEA: PATIENT PERSPECTIVES AND A QUALITY IMPROVEMENT STUDY

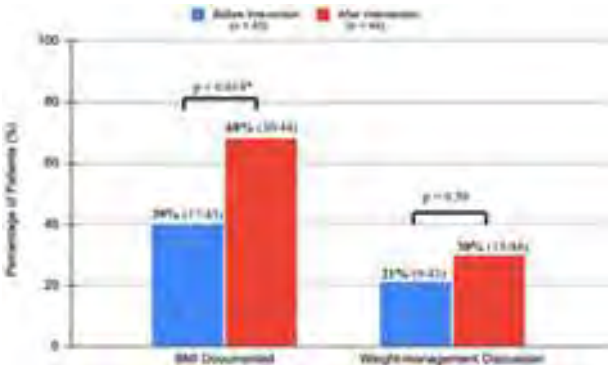
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10.1136/thorax-2026-BTSabstracts.487

Introduction Prevalence of obstructive sleep apnoea(OSA) is estimated to be around 45% in obese patients.[1] Significant weight loss can reduce OSA severity and mitigate cardiovascular complications of obesity.[1] We aimed to evaluate patients' perspectives and assess clinician engagement with weight-management support.

Methods 18 day unit inpatients with OSA and BMI >30(mean=42.6kg/m^2) completed a survey. This included qualitative questions regarding previous weight loss support and understanding of the relationship between obesity and OSA. Additionally, a visual analogue scale was used for the perceived usefulness of weight-management interventions. Separately, an audit was completed across one week of clinics(n=43), before a clinician education intervention was delivered on the importance of weight loss advice and a second audit cycle was repeated over a subsequent clinic week(n=44). Results were analysed using chi-squared testing.

Results 7/18(39%) of patients did not understand the relationship between obesity and OSA and 9/18(50%) of patients had no previous weight loss support. Diet/exercise guidance and pharmacotherapy demonstrated the highest perceived usefulness scores(70% and 71% respectively). Surgery had the least perceived usefulness with 38%. Pharmacotherapy was most frequently ranked as the preferred intervention. In the pre-intervention cohort, 17/43(40%) of patients had their BMI recorded but only 9/43(21%) had documentation of weight-management discussion. Following intervention, BMI documentation increased to 30/44(68%)(p=0.014), while weight-management discussion increased to 13/44(30%)(p=0.50) [figure 1].



Abstract M41 Figure 1 Graph showing documentation and discussion of obesity and weight-loss management in OSA clinics before and after clinician educational intervention. *P<0.05 judged as statistically significant. Percentage rounded to the nearest integer

Conclusions Many patients demonstrated limited understanding of the relationship between obesity and OSA and reported limited prior weight-loss support. Although clinician education significantly improved BMI documentation, discussion of weight-management strategies remained infrequent and did not significantly improve, suggesting persistent barriers to obesity-focused counselling in sleep clinics. These findings have prompted development of integrated obesity-support pathways within our service. Pharmacotherapy was perceived as the most useful intervention, reflecting growing patient interest in medical obesity therapies.

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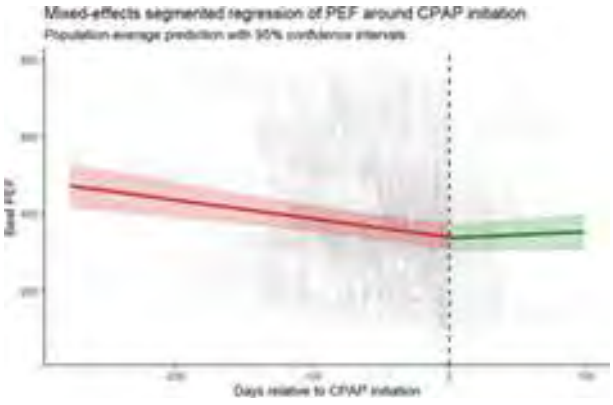
M42 OBSTRUCTIVE SLEEP APNOEA AND DIFFICULT ASTHMA: PROVISIONAL FINDINGS FROM THE OSADA TRIAL

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10.1136/thorax-2026-BTSabstracts.488

Background Obstructive sleep apnoea (OSA) is increasingly recognised as a potentially modifiable comorbidity in difficult asthma. The OSADA trial investigates whether OSA and continuous positive airway pressure (CPAP) are associated with asthma control, lung function, and peak expiratory flow variability.

Methods OSADA is an open-label, multi-centre, randomised controlled trial of adults attending a difficult asthma service, with 81 participants enrolled. Participants underwent home sleep apnoea testing and serial electronic lung function monitoring using the Vitalograph Lung Monitor Model 4000. Participants without OSA formed a comparator group; those with OSA were randomised to CPAP or no CPAP. For this provision-



Abstract M42 Figure 1 Population-average fitted PEF trajectory from a segmented linear mixed-effects model centred on CPAP initiation (day 0). The red line represents the estimated pre-CPAP trend, the green estimated post-CPAP trend, with shaded regions indicating 95% confidence intervals. Grey lines depict individual patient trajectories aligned to the date of CPAP initiation

al analysis, CPAP initiation was defined as day 0 for treated patients, while untreated OSA participants and non-OSA controls were aligned using a pseudo-index date. Linear mixed-effects segmented regression models assessed changes in FEV1 and peak expiratory flow (PEF), incorporating time, post-index status, their interaction, diurnal timing, and patient-level random effects. Peak expiratory flow variability (PEFV) was calculated from paired morning and evening readings. Epworth Sleepiness Scale (ESS) and Asthma Control Test (ACT) scores were compared before and after CPAP using paired t-tests with Bonferroni correction.

Results Following exclusions, 72 participants were included. Thirty participants (41.6%) had OSA, defined by AHI ≥10 events/hour, and were randomised 1:1 to CPAP or no CPAP. The cohort contributed 10,274 FEV1 and 10,272 PEF observations. Before the index date, FEV1 declined significantly over time, while the post-index interaction showed a positive slope change, consistent with attenuation of decline (β=1.83 per 10-day unit, SE=0.47, t=3.90). PEF showed a similar but more pronounced pattern (β=6.43 per 10-day unit, SE=0.93, t=6.93), with reversal of the pre-index downward trajectory, as shown in figure 1. Morning and evening PEF both improved. ACT improved significantly following CPAP, from 17.25 to 20.19 (p=0.0058). ESS improved from 9.38 to 7.06 but did not meet the corrected significance threshold (p=0.06).

Conclusion In this provisional analysis, the indexed CPAP period was associated with favourable changes in lung function trajectories and asthma control, supporting further investigation of OSA as a treatable trait in difficult asthma. However, causality cannot be inferred from these provisional models.

M43 DELIVERING THE NHS LONG TERM PLAN: OUTCOMES FROM A DIGITALLY ENABLED COMMUNITY BREATHLESSNESS SERVICE

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10.1136/thorax-2026-BTSabstracts.489

Breathlessness has multifactorial causes leading to delayed diagnosis and inefficient, hospitalcentred pathways. The NHS 10 Year Health Plan prioritises earlier and more accurate diagnosis, analoguetodigital transformation, and a shift from hospital to community care. We evaluated how our integrated Breathlessness Service delivers these ambitions.

A retrospective analysis of referrals was undertaken. Diagnostic contributors (N=71) and pathway timings were extracted from structured proformas and EPIC workflow data. Key metrics included time to triage, diagnostics, MDT review, and discharge.

Common diagnostic contributors (in isolation or in combination) were: Breathing Pattern Disorder 38%, anxiety 23%, obesity 23%, asthma 18%, COPD/emphysema 6%; other contributors (deconditioning, musculoskeletal, anaemia, ILD, allergy) each ≤5%. Pathway timings demonstrated accelerated access: referral to triage 1 day (median); referral to first diagnostic 22 days; referral to first MDT 38 days; triage to diagnostic 20 days; diagnostic to MDT 15 days. Patients without CPET had referral to discharge in 38 days, compared with 80 days when CPET was required. All referrals, diagnostics and MDT actions were captured digitally via EPIC, supporting the national shift from analogue to digital. Most patients were managed fully within community-based pathways, aligning with the NHS move from hospital to community.

This service delivers rapid, holistic assessment and digitally integrated care for complex breathlessness, demonstrating real-world implementation of NHS Long-Term Plan reforms. Improved timelines, digital integration, and community-based MDT care highlight its potential as a scalable model for modern respiratory services.

M44 RESPIRATORY FUNCTIONS IN ADULT WITH PERINATAL HIV INFECTION

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10.1136/thorax-2026-BTSabstracts.490

Background Chronic lung disease is a recognised complication of perinatally acquired HIV (PaHIV), associated with impaired pulmonary function in some children but there are no prior data in adults with PaHIV. We have therefore evaluated spirometry in this group.

Methods This single-centre cross-sectional study included adults with PaHIV attending the 900 Clinic at Imperial College Healthcare NHS Trust between 1 February and 1 May 2026. Routine screening spirometry was performed using a Vyaire Medical Microlab spirometer. Clinical records were reviewed for baseline characteristics, spirometry, underlying respiratory diseases and their associations.

Result Among 60 adults with PaHIV, 50 (83.3%) were of Black ethnicity and 32 (53.3%) were female. The mean age was 27.7 ±4.8 years. Fifty-four participants produced acceptable spirometry results according to British Thoracic Society criteria. Of these, 38/54 (70.4%) had a restrictive spirometry pattern, while 16/54 (29.6%) had normal spirometry. No obstructive spirometry pattern was observed.

Respiratory diagnoses according to spirometry pattern are shown in table 1. Overall, respiratory diagnosis was present in

Abstract M44 Table 1 Respiratory diagnoses according to spirometry pattern

Variable	Restrictive spirometry (n=38)	Normal spirometry (n=16)
Number of respiratory diagnoses		
0	18 (47.4%)	12 (75.0%)
1	10 (26.3%)	2 (12.5%)
2	6 (15.8%)	1 (6.3%)
≥3	4 (10.5%)	1 (6.3%)
Respiratory diagnoses*		
Previous pneumonia	15 (39.5%)	3 (18.8%)
Asthma	5 (13.2%)	2 (12.5%)
Tuberculosis	4 (10.5%)	2 (12.5%)
Pneumocystis pneumonitis	4 (10.5%)	0
Cytomegalovirus pneumonitis	3 (7.9%)	0
Bronchiectasis	3 (7.9%)	1 (6.3%)
Lymphocytic interstitial pneumonitis	2 (5.3%)	0

*As participants could have more than one respiratory diagnosis, percentages in this section do not add to 100%.

20/38 (52.6%) participants with restrictive spirometry compared with 4/16 (25.0%) participants with normal spirometry. Restrictive spirometry was associated with increased odds of an underlying respiratory diagnosis (OR 3.3, 95% CI 0.8–16.4, p=0.07).

Conclusion In our study, restrictive spirometry was common among adults with perinatally acquired HIV. Participants with restrictive spirometry had a greater burden of underlying respiratory diagnosis than those with normal spirometry. Further studies incorporating full pulmonary function testing are needed to better understand the mechanisms underlying restrictive lung impairment in this population.

M45 IMPULSE OSCILLOMETRY IN EXTRATHORACIC RESTRICTION FRIEND OR FOE?

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10.1136/thorax-2026-BTSabstracts.491

Introduction Impulse oscillometry (IOS) is a novel lung function test (LFT) that measures respiratory system impedance using externally applied sound waves. It has a well-defined role in the diagnosis of obstructive airways disease, and may be particularly useful in excluding obstruction in those with poor spirometric technique. IOS has also been shown to correlate with restrictive lung function in interstitial lung disease; however, its role in extra-thoracic restriction (ETR), such as that caused by neuromuscular or musculoskeletal disease is less well-defined (Bednarek M et al Respir Med. 2020 Aug-Sep;170:105952).

Methods All IOS tests performed at a large London teaching hospital in 2025 for indications other than obstructive airways disease were identified. Cases were independently reviewed by at least two consultant chest physicians to determine an agreed diagnosis.

Results IOS was requested on 84 occasions, with 28 tests requested for indications other than overt obstructive airways disease (7 restrictive lung disease, 3 interstitial lung disease, 2 neuromuscular/diaphragm disorder, 1 obstructive sleep apnoea/sleep related breathing disorder, 3 pre-operative assessment and 12 other). Of these, 8 patients with an MDT diagnosis of an ETR disease had undergone IOS: 6 had normal IOS, 1 an obstructive pattern and 1 a mixed pattern (mixed COPD and kyphoscoliosis). No patients with an MDT diagnosis of ETR had a restrictive pattern on IOS.

Conclusions Whilst IOS may be useful in diagnosing restrictive lung diseases, especially in those with poor lung function technique, care needs to be taken in its interpretation in extra-tho-

Abstract M45 Table 1 Summary of impulse oscillometry (IOS) cases with extra-thoracic restriction as final MDT (multi-disciplinary team) agreed diagnosis. FEV1 = Forced expiratory volume in one second, FVC = Forced vital capacity, COPD = Chronic obstructive pulmonary disease

Suspected Diagnosis on IOS Request	FEV1 (L)	FEV1 (% predicted)	FVC (L)	FVC (% predicted)	FEV1/ FVC	FEV1/FVC Z-Score	IOS Result	MDT Diagnosis
Neuromuscular Disease / Diaphragm disorder	1.18	42%	1.45	41%	0.81	0.43	Normal	Diaphragmatic palsy
OSA/Sleep related breathing disorder	1.92	77%	2.34	73%	0.82	0.46	Normal	Neuromuscular weakness
Other	1.55	65%	1.90	64%	0.81	0.16	Normal	Unclear
Other	4.45	92%	4.87	86%	0.92	0.97	Normal	Pectus excavatum
Restrictive Lung Disease	0.97	41%	1.06	36%	0.93	1.89	Obstructive	Obesity and hemidiaphragmatic palsy
Restrictive Lung Disease	Unable						Mixed	COPD and kyphoscoliosis
Restrictive Lung Disease	1.97	70%	2.47	69%	0.80	0.19	Normal	Extra-thoracic restriction due to previous surgery and post-operative neuropathic pain
Restrictive Lung Disease	1.68	45%	2.17	49%	0.77	-1.21	Normal	Kyphoscoliosis

racic restriction. Its role as a 'rule out' test for restriction in this context should be questioned.

M46 EXERCISE LIMITATION IN COPD: INSIGHTS FROM CARDIOPULMONARY EXERCISE TESTING

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Introduction Exercise limitation due to dyspnoea is common in patients with chronic obstructive pulmonary disease (COPD) and is commonly attributed to abnormal ventilatory mechanics (reduced FEV_1). Cardiopulmonary exercise testing (CPET) can provide insight into mechanisms and may identify secondary or co-existing processes, such as deconditioning or cardiovascular limitation like chronotropic incompetence, contributing to exercise limitation. Despite this, there is limited data describing CPET outcomes in patients with COPD. This retrospective cohort study aimed to review CPET outcomes in patients with COPD and determine the relative contribution of ventilatory and non-ventilatory mechanisms to exertional dyspnoea.

Methods Bicycle ergometric CPET data was retrospectively extracted from referrals for abdominal aortic aneurysm (AAA) pre-assessment between March 2021 and March 2025 as part of a service improvement project. The AAA pre-assessment cohort was selected as it represented a large group of patients undergoing routine CPET. Patients with COPD were identified through spirometry and smoking status. Spirometric obstruction was defined as FEV_1/FVC below the lower limit of normal using GLI 2012 reference values. Data collected included smoking status, inhaled medication use, demographics and CPET outcomes.

Results Of 703 AAA pre-operative assessment referrals, 222 (31.6%) had COPD. Of these, 107 (48.2%) were current smokers, 113 (50.9%) were ex-smokers and 2 (0.9%) were never smokers. Inhaled therapy was prescribed in 108 (48.6%) patients, while 114 (51.4%) were not receiving inhaled medication. Fifty-eight patients with COPD (26.1%) did not complete CPET due to either: patient choice, functional limitation preventing cycling or contraindications to testing. Among 164 patients who completed CPET, 156 (95.1%) demonstrated severe functional limitation. Only 52 patients (31.7%) had ventilatory limitation; 37 (22.6%) had gas-exchange abnormality. In contrast, 113 (68.9%) demonstrated chronotropic incompetence and 68 (41.5%) had deconditioning [table 1].

Conclusion Almost all patients with COPD had severe functional limitation confirming poor exercise tolerance. However, only one-third of patients were limited by ventilatory mechanics. Non-ventilatory causes were frequent, particularly chronotropic incompetence and deconditioning. These findings suggest that exercise limitation in COPD is often multifactorial rather than solely explained by ventilatory mechanics. CPET may help identify treatable contributors to exertional dyspnoea allowing targeted interventions such as pulmonary rehabilitation.

Abstract M46 Table 1 CPET outcomes in patients with COPD

Outcome	Frequency (n/N)	Percentage (%)
Severe functional defect (VO_2peak < 15 mL/kg/min)	156 / 164	95.1%
Chronotropic incompetence (chronotropic index < 0.8)	113 / 164	68.9%
Deconditioned (VO_2 at AT < 50% of predicted VO_2peak)	68 / 164	41.5%
Ventilatory limited (breathing reserve < 20%)	52 / 164	31.7%
Gas exchange abnormality	37 / 164	22.6%

VO_2= Oxygen Uptake; VO_2peak= Maximal Oxygen Uptake; AT=Anaerobic Threshold

M47 MIND THE GAP: COMPARISON BETWEEN GLI RACE NEUTRAL LUNG FUNCTION WITH ECCS IN DIFFERENT ETHNICITIES

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Background and Objective Population level standardised mean is used to define predicted physiological values. They may function differently across ethnicities. Moreover, new iteration of standards can make this issue even more complex. Pulmonary function test(PFT) has changed over from European Community of Coal and Steel (ECCS) to Global Lung Function Initiative(GLI) defined standards. This study examines whether a difference exists between the GLI race neutral equation and ECCS, in their performance in whites versus non-whites.

Method Age and sex matched white with non-white pairs were identified during August'25-December'25 and PFTs were generated using both ECCS and GLI standards. Within the matched pairs, we calculated [predicted

FVC% (GLI) - pred FVC%(ECCS)] white- [predicted FVC% (GLI) - pred FVC%(ECCS)]non-white. The differences between each pair across the cohort were examined by paired t test and then by linear regression, adjusting for body mass index(BMI). The same was done for FEV1.

Results In 22 age(mean 53±17 years, range 24-86)/ sex matched pairs, white patients' GLI predicted FVC was, on an average, 10% less than the ECCS predicted FVC, as compared to non-white patients (95% C.I. -13, -7; (p<0.001). The similar differences for FEV1 was 8% (95% C.I. -11, -5; p<0.001). These diifferences remained the same in linear regression adjusted for BMI.

Abstract M47 Table 1 Comparison of GLI and ECCS lung function in age and sex-matched white and non-white patients

	Non-white	White	
BMI	28	29	
Obstructive lung disease	9(41%)	83(37%)	
Interstitial lung disease	1(5%)	3(14%)	
FVC^{GLI}, FVC^{ECCS}	2%	-8%	p<0.0001
$FEV1^{GLI}$, $FEV1^{ECCS}$	5%	-3%	p<0.0001

Conclusion The predicted values obtained from GLI equation significantly underpredicts FVC and FEV1 in whites than non-white, compared with ECCS values. This may have clinical significance in longitudinal follow up using different methods.

2026 Abstract disclosures

T1 **INVESTIGATING THE INFLAMMATORY PROFILE OF EXACERBATIONS IN PATIENTS WITH SEVERE ASTHMA RECEIVING TEZEPELUMAB: THE PHASE IV TEZEX STUDY**

This study was partly funded by AstraZeneca (AZ). AZ had no role in study design, data collection, analysis, interpretation, or abstract preparation.

T2 **ACQUIRED MONOCYTE REPROGRAMMING DRIVES DIVERGENT RESPONSES TO MICROBIAL STIMULI IN IDIOPATHIC PULMONARY FIBROSIS.**

Funded by Asthma+Lung UK.

T4 **FENO RESPONSE TO BENRALIZUMAB IS DETERMINED BY BRONCHIAL EPITHELIAL PHENOTYPE**

Investigator led, AstraZeneca funded study.

S2 **HOME-BASED BODY-WEIGHT EXERCISE WITH BLOOD FLOW RESTRICTION IN COPD: A RANDOMISED FEASIBILITY TRIAL**

This abstract was presented at European Respiratory Society Congress.

S6 **SUPPORTED "RESCUE PACKS" POST-DISCHARGE FOR PEOPLE LIVING WITH COPD – QUALITATIVE INTERVIEW STUDY**

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S8 **EVALUATING THE CAUSAL EFFECT OF EARLY-LIFE PHENOTYPIC TRAITS ON LUNG FUNCTION TRAJECTORY: A MENDELIAN RANDOMISATION STUDY IN THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN (ALSPAC)**

This abstract accepted for European Respiratory Society Congress, Barcelona, 5-9 September 2026.

S9 **ASSOCIATION OF PRESCHOOL WHEEZE ENDOTYPES WITH WHEEZE ATTACKS AND ICS TREATMENT**

Funding: Asthma UK Centre for Applied Research.

S21 **STANDBY RESCUE PACK PRESCRIBING CREATES SUBSTANTIAL UNDER-RECORDING OF COPD EXACERBATIONS IN PRIMARY CARE ELECTRONIC HEALTH RECORDS**

This work has been funded by an NIHR grant. There are no conflicts of interest in relation to this work.

S22 **PREVALENCE AND CLINICAL CHARACTERISTICS OF PATIENTS WITH COPD WHO CONTINUE TO EXACERBATE DESPITE TRIPLE THERAPY IN ENGLAND: THE SIRIUS-UK STUDY**

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S23 **BEYOND-FRONTIER: UTILISING HULL LUNG HEALTH STUDY DATA TO INVESTIGATE THE DIAGNOSTIC ACCURACY OF N-TIDAL DIAGNOSE DIAGNOSTIC ALGORITHMS TO IDENTIFY COPD AND PRE-COPD AMONG PARTICIPANTS IN THE FRONTIER PROGRAMME.**

This study received funding from Asthma + Lung UK (reference DPG23\78) and Chiesi Ltd.

S28 **RANDOMISED CONTROLLED TRIAL OF PATIENT NAVIGATION FOR LUNG CANCER SCREENING NON RESPONDERS**

Yorkshire Cancer Research.

S35 **INHIBITION OF EXTRACELLULAR CYCLOPHILIN A SIGNALING ATTENUATES ACID ASPIRATION-INDUCED ACUTE LUNG INJURY**

Work was funded by a BJA/RCoA project grant (WKR0-2021-0010). YZ is supported by a BJA/RCoA PhD studentship (WKR0-2023-0017).

S36 **MECHANICAL VENTILATION PROMOTES INFLAMMATORY AND PROFIBROTIC SIGNALING IN THE PLEURAL SPACE FOLLOWING ACUTE LUNG INJURY**

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S39 **LBP ELEVATION INDICATES GUT BARRIER DYSFUNCTION IN OUTPATIENT AND HOSPITALISED SEVERE ASTHMA EXACERBATIONS.**

This work was supported by the Nottingham Trent University PhD studentship.

S42 **DETERMINING THE EFFECTIVENESS OF AN ONLINE FENO-GUIDED ASTHMA MANAGEMENT INTERVENTION IN PRIMARY CARE (DEFINE): A PRAGMATIC RANDOMISED CONTROLLED TRIAL**

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S49 **MECHANISTIC INSIGHTS INTO STEROID CHRONOTHERAPY IN ASTHMA.**

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S50 **EXACERBATION TRAJECTORIES IN BIOLOGIC-NAÏVE SEVERE ASTHMA PATIENTS**

This abstract was presented at American Thoracic Society Meeting 2026 in Orlando (oral presentation).

S52 **EFFECT OF TEZEPELUMAB ON LUNG FUNCTION IN PATIENTS WITH OCS-DEPENDENT SEVERE ASTHMA BY BASELINE INFLAMMATORY BIOMARKER LEVELS: RESULTS FROM THE PHASE 3 SUNRISE STUDY**

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S54 **TWICE-YEARLY DEPEMOKIMAB DEMONSTRATES EFFICACY IN PATIENTS WITH ASTHMA AND COMORBID CHRONIC RHINOSINUSITIS WITH NASAL POLYPS (CRSwNP): PHASE III SWIFT-1/-2 STUDIES**

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S55 MOLECULAR DETECTION REVEALS PATHOGEN-SPECIFIC INFLAMMATORY SIGNATURES IN BRONCHIECTASIS

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S57 CROSS-COMPARTMENT INFLAMMATORY PROGRAMMES IDENTIFIED BY MULTI-OMIC INTEGRATION ARE ASSOCIATED WITH DISEASE SEVERITY AND INFECTION IN BRONCHIECTASIS: RESULTS FROM EMBARC-BRIDGE

This abstract was presented at the European Respiratory Society Congress 2026.

S58 PATIENT-REPORTED SPUTUM COLOR AND ITS ASSOCIATION WITH NEUTROPHIL SERINE PROTEASES AND EXACERBATIONS IN PATIENTS WITH BRONCHIECTASIS: A POST HOC ANALYSIS FROM THE ASPEN TRIAL

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S59 THE BRACE STUDY: A MULTICENTRE UK AUDIT OF MULTIDISCIPLINARY BRONCHIECTASIS CARE

Collaborative agreement with Insmed Incorporated.

S60 EFFECT OF BRENSOCATIB ON PATIENT-REPORTED SYMPTOMS IN PATIENTS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS: A POST HOC ANALYSIS OF QOL-B RSS INDIVIDUAL ITEMS FROM THE ASPEN PHASE 3 TRIAL

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S64 EXTERNAL VALIDATION OF MORTALITY PREDICTION MODELS IN IDIOPATHIC PULMONARY FIBROSIS USING REAL-WORLD DATA FROM THE BRITISH THORACIC SOCIETY (BTS) INTERSTITIAL LUNG DISEASE (ILD) REGISTRY

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S68 LEFT BREATHLESS: GAPS IN BASIC ASTHMA CARE FOR CHILDREN AND YOUNG PEOPLE IN ENGLAND — A RETROSPECTIVE COHORT STUDY

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S72 EFFICACY AND SAFETY OF TWICE-YEARLY DEPEMOKIMAB IN ADOLESCENT PATIENTS WITH ASTHMA: SUBGROUP ANALYSIS OF THE SWIFT-1/-2 STUDIES

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S75 MULTI-NIGHT HOME SLEEP APNEA TESTING IMPROVES DIAGNOSTIC ACCURACY COMPARED WITH SINGLE-NIGHT ASSESSMENT

Nygate, Rusk, Waters, and Fernandez are employees of EnsoData, Inc. Watson holds a consultation relationship with EnsoData, Inc., which developed and markets the PPG-based home sleep apnea testing platform used in this study. Manuel reports no conflicts of interest.

S78 CPAP IS ASSOCIATED WITH IMPROVEMENTS IN REAL-WORLD DRIVING PERFORMANCE AND SLEEP QUALITY IN OBSTRUCTIVE SLEEP APNOEA

This research was funded by the Royal Papworth Charity.

S79 DEVELOPMENT AND VALIDATION OF PHENOTYPING ALGORITHMS FOR ACCURATE IDENTIFICATION OF ADULTS WITH CYSTIC FIBROSIS IN ELECTRONIC HEALTH RECORDS

Funded by the Cystic Fibrosis Trust.

S94 MEASURING RESPIRATORY VOLUMES WITH A 3D DEPTH CAMERA

This research was funded by Engineering and Physical Sciences Research Council, grant number EP/W003600/1.

S98 JAK INHIBITION ATTENUATES RHINOVIRUS-DRIVEN INTERFERON RESPONSES IN COPD AIRWAY EPITHELIUM WITHOUT ENHANCING VIRAL REPLICATION: A SINGLE-CELL TRANSCRIPTOMIC STUDY

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S104 EXPLORING COSTS ASSOCIATED WITH SUSPECTED NON-VENTILATOR-ASSOCIATED HOSPITAL-ACQUIRED PNEUMONIA (NV-HAP) WITHIN THE HAP-FAST TRIAL

The HAP-FAST study was funded by a National Institute of Health Research Advanced Fellowship award (NIHR300669).

S107 A RANDOMISED TRIAL EVALUATING ADHERENCE AND SAFETY OF RIFAPENTINE-BASED TB PREVENTIVE TREATMENT

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S108 **MICROBIOLOGIC OUTCOMES AND RECURRENCE IN PATIENTS WITH NEWLY DIAGNOSED MYCOBACTERIUM AVIUM COMPLEX LUNG DISEASE (MACLD) IN THE PHASE 3B ENCORE TRIAL**

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S109 **SINGLE-CELL AND SPATIAL DISSECTION OF AIRWAY NICHES REVEAL DISTINCT IMMUNE AND EPITHELIAL DRIVERS OF ASTHMA PHENOTYPES**

This study was founded by Arcturis Data, Kymab (Sanofi), and MRC grant MR/R015708/1.

S114 **A 3D MULTI-OMICS ATLAS OF HUMAN LUNG FIBROSIS DEFINES MOLECULAR–ARCHITECTURAL NICHES**

This abstract was presented at European Respiratory Society Lung Science Conference March 2026.

S116 **STANDARDISING PLEURAL MANAGEMENT IN SUSPECTED MALIGNANT PLEURAL EFFUSION USING UNIFIED PRE-TEST PROBABILITY AND THE RED SCORE: A QUALITY IMPROVEMENT PROJECT**

This abstract was presented at All Wales Pleural Seminar 13/11/2026.

S118 **PLEURAL COMPLICATIONS ASSOCIATED WITH TYROSINE KINASE INHIBITORS**

This abstract was presented as a different abstract using similar data at European Respiratory Society Congress 2026, this September.

S125 **TRANSCUTANEOUS CO_2 MONITORING VERSUS ARTERIAL $PACO_2$ DURING ACUTE NIV IN ACUTE HYPERCAPNIC RESPIRATORY FAILURE: A SYSTEMATIC REVIEW**

This abstract is an encore presentation.

S126 **COST-EFFECTIVENESS OF ACCELERATED VERSUS STANDARD WEANING FROM NIV IN COPD: AN ECONOMIC EVALUATION WITHIN A RANDOMISED CONTROLLED TRIAL**

The RCT was funded by NIHR and loan devices from Radiometer. Neither had contribution to the trial design, data collection, analysis or writing of results.

S131 **ROBUSTNESS OF TIME-TO-EVENT BENEFITS ASSOCIATED WITH BENRALIZUMAB-CONTAINING TREATMENT IN EOSINOPHILIC ASTHMA AND COPD: SENSITIVITY ANALYSES FROM THE ABRA TRIAL**

This work is funded by the National Institute of Health and Care Research (NIHR304263).

S133 **RISK STRATIFICATION AND LONGITUDINAL ASSESSMENT OF INTERSTITIAL LUNG ABNORMALITIES USING E-LUNG QUANTITATIVE CT BIOMARKERS**

Conflict of interest for PM George and A Devaraj exist in relation to Brainomix. A. Devaraj is a medical director and holds stock options for Brainomix. PM George is Senior Medical Director and holds stock options for Brainomix. Additionally e-Lung software is currently deployed and in use at the relevant hospital as part of a zero cost trial.

S135 **CHARACTERISATION OF INTERSTITIAL LUNG ABNORMALITIES IN A UK MULTICENTRE ILA CLINIC COHORT: EARLY FINDINGS FROM THE ILARC-UK PILOT**

RJH is supported by the Sir Robert Finch Fellowship from the Royal Brompton and Harefield Hospitals Charity (F44/C0097), Asthma + Lung UK Early Career Starter Grant (ECSG24\36) and King's Health Partners Centre for Translational Medicine Translational Medicine Pilot Award. RJH has received, unrelated to the submitted work, speaker fees from Boehringer Ingelheim.

S143 **USING HYPERPOLARISED 129XENON AND PROTON MRI TO DETECT IMPAIRED GAS EXCHANGE IN STRUCTURALLY NORMAL LUNG REGIONS IN SYSTEMIC SCLEROSIS PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION**

Funded by Heart Research UK.

S152 **LUNG EPITHELIAL CELL-DERIVED LEUCINE-RICH ALPHA2 GLYCOPROTEIN-1 (LRG1) DRIVES THE DEVELOPMENT OF TUBERCULOSIS-INDUCED PULMONARY FIBROSIS**

Financial support for this study was provided by Legacy St George's, University of London-funded PhD studentship for Dr J Kutschenreuter and a new lecturer start-up grant awarded to Dr D Chong and a Rosetrees Trust funded-Intermediate Research Grant (PGS22/100080).

S154 **GαQ/11 MODULATES THE EXTRACELLULAR MATRIX AND FIBROBLAST-EPITHELIAL INTERACTIONS TO CONTROL ALVEOLAR REPAIR**

Funded by an ALUK Malcolm Weallans Pulmonary Fibrosis Grant and an Academy of Medical Sciences Starter Grant for Clinical Lecturers.

P8 **TRANSFORMING TRAINING IN LUNG CANCER DIAGNOSTICS: EVALUATION OF NOVEL PATIENT-DERIVED LUNG PHANTOMS**

Efthymios Maneas and Adrien Desjardins are co-founders of OmioLabs and were involved in the development of the lung phantom evaluated in this work. Neal Navani and Ricky Thakrar serve as Clinical Advisors to OmioLabs.

P19 **EVALUATING CLINICAL DELIVERY COSTS OF A VIRTUAL GROUP PHYSIOTHERAPY INTERVENTION (VIP-COUGH) VERSUS TRADITIONAL ONE-TO-ONE COUGH SUPPRESSION THERAPY**

Dr Charles Reilly holds a National Institute for Health and Care Research (NIHR) Advanced Clinical and Practitioner Academic Fellowship (NIHR302904). The views expressed in this publication are those of the author(s) and not necessarily those of the NHS, NIHR or the Department of Health and Social Care.

P20 **PREVALENCE AND CHARACTERISTICS OF BREATHING PATTERN DISORDERS IN ADULTS WITH REFRACTORY CHRONIC COUGH OR INDUCIBLE LARYNGEAL OBSTRUCTION: A SYSTEMATIC REVIEW**

This work was supported by the NIHR as part of a Predoctoral Fellowship.

P28 **INCIDENCE AND PREDICTORS OF FUTURE ASTHMA DIAGNOSIS IN SUBJECTS WITH COUGH.**

Study has received funding from Kuopion seudun hengityssäätiö.

P30 **THE PULSE-PH REGISTRY: REAL-WORLD UK DATA IN PATIENTS WITH INTERSTITIAL LUNG DISEASE AND SARCOIDOSIS-ASSOCIATED PULMONARY HYPERTENSION**

This abstract is planned to be presented at the European Respiratory Society Congress 2026.

P44 **FEASIBILITY AND ACCEPTABILITY OF N-TIDAL DIAGNOSE: COMMUNITY LUNG HEALTH EVENT FEEDBACK**

N-Tidal Diagnose device funding from Health Innovation Network – Respiratory Transformation Partnership (HIN-RTP): Pathway transformation funding. TW/MG/ZS have received research funding from TidalSense and TW has received consulting fees from TidalSense. GL is currently employed by TidalSense.

P45 **TARGETED LUNG HEALTH CHECKS: ARE WE MISSING THE OPPORTUNITY TO DIAGNOSE AND TREAT COPD?**

This abstract was presented at the European Respiratory Society Congress 2026 (September 5-9).

P47 **TARGETED EDUCATION IS A POTENTIAL STRATEGY TO IMPROVE THE LIVED EXPERIENCE OF YOUNG ADULTS (16-25YRS) WITH SEVERE ASTHMA.**

Funding from the Burdett nursing trust to undertake the study.

P49 **'NOTHING ABOUT ME WITHOUT ME'. YOUNG PEOPLE WITH ASTHMA AND UPPER AIRWAY DISORDERS TRANSITIONING TO ADULT SERVICES: SERVICE EVALUATION OF A UK HOSPITAL**

Fellowship funded by the OUH Hospital Charities.

P50 **RESEARCH EQUITY BY DESIGN: INSIGHTS FROM THE A-TREC COHORT**

Funding for this study was provided by Northumbria Healthcare NHS Foundation Trust, GSK and Chiesi.

P52 **DEVELOPING A BLUEPRINT FOR USING CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) TO ILLUSTRATE BEST NEIGHBOURHOOD HEALTH AND OUTCOMES ACROSS THE UK**

This work is being funded by PCRS core funds, and not directly by any pharmaceutical companies. However, PCRS corporate sponsors include AstraZeneca UK Ltd, Chiesi Ltd, GlaxoSmithKline (GSK) plc, Lupin Healthcare Limited, and Trudell Medical.

P53 **FINDINGS FROM THE HERON NEEDS PRIORITISATION PARTNERSHIP: EXPLORING AND MAPPING UNMET NEEDS AND POTENTIAL HEALTH TECHNOLOGY SOLUTIONS IN CHRONIC RESPIRATORY DISEASE**

ZO, SP, PT, GH and HA work within, and receive funding from, the NIHR HealthTech Research Centre for Community Healthcare at Oxford Health NHS Foundation Trust. The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR or the Department of Health and Social Care. This project is funded by LifeArc, a not-for-profit medical research charity.

P54 **EXPERIENCES OF SPECIALIST NURSE REVIEW DELIVERED REMOTELY IN PROGRESSIVE PULMONARY FIBROSIS**

This work was funded though Oxford University Hospitals NHS Foundation Trust and The Oxford Hospitals Charity.

P56 **RIGHT CARE, EVERY TIME: DESIGNING PATIENT AND HEALTHCARE PROVIDER CHECKLISTS TO SUPPORT NON TUBERCULOUS MYCOBACTERIA PULMONARY DISEASE (NTM-PD) MANAGEMENT ACROSS THE UK**

Funding for medical writing and editorial support of the six NTM checklists was provided by Insmed Limited to Axiom Health Ltd. Insmed Limited had no involvement in checklist content or this abstract.

P62 **LOCAL, BUT NOT SYSTEMIC, NEUTROPHIL-DRIVEN INFLAMMATION ASSOCIATES WITH LUNG PATHOLOGY IN TUBERCULOSIS**

This work was supported by a Rosetrees Trust PhD-Plus Award (PhD2023\100011, DLWC and JK) and a St George's University Hospital NHS Foundation Trust Translational and Clinical Research Institute Fellowship (DEK).

P67 **CAN AN RD-1 BASED SKIN TEST STRATEGY IMPROVE THE TB INFECTION CARE CASCADE WHERE IGRA IS STANDARD?**

NIHR (RP-PG-0217-20009).

P69 **DECISION-MAKING IN LATENT TUBERCULOSIS TESTING: QUALITATIVE INSIGHTS ON NOVEL TB-SPECIFIC SKIN TESTS**

Funder: NIHR RID-TB Programme [RP-PG-0217-20009].

P73 **BENRALIZUMAB DOES NOT ALTER THE AIRWAY MICROBIOME IN SEVERE ASTHMA, BUT EXACERBATIONS ARE ASSOCIATED WITH INCREASED DYSBIOSIS IDENTIFIED BY THE LOG_2 PSEUDOMONADOTA:BACILLOTA RATIO**

BenRex was a phase IV, open-label, prospective, multicentre cohort study conducted across 15 centres in the United Kingdom. Participants with severe eosinophilic asthma were recruited after being prescribed benralizumab by a specialist in

accordance with national prescribing criteria. The study was investigator-initiated and funded by AstraZeneca, which also supplied benralizumab injections. AstraZeneca had no involvement in the study design, conduct, data analysis, interpretation of findings, preparation of the abstract, or dissemination of the results.

P78 EFFECT OF TEZEPELUMAB ON TYPE 2 INFLAMMATORY BIOMARKER LEVELS IN THE NASAL LINING FLUID OF PATIENTS WITH CRSWNP AND COMORBID ASTHMA

Funding: This study was funded by AstraZeneca and Amgen Inc. Acknowledgments: Medical writing support was provided by Freyja McClenahan, PhD, on behalf of PharmaGenesis London, London, UK, with funding from AstraZeneca and Amgen Inc. Disclosures: Brian J Lipworth reports nonfinancial 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; has received 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. Claudio Marchese, Claudia Chen, Andrew Foster, Sandhia S Ponnarambil and Joseph D Sherrill are employees of AstraZeneca and may own stock or stock options in AstraZeneca. Jyotsna Reddy, Jane R Parnes and Jean Publicover are employees of Amgen and own stock in Amgen.

P79 EFFECT OF TEZEPELUMAB ON LUNG FUNCTION IN ORAL CORTICOSTEROID-DEPENDENT PATIENTS WITH SEVERE ASTHMA AND COMORBID CRSWNP OR CRSSNP IN THE PHASE 3B WAYFINDER STUDY

Funding: This study was funded by AstraZeneca and Amgen Inc. Acknowledgments: Medical writing support was provided by Shanelle Oko-Osi, PhD, of PharmaGenesis London, London, UK, with funding from AstraZeneca and Amgen Inc. Disclosures: DJJ has received advisory board and speaker fees from AstraZeneca, GSK, Novartis, Sanofi and Teva Pharmaceuticals, and has received research grant support from AstraZeneca and GSK. PC 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 » from 2021–2025. NLL has received consulting fees from AbbVie, Amgen, Apogee, AstraZeneca, Avillion, Foresee, Genentech, GSK, Inctye, Niox, Novartis, Regeneron Pharmaceuticals, Sanofi, and Teva Pharmaceuticals; has received fees for non-speakers bureau presentations from GSK, Teva Pharmaceuticals, Sanofi/Regeneron Pharmaceuticals and Astra Zeneca; and has received travel support from AstraZeneca, Sanofi, Teva Pharmaceuticals, Regeneron Pharmaceuticals and GSK; her institution received research support from Amgen, AstraZeneca, Avillion, Bellus, Evidera, Gossamer Bio, Genentech, GSK, Janssen, Regeneron Pharmaceuticals, Roche, Sanofi, Novartis and Teva Pharmaceuticals. She is an honorary faculty member of Observational and Pragmatic Research Institute (OPRI) but does not receive compensation for this role. MG has attended advisory board meetings for AstraZeneca and has received speaker fees from AstraZeneca, HRA Pharma and Novartis. LGH 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 for 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. SK has received fees for lectures and/or advisory board meetings from AstraZeneca, GSK, Novartis, Roche, Sanofi Aventis and Teva Pharmaceuticals. GB has received fees for advisory boards and/or speaker fees from Amgen, AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, MSD, Novartis and Sanofi. RdO has received research, advisory board and speaker fees from AstraZeneca, Boehringer Ingelheim, Gador, GSK, Mundipharma, Novartis and Sanofi Genzyme; and travel support from AstraZeneca, GSK and Sanofi. LB is an employee of Amgen and owns stock in Amgen. KS, CM and SC are employees of AstraZeneca and may own stock or stock options in AstraZeneca.

P81 TEZEPELUMAB TO IMPROVE SYMPTOM AND TREATMENT BURDEN IN SEVERE ASTHMA: AN ENGLAND BASED SINGLE-CENTRE RETROSPECTIVE COHORT STUDY

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. The authors declare no competing interests.

P82 A COMPREHENSIVE ASSESSMENT OF THE IMPACT OF ANTI-IL5 THERAPY ON WELLBEING, ILLNESS PERCEPTION AND COGNITION IN SEVERE ASTHMA: LONGITUDINAL DATA FROM THE CONTENTED STUDY.

Funding for this study was provided by GSK [ISRCTN74240789]. GSK was provided the opportunity to review a preliminary version of this publication for factual accuracy, but the authors are solely responsible for the final content and interpretation.

P83 DUPILUMAB IMPROVES SPIROMETRY- AND OSCILLOMETRY-DERIVED RATIOS INDICATING LARGE AND SMALL AIRWAY FUNCTION IN PATIENTS WITH MODERATE-TO-SEVERE TYPE 2 ASTHMA: VESTIGE

Lipworth BJ: AstraZeneca, Chiesi, Circassia, Glenmark, Lupin, Regeneron Pharmaceuticals Inc., Sanofi – consultant, speaker fees, participation on advisory boards. Taube C: ALK, Chiesi, – speaker fees; AstraZeneca, GSK, Sanofi – speaker fees, advisory board. 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. Shirai T: AstraZeneca, GSK, Kyorin, Sanofi – speaker fees. Wen F: National Natural Science Foundation Committee of China – grant support. González-Barcala F-J: ALK, AstraZeneca, BIAL, Boehringer Ingelheim, Chiesi, Gebro Pharma, GSK, Laboratorios Esteve, Menarini, Mundipharma, Novartis, Rovi, ROXALL, Sanofi, Stallergenes Greer, Teva – speaker fees, consulting fees, research grants. Mohan A: Verona Pharma LLC – consulting fees. Altincatal A, Awad H: Sanofi – employees, may hold stock and/or stock options in the company. Gall R: Regeneron Pharmaceuticals Inc. – employee and shareholder. Castro M: American Lung Association, AstraZeneca, Gala Therapeutics, Genentech, GSK, NIH, Nocion, Novartis, PCORI, Pfizer, PULMATRiX, Sanofi, Shionogi, Teva, Theravance Biopharma, Uniquity – grants/research support; Allakos, Amgen, Apogee, Apreo, Arrowhead Pharmaceuticals, AstraZeneca, Connect BioPharma, Evommune, Genentech, GSK, Jasper, Merck, Novartis, OM Pharma, Pfizer, Pioneering Medicines, Regeneron Pharmaceuticals Inc., Sanofi, Teva, Upstream Bio – consultant; Amgen, AstraZeneca, Genentech, Regeneron Pharmaceuticals Inc., Sanofi, Teva – speaker fees; Aer Therapeutics, Elsevier – royalties.

P84 TWICE-YEARLY DEPEMOKIMAB MAINTAINS EXACERBATION REDUCTION AND CLINICAL REMISSION OVER 2 YEARS IN PATIENTS WITH ASTHMA, REGARDLESS OF BASELINE ASTHMA DURATION: SWIFT/AGILE INTEGRATED ANALYSIS

Funding: GSK (SWIFT-1/-2: 206713/213744,NCT04719832/NCT04718103; AGILE: 212895,NCT05243680). vConflicts of Interest MK has received research funding paid to her institution from NIH, ALA, Sanofi and Areteia; she receives personal fees from GSK, Sanofi, Regeneron, Genentech, Chiesi, AstraZeneca and Kinaset. NJ has received consulting fees from GSK, AstraZeneca and Chiesi. RB has received consulting/speaking honoraria from AstraZeneca, Berlin-Chemie, Boehringer Ingelheim, Celltrion, Chiesi, GSK, Novartis and Sanofi. CT, DM and C-QZ are employed by GSK and hold financial equities in GSK. DJJ has received consulting/speaking honoraria from GSK, AstraZeneca and Sanofi Regeneron.

P87 FEVER-DEPENDENT WHO SARI CRITERIA INCOMPLETELY ASCERTAIN ADULT HOSPITALISED RESPIRATORY VIRAL INFECTION

The UNIVERSAL study was supported by Janssen Pharmaceuticals, GlaxoSmithKline, Sanofi and AstraZeneca, with BioFire FilmArray systems/kits and operational support provided by Synairgen Research Ltd and additional systems/kits provided by bioMérieux SA.

P89 HEALTHCARE UTILISATION AFTER HOSPITALISATION FOR INFLUENZA IN ENGLAND: A POPULATION-BASED STUDY.

This study is funded by the National Institute for Health and Care Research (NIHR) Nottingham Biomedical Research Centre (BRC). SM, TMM and CEB are supported by the NIHR Nottingham BRC. CEB has received research grant funding to support projects in recovery from acute respiratory infection from NIHR, University of Nottingham charitable donations and Nottingham Hospitals Charity. No other conflict of Interests reported by other authors.

P107 HIGH AMBIENT TEMPERATURE AND RESPIRATORY EMERGENCY ADMISSIONS DURING THE WARM SEASON: A DAILY TIME-SERIES ANALYSIS

The authors declare no conflicts of interest. JL was supported by a Future 50 PhD Scholarship from the University of Leicester. YSC and AH acknowledge funding from the National Institute for Health and Care Research (NIHR) Leicester Biomedical Research Centre (BRC) (NIHR203327) and the NIHR Health Protection Research Unit (HPRU) in Chemical Threats and Hazards at the University of Leicester, a partnership between the UK Health Security Agency, the Health and Safety Executive and the University of Leicester. AH also acknowledges support from a British Heart Foundation Research Excellence Award (RE/24/130031). The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR, the Department of Health and Social Care, the UK Health Security Agency or the British Heart Foundation. The funders had no role in the study design, analysis or interpretation of data, decision to publish, or preparation of the manuscript.

P112 SHORT-TERM AIR POLLUTION EXPOSURE AND RISK OF AIRWAY INFLAMMATORY RESPONSE IN CHILDREN (CHERISH): A RANDOMISED MIXED FACTORIAL STUDY.

This work was supported by Barts Charity (Grant No: G-002887).

P123 **MB, AS, CM, RW AND SJF ARE SUPPORTED BY THE MANCHESTER NIHR OPTIMISING DIAGNOSTIC TEST THRESHOLDS AND PATHWAYS FOR ADULT ASTHMA**

Biomedical Research Centre (BRC) (NIHR203308), MB is also supported by Wellcome (223521/Z/21/Z). HD holds a MRC CSF MR/V029460/1.

P126 **MULTIMORBIDITY CLUSTERS AND ASSOCIATED CHARACTERISTICS IN MILD, MODERATE AND SEVERE ASTHMA COHORTS**

RW is funded by NIHR Clinical Lectureship

P127 **BEYOND THE WHEEZE: A PHENOTYPE-DRIVEN APPROACH TO ASTHMA EXACERBATIONS**

Funded by AstraZeneca.

P128 **LONGER ASTHMA DURATION IS ASSOCIATED WITH ELEVATED NON-T2 SPUTUM BIOMARKERS AND REDUCED T2 INFLAMMATION IN SEVERE ASTHMA**

This abstract was presented at European Respiratory Society Meeting 2026 Barcelona (accepted for oral presentation).

P129 **EARLY IDENTIFICATION OF "HIGH-RISK" ASTHMA AND PROGRESSION TO BIO-ELIGIBILITY: A RETROSPECTIVE COHORT STUDY USING UK DATA**

Funding: HDR-UK. A manuscript based on this research has been submitted to Thorax for consideration.

P131 **SARCOPENIA IN ASTHMA: A CROSS-SECTIONAL ASSESSMENT OF MUSCLE STRENGTH, MASS AND PHYSICAL PERFORMANCE REEM ALAJMI, IAN J. CLIFTON, DANIEL PECKHAM, T. SCOTT BOWEN, OLIVER J. PRICE**

This study is funded by the University of Leeds. The authors declare that they have no conflicts of interest.

P138 **CARDIOPULMONARY EVENT OUTCOMES AND RISKS IN COPD ACCORDING TO GOLD STAGE - AN OBSERVATIONAL STUDY IN UK PRIMARY AND SECONDARY CARE**

This study was sponsored by AstraZeneca UK. DS reports consultancy and speaker fees from AstraZeneca, GSK, Boehringer Ingelheim, Chiesi, Cipla, Pfizer, Sanofi, Regeneron and TEVA. MJH, MW and JL are all employees of and may hold stock in AstraZeneca. DMGH reports honoraria for lecturing, attending advisory boards, sitting on DSMBs and preparing educational materials from AstraZeneca, Berlin-Chemie, Boehringer Ingelheim, Chiesi, Cipla, GlaxoSmithKline, Inogen, Pfizer, Menarini, Novartis, Sanofi, Windward Bio and Zuellig Pharma. Member Board of Directors & Science Committee GOLD. CPG is the Founder and Director of FIND-Health Ltd. He reports Institutional grants from British Heart Foundation, National Institute for Health Research, Horizon 2020, European Society of Cardiology, Abbott Diabetes, Bristol Myers Squibb and Honoraria from AstraZeneca, Amgen, Bayer, Boston Scientific, Bristol Myers Squibb, Berlin-Chemie, Boehringer Ingelheim, Chiesi, Daiichi Sankyo, GPRI Research B.V., Menarini, Novartis, iRhythm.

P139 **OPPORTUNISTIC THORACIC CT-AI BODY COMPOSITION AS A SARCOPAENIA CASE-FINDING FLAG IN RESPIRATORY DISEASE: DXA VALIDATION**

No disclosures

P141 **REGIONAL MUCUS PLUG BURDEN PREDICTS ADVERSE OUTCOMES IN COPD PATIENTS IDENTIFIED THROUGH LUNG CANCER SCREENING: AN AUTOMATED QUANTITATIVE CT ANALYSIS**

Wellcome Trust.

P143 **A STUDY OF USE OF MRI TO MEASURE MODULATION OF EOSINOPHIL RELATED INFLAMMATION IN COPD**

Source of funding: Grant given by GlaxoSmithKline.

P144 **50 SHADES OF GREEN: PROOF-OF-CONCEPT FOR DIGITAL SPUTUM COLOUR QUANTIFICATION**

The POSTED study was supported by Asthma + Lung UK and a VIA award from the Cystic Fibrosis Trust. The authors declare no other conflicts of interest.

P145 **ABSTRACT WITHDRAWN**

P149 **EQUITY OF ACCESS TO VIRTUAL WARD (VW) CARE FOR EXACERBATIONS OF COPD: A RETROSPECTIVE COHORT STUDY**

This work forms part of a PhD project.

P155 **SURVIVAL AFTER SURGICAL LUNG BIOPSY IN INTERSTITIAL LUNG DISEASE: INFLUENCE OF HISTOPATHOLOGICAL DIAGNOSIS. A RETROSPECTIVE COHORT STUDY.**

This abstract was presented at Scottish Thoracic Society Annual meeting 2026.

P160 **INCIDENCE, PREVALENCE AND THE DIAGNOSTIC ODYSSEY IN RARE CYSTIC LUNG DISEASE**

This work is funded by NIHR Nottingham Biomedical Research Centre, Nottingham.

P162 **PREVALENCE AND CLINICAL MANAGEMENT OF PATIENTS WITH ILD ASSOCIATED WITH PH – A UK REGIONAL ILD CENTRE PERSPECTIVE**

This abstract was presented at European Respiratory Society Conference 2026.

P165 **ANTI-CARBAMYLATED PROTEIN ANTIBODIES IN FIBROINFLAMMATORY LUNG DISEASE INCLUDING RHEUMATOID ARTHRITIS & SARCOIDOSIS**

This abstract was presented at BALR Annual Meeting 8-10th September 2025, Cardiff.

P170 **THE DIAGNOSIS DETECTIVES: IMPROVED RECOGNITION OF AETIOLOGY IN BRONCHIECTASIS THROUGH A REGIONAL DIAGNOSTIC CLINIC**

This abstract planned presentation: World Bronchiectasis Conference June 2026.

P174 **FEASIBILITY AND ACCEPTABILITY OF USING A SHARED DECISION MAKING INTERVENTION TO SUPPORT PATIENT CHOICE OF AIRWAY CLEARANCE TECHNIQUES IN BRONCHIECTASIS**

This study is part of the authors National Institute for Health and Care Research fellowship on the co-production of a SDM intervention for ACTs in bronchiectasis (reference: NIHR302892).

P185 **SELF REQUEST CHEST X RAY PATHWAY: A LOW COST STRATEGY TO ACCELERATE LUNG CANCER DETECTION IN SYMPTOMATIC PATIENTS**

This model was presented at the BTOG Annual Conference 2026. The abstract has been updated by integrating data from previous abstracts of the same study.

P191 **STREAMLINING LUNG MULTI-DISCIPLINARY TEAM MEETINGS USING AI BASED CLINICAL DECISION SUPPORT SYSTEM: RETROSPECTIVE DATA ANALYSIS TO VALIDATE STANDARD OF CARE**

This analysis is part of a collaborative working initiative between Barts Health NHS Trust and AstraZeneca.

P193 **THE VALUE OF CIRCULATING TUMOUR DNA IN SECONDARY CARE THORACIC ONCOLOGY: SEARCH AND RESCUE**

This abstract was presented at The abstract has been submitted to European Respiratory Society Congress 2026, and is currently under consideration. If successful, the abstract would be presented at ERS Congress before the BTS Winter Meeting, so this has been disclosed for transparency.

P198 **PREVALENCE AND INFLUENCE OF OXYGEN TOXICITY IN COPD EXACERBATIONS REQUIRING NON-INVASIVE VENTILATION.**

NIHR- RfPB.

P214 **DEVELOPING INTERVENTIONS TO TACKLE ETHNICITY-BASED DISPARITIES IN ASTHMA MANAGEMENT IN BRITISH SOUTH ASIANS "TEAMS STUDY"**

This study is funded through an NIHR grant number NIHR207152.

P218 **BARRIERS AND FACILITATORS TO IMPLEMENTING ANTI-INFLAMMATORY RELIEVER-BASED ASTHMA CARE IN COMMUNITY PHARMACY: A QUALITATIVE STUDY**

HH – none to declare. JD has received a fee for delivering a conference presentation for AstraZeneca and honoraria for offering independent study design support to Astellas Pharma Inc. DS – has received honoraria from AstraZeneca and Trevi Therapeutics, speakers fees from AstraZeneca and Chiesi Ltd. GG, KS and MW GC are employees of AstraZeneca and may own shares in AstraZeneca. LB and MH None to declare. MC 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.

P219 **CAN HOME SPIROMETRY AND FENO TESTING IMPROVE ASTHMA DIAGNOSIS? – A FEASIBILITY STUDY**

RW is funded by NIHR Clinical Lectureship.

P220 **DEPEMOKIMAB DEMONSTRATES SUSTAINED LONG-TERM EFFICACY AND CONSISTENT PATIENT-REPORTED OUTCOMES OVER 2 YEARS IN PATIENTS WITH TYPE 2 ASTHMA IRRESPECTIVE OF BASELINE INHALED CORTICOSTEROID DOSE: AN INTEGRATED ANALYSIS OF THE PHASE III SWIFT-1/-2 AND THE OPE**

Funding: GSK (SWIFT-1/-2:206713/213744, NCT04719832/NCT04718103; AGILE:212895/NCT05243680). RAP: Research grant, Consultant- AstraZeneca; Research grant-GSK, NIH, Origo Pharma; Consultant: Regeneration, Sanofi; MB: Research grant, Advisory committee member, Consultant, Speaker: AstraZeneca, Chiesi, GSK, Sanofi; Speaker: Grifols, Lusofarmaco, Menarini, Advisory committee member: Niox; Consultant: Omron; C-QZ, DJ, CT and PH: Employee, Stocks or options held: GSK; DJJ: Consultant : AstraZeneca, GSK, Sanofi Regeneron.

P221 **PHENOTYPING SEVERE ASTHMA ATTACKS IN HOSPITALISED PATIENTS**

This abstract was presented at at the European Respiratory Society Congress on Exacerbations of Airways Disease (Barcelona 22/1/26).

P222 **ECHOCARDIOGRAPHIC PROBABILITY OF PULMONARY HYPERTENSION AND THE ASSOCIATION WITH 90-DAY MORTALITY AND POST-OPERATIVE COMPLICATIONS IN PEOPLE UNDERGOING LUNG CANCER RESECTION SURGERY**

This abstract was presented at BTOG 2026 (4-6th March) at Edinburgh- this was with a lower dataset of 142 people. This submission has a larger dataset of 250 people and has shown significance in certain areas.

P226 **SMOKING CESSATION IN COPD: A MIXED-METHODS ANALYSIS OF BEHAVIOURAL, SOCIOECONOMIC AND HEALTHCARE DETERMINANTS IN REAL-WORLD PRACTICE**

Yerramasu.C, Honorarium from GSK, AZ and Chiesi, and conference funding from Chiesi and GSK, Phd Innovation fellowship Chief Scientist Office (Scottish Government). Preece. R, honorarium Pfizer, Choudhury.G, COPD lead for Scotland, paid honorarium and grants from AZ, Chiesi, Sanofi, GSK, BI.

P230 **EFFECTIVENESS OF PULMONARY REHABILITATION ACROSS INDIVIDUAL CHARACTERISTICS: FINDINGS FROM THE NATIONAL RESPIRATORY AUDIT PROGRAMME**

This study is funded by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme [223512/Z/21/Z]. This study is supported by 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. R. Evans reports support by institutional research grants from the NIHR, UKRI, Wolfson Foundation, Genentec/Roche; and personal fees for consulting from AstraZeneca and lecture honoraria from Moderna. J.K Quint has been supported by institutional research grants from UKRI, NIHR, Health Data Research UK, BI, AZ, Insmed, and received personal fees for advisory board participation, consultancy or speaking fees from GlaxoSmithKline, BI. She is the analysis lead for the NRAP. E. Daynes reports consultancy fees from the Royal College of Physicians and Chiesi.

P231 **DESIGNING THE UPTURN INTERVENTION TO SUPPORT UPTAKE OF PULMONARY REHABILITATION: A COLLABORATIVE CO-DESIGN PROCESS**

This abstract was presented at International Behaviour Change conference Lisbon 2026. The focus of the oral presentation will differ in the BTS conference where the emphasis is the co-design process and final intervention.

P234 **EXPERIENCE BASED CO-DESIGN TO ADAPT PULMONARY REHABILITATION FOR SOUTH ASIAN INDIVIDUALS**

This study is funded by the Wellcome Trust as part of the Leicestershire Health Inequalities Improvement Doctoral Training Programme [223512/Z/21/Z]. This study is supported by the National Institute for Health and Care Research (NIHR) Leicester Biomedical Research Centre (BRC). R. Evans reports support by institutional research grants from the NIHR, UKRI, Wolfson Foundation, Genentec/Roche; and personal fees for consulting from AstraZeneca and lecture honoraria from Moderna. J.K Quint has been supported by institutional research grants from UKRI, NIHR, Health Data Research UK, BI, AZ, Insmed, and received personal fees for advisory board participation, consultancy or speaking fees from GlaxoSmithKline, BI. She is the analysis lead for the NRAP. E. Daynes reports consultancy fees from the Royal College of Physicians and Chiesi.

P235 **CO-DESIGN OF A VERY BRIEF BEHAVIOURAL CHANGE APPROACH TO ASSIST REFERRERS DISCUSS PULMONARY REHABILITATION WITH COPD PATIENTS**

This study is funded by the NIHR [Dr. Claire Nolan's Advanced Fellowship (Award ID: NIHR303175)]. The views expressed

are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care.

P236 **'DESIGNING REHABILITATION FOR INDIVIDUALS WITH LONG COVID – THE 4PS OF ACCEPTABILITY'**

KK is undertaking a PhD funded by the Wellcome Trust.

P237 **A BEHAVIOURAL INTERVENTION FOR PATIENTS WITH LONG-COVID**

Funded by the Dowager Countess Eleanor Peel Trust.

P243 **THE PNEUMORATER CHEST-WORN SENSOR CAN CONTINUALLY MEASURE RESPIRATORY RATE AND SIGNS OF AIR HUNGER IN ADULTS WITH AND WITHOUT BREATHING PATTERN DISORDERS.**

Professor Neil White is the creator of the PneumoRater device but was not included in the analyses of these data and lent devices for the project free of charge.

P244 **AN ONLINE BREATHING AND WELLBEING PROGRAMME (ENO BREATHE) FOR PEOPLE WITH LONG COVID BREATHLESSNESS: A MIXED METHODS ANALYSIS OF 3-MONTH POST-INTERVENTION FOLLOW-UP DATA**

KEJP was supported by the Imperial College National Heart and Lung Institute Clinical Lecturer scheme. KEJP has received honoraria from Chiesi for non-promotional lectures and from GSK for conference attendance. ALe receives a £250 per annum honorarium for being the Association of Chartered Physiotherapists in Respiratory Care Research Champion. ALa is a trustee and non-executive director of the English National Opera (ENO). NSH is chair of Action on Smoking and Health (UK) and medical director at Asthma and Lung UK. JM holds the following positions: The Clod Ensemble (chair—unpaid), National Opera Studio (trustee—unpaid position), and The Lord-Lieutenant of Greater London's Council on Culture and Heritage (member—unpaid position). SM, TP, KB, BW, AC, HB, JM, SZ, and THH work for ENO, who developed and deliver ENO Breathe; however, the programme is delivered free of charge to participants. All other authors declare no competing interests.

P249 **BUDESONIDE/GLYCOPYRRONIUM/FORMOTEROL FUMARATE DIHYDRATE EFFECTS ON LUNG FUNCTION AND ASTHMA EXACERBATIONS BY PERSISTENT AIRFLOW LIMITATION STATUS: POST HOC ANALYSES OF KALOS AND LOGOS STUDIES**

This study was sponsored by AstraZeneca.

P251 **EFFECTIVENESS OF A FACILITATED IMPLEMENTATION STRATEGY TO IMPROVE ROUTINE DELIVERY OF SUPPORTED ASTHMA SELF-MANAGEMENT IN UK PRIMARY CARE: A CLUSTER RANDOMISED IMPLEMENTATION TRIAL (IMP2ART)**

Funding: NIHR RP-PG-1016-20008.

P252 **AN ECONOMIC EVALUATION OF THE IMP²ART WHOLE-SYSTEMS INTERVENTION TO SUPPORT ASTHMA SELF-MANAGEMENT IN PRIMARY CARE**

This project was funded by the NIHR.

P256 **ASSESSING THE ROLE OF SOCIAL DETERMINANTS OF HEALTH IN ASTHMA HOSPITALISATIONS USING THE CLINICAL PRACTICE RESEARCH DATALINK.**

This study was funded by a local clinical lecturer starter grant from the East Midlands North Clinical Academic Training Programme. The authors have no other disclosures related to this work.

P260 **BLOOD EOSINOPHIL COUNTS AND INCIDENT ASTHMA AND COPD: PROSPECTIVE ANALYSES IN UK ADULTS**

This abstract was presented at European Respiratory Society Congress 2026.

P261 **PREDICTORS OF BLOOD EOSINOPHIL TESTING IN MODERATE-TO-SEVERE COPD: A UK RETROSPECTIVE COHORT STUDY**

Funding: GSK (222888). Conflicts of Interests: JP, JB and SY are employees of GSK and hold financial equities in GSK. TT, PP, DM and WK are employees of Adelphi Real World; Adelphi Real World received funding to conduct the study from GSK. JKQ declares personal fees for advisory board participation or speaking fees from GSK, Boehringer Ingelheim, AstraZeneca and Chiesi, but declares no non-financial competing interests.

P262 **PERSISTENT EXACERBATION AND MORTALITY IN COPD WITH TYPE 2 INFLAMMATION DESPITE TRIPLE THERAPY: A UK RETROSPECTIVE COHORT STUDY**

Funding: GSK (222888). Conflicts of Interests: JP, SY and JB are employees of GSK and hold financial equities in GSK. TT, PP, DM and WK are employees of Adelphi Real World; Adelphi Real World received funding to conduct the study from GSK. JKQ declares personal fees for advisory board participation or speaking fees from GSK, Boehringer Ingelheim, AstraZeneca and Chiesi, but declares no non-financial competing interests.

P266 MEPOLIZUMAB REDUCES SYSTEMIC CORTICOSTEROID UTILISATION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION: POOLED RESULTS FROM PHASE III RANDOMIZED TRIALS

Funding: GSK (117106/117113/208657). Conflicts of interest: NAH reports advisory board honoraria from Sanofi, Pfizer, Genentech, AstraZeneca, GSK, Amgen, and Verona; lecture honoraria from Sanofi and Regeneron. His institution has received grants from GSK, AstraZeneca, Genentech, and Sanofi. MK reports grants paid to her institution by National Institutes of Health, American Lung Association, AstraZeneca, and Sanofi; personal consulting fees from AstraZeneca, Sanofi, Chiesi, GSK, Regeneron, Kinaset, and Genentech; presentation fees from Chiesi, Sanofi, Regeneron, and GSK. EB, AB, SG, and JM are employed by GSK and hold financial equities in GSK.

P267 SUSTAINED FEV1 IMPROVEMENTS WITH MEPOLIZUMAB UP TO 104 WEEKS IN PATIENTS WITH COPD AND LESS SEVERE AIRFLOW OBSTRUCTION: POST HOC ANALYSIS OF THE MATINEE PHASE III STUDY

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P268 DUPILUMAB IS EFFICACIOUS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE 2 INFLAMMATION REGARDLESS OF SMOKING STATUS

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. Wedzicha JA: AstraZeneca, Boehringer, Chiesi, GSK, Novartis, Genentech, 37Clinical – funds paid to institute; AstraZeneca, Empiricio, Epiendo, GSK, Gilead, Novartis, Pieris, Pulmatrix, Roche, Pfizer Sanofi, – advisory boards or consultant; AstraZeneca, GSK, Boehringer, Novartis, Recipharm, Roche, Sanofi – speaker fees; Viturs – DSMB chair. Brusselle GG: AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Novartis, Sanofi, Teva – consultant, speaker fees. Backman H: Chiesi – consultancy fee. Sugiura H: AstraZeneca, Boehringer Ingelheim, GSK, Sanofi – advisory board. Watz H: AstraZeneca, Bayer, Boehringer Ingelheim, Chiesi, GSK, Novartis, Sanofi, Takeda – consultant, travel, speaker fees. Saha-Chaudhuri P, Heble J: Sanofi – employees, may hold stock and/or stock options in the company. Soliman M: Regeneron Pharmaceuticals Inc. – employee and shareholder. Papi A: AstraZeneca, Chiesi, GSK, Sanofi – payments to institution; AstraZeneca, Avillion, Chiesi, GSK, Moderna, Roche, Sanofi – consultancy fees; AstraZeneca, Avillion, Chiesi, GSK, IQVIA, Moderna, Roche, Sanofi, Zambon – payment or honoraria for lectures, presentations, speakers' bureaus, manuscript writing, or educational events.

P270 TREATMENT FAILURE AT 1 YEAR FOLLOWING BENRALIZUMAB DURING AN EOSINOPHILIC EXACERBATION: DATA FROM ABRA

The ABRA study was funded by AstraZeneca through an unrestricted grant to MB's institution.

P271 CRP MODIFIES RESPONSE TO MEPOLIZUMAB FOLLOWING HOSPITALISATION FOR COPD EXACERBATION: POST-HOC ANALYSIS OF COPD-HELP

GSK supported study (209724)

This abstract was presented at European Respiratory Society Congress 2026 (poster).

P275 PRIOR EXACERBATION HISTORY SHAPES SHORT-TERM PREDICTION OF COPD EXACERBATIONS IN REAL-WORLD MYCOPD DATA

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests. Tom Wilkinson reports a relationship with my mhealth ltd that includes: board membership. Alison Blythin reports a relationship with my mhealth ltd that includes: employment. The other authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

P276 PLATELET FUNCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) PATIENTS, WITH AND WITHOUT CARDIOVASCULAR CO-MORBIDITIES

Funded by the medical research council (MRC).

P277 A MULTIVARIATE SPUTUM PROTEOMIC SIGNATURE ASSOCIATED WITH AIRFLOW LIMITATION IN COPD: BASELINE FINDINGS FROM THE PHASE 2A, RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED COPD-ST2OP TRIAL

CEB: grants from AstraZeneca, GlaxoSmithKline, Roche/Genentech, Boehringer Ingelheim, Novartis, Chiesi, Merck Sharp & Dohme, Sanofi, Regeneron, 4DPharma, and Mologic; and consulting fee paid to institution from AstraZeneca, GlaxoSmithKline, Roche/Genentech, Boehringer Ingelheim, Novartis, Chiesi, Merck Sharp & Dohme, Sanofi, Regeneron, and 4DPharma. NJG: investigator led grant from GlaxoSmithKline; National Institute for Health Research fellowship; and lecture honoraria from GlaxoSmithKline and Chiesi. AW: research grant from Sanofi Genzyme. MAG, DFC: employed by Genentech and receive stock options. All other authors declare no competing interests.

P278 BASELINE SERUM IL1RL1 IDENTIFIES A COPD SUBGROUP WITH INCREASED EXACERBATION BURDEN AND REDUCED EXACERBATION RATES FOLLOWING ASTEGOLIMAB: A POST-HOC ANALYSIS OF THE PHASE 2A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED COPD-ST2OP TRIAL

CEB: grants from AstraZeneca, GlaxoSmithKline, Roche/Genentech, Boehringer Ingelheim, Novartis, Chiesi, Merck Sharp & Dohme, Sanofi, Regeneron, 4DPharma, and Mologic; and consulting fee paid to institution from AstraZeneca, GlaxoSmithKline, Roche/Genentech, Boehringer Ingelheim, Novartis, Chiesi, Merck Sharp & Dohme, Sanofi, Regeneron, and 4DPharma. NJG: investigator led grant from GlaxoSmithKline; National Institute for Health Research fellowship; and lecture honoraria from GlaxoSmithKline and Chiesi. AW: research grant from Sanofi Genzyme. MAG, DFC: employed by Genentech and receive stock options. All other authors declare no competing interests.

P281 ADMISSION IL-33/ST2 PROFILING IDENTIFIES COMPARTMENT-SPECIFIC SEVERITY SIGNALS IN ADULTS HOSPITALISED WITH RESPIRATORY VIRAL INFECTIONS

The UNIVERSAL study was supported by Janssen Pharmaceuticals, GlaxoSmithKline, Sanofi and AstraZeneca, with BioFire FilmArray systems/kits and operational support provided by Synairgen Research Ltd and additional systems/kits provided by bioMérieux SA. The IL-33/ST2 biomarker analyses were supported by AstraZeneca.

P282 BEYOND THE EOSINOPHIL: BENRALIZUMAB DRIVES AIRWAY-SPECIFIC T CELL IMMUNE REMODELLING IN SEVERE EOSINOPHILIC ASTHMA

Investigator led, Astrazeneca funded study.

P283 HOST-DERIVED CLINICAL PHENOTYPES DELINEATE DISTINCT SEVERITY TRAJECTORIES IN ADULT RESPIRATORY VIRAL HOSPITALISATION

The UNIVERSAL study was supported by Janssen Pharmaceuticals, GlaxoSmithKline, Sanofi and AstraZeneca, with BioFire FilmArray systems/kits and operational support provided by Synairgen Research Ltd and additional systems/kits provided by bioMérieux SA.

M6 NUTRITIONAL STATUS AND MALNUTRITION SCREENING IN INTERSTITIAL LUNG DISEASE: A SYSTEMATIC REVIEW.

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M10 COMBINED EBUS-TBNA/TBNB AND EUS-B-FNA/FNB IMPROVES TISSUE DIAGNOSIS OF GRANULOMATOUS LUNG DISEASE PRESENTING WITH ISOLATED MEDIASTINAL LYMPHADENOPATHY: A PROSPECTIVE RANDOMIZED TRIAL

This study was supported by a research fellowship from the Hellenic Thoracic Society (Hellenic Pneumonology Society). The authors declare no conflicts of interest.

M14 **CLINICAL AND HUMANISTIC BURDEN OF PULMONARY HYPERTENSION ASSOCIATED WITH INTERSTITIAL LUNG DISEASE (PH-ILD): SECONDARY ANALYSIS OF THE ADELPHI DISEASE SPECIFIC PROGRAMME (DSP)**

Data collection was undertaken by Adelphi Real World as part of an independent survey, the Adelphi Real World PH-ILD Disease Specific Programme (DSP). The DSP is wholly owned by Adelphi Real World. Insmed Incorporated are one of multiple subscribers to the DSP. This study was funded by Insmed Incorporated (Bridgewater, NJ, USA). All authors contributed to and approved the abstract; writing and editorial assistance were provided by Emanuela Marcantoni, PhD, of The Lockwood Group (Stamford, CT, USA), funded by Insmed Incorporated.

M16 **CORRELATION OF CHEST CT PATTERNS WITH PLEURAL FLUID BIOCHEMISTRY, INFLAMMATORY MARKERS AND MICROBIOLOGY IN ADULT PLEURAL INFECTION: AN EXPLORATORY STUDY.**

This abstract accepted for poster presentation at European Respiratory Society Congress 2026 but we have added more data and figure

M24 **CELL-FREE DNA IN SUSPECTED MALIGNANT PLEURAL EFFUSION: DIAGNOSTIC AND MOLECULAR APPLICATIONS**

This abstract was presented at European Respiratory Society Congress - September 2026

M37 **DOES HYPOXIC BURDEN BETTER EXPLAIN SEX DIFFERENCES IN FUNCTIONAL IMPAIRMENTS THAN APNOEA-HYPOPNOEA INDEX IN OBSTRUCTIVE SLEEP APNOEA?**

ASLC has received research support from Bespoke Medical Innovations Pty Ltd, and honoraria and travel support for educational events from SomnoMed Limited. PAC reports having an appointment to an endowed academic Chair at the University of Sydney that was established from Resmed funding, has received research support from Resmed and SomnoMed, and is a consultant to Resmed, SomnoMed, Eli Lilly, and MyCardio. PdC holds an endowed academic Chair at the University of Sydney that was established from Resmed funding, and is a consultant to SomnoMed.

M40 **OPTIMISING TRANSCUTANEOUS ELECTRICAL STIMULATION STRATEGIES FOR OBSTRUCTIVE SLEEP APNOEA: COMPARING CONTINUOUS AND INTERMITTENT STIMULATION USING A SKELETAL MUSCLE MODEL**

Governmental Fund (Saudi Arabian Cultural Bureau in London, KCL Student ID: 21133372/1, 21st July 2024)

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