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(copd OR "Pulmonary Disease, Chronic Obstructive"[Mesh])

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Sci Rep

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. 2026 Mar 31.

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Immune-inflammatory profiles are associated with exercise capacity and psychological status in hospitalized patients with acute exacerbation of COPD

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Affiliations Expand

- **PMID: 41917220**

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Abstract

To examine the associations between immune-inflammatory status and exercise capacity and psychological status in hospitalized patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD), and to inform multidimensional patient characterization. In this cross-sectional study, consecutively hospitalized patients with AECOPD were enrolled and classified into Grade I, II, and III according to predefined severity criteria. General clinical data

were collected. Peripheral blood T-lymphocyte subsets ($CD3^+$%, $CD4^+$%, $CD8^+$%, and $CD4^+/CD8^+$ ratio) and inflammatory markers (IL-6, IL-8, TNF-α, WBC, and hs-CRP) were measured. Exercise capacity, symptom burden, psychological status, and activities of daily living were assessed using the 6-minute walk test (6MWT), modified Medical Research Council (mMRC) dyspnea scale, Borg scale, COPD Assessment Test (CAT), Hospital Anxiety and Depression Scale (HADS), and Activities of Daily Living (ADL) scale. Group differences across severity grades were compared, and correlation analyses were performed to evaluate relationships between immune-inflammatory markers and clinical outcomes. Across increasing AECOPD severity grades, $CD3^+$% and $CD4^+$% decreased, whereas $CD8^+$% showed a non-significant increasing trend. Inflammatory markers such as IL-8, WBC, and hs-CRP increased with severity grade, whereas IL-6 and TNF-α did not show significant overall differences across the three groups. The 6MWT distance decreased with higher severity grade, and Borg, CAT, HADS-A, and HADS-D scores increased. mMRC and ADL did not show statistically significant overall differences across severity grades. $CD4^+$% and the $CD4^+/CD8^+$ ratio were positively correlated with 6MWT distance and negatively correlated with CAT, while $CD8^+$% was negatively correlated with 6MWT distance and ADL and positively correlated with CAT.Among inflammatory markers, several showed significant correlations with 6MWT distance, Borg, CAT, and selected psychological or ADL outcomes, while others were non-significant. In hospitalized AECOPD patients, immune-inflammatory profiles varied across severity grades and were associated with exercise capacity, symptom burden, psychological status, and daily functioning. Integrating immune-inflammatory markers with functional and psychological assessments may support more comprehensive characterization of patients. Prospective multicenter studies are warranted to evaluate prognostic utility and clarify temporal relationships.

Keywords: AECOPD; Exercise capacity; Immunity; Inflammation; Inflammatory cytokines; Psychological status; T lymphocyte subsets.

Conflict of interest statement

Declarations. Competing interests: The authors declare no competing interests. Ethics approval: This study was conducted in accordance with the Declaration of Helsinki. The study protocol was reviewed and approved by the Ethics Committee of the Second Affiliated Hospital of Zunyi Medical University (Approval No. YXLL(KY)-2025-045). Written informed consent was obtained from all participants prior to enrollment. All data were collected anonymously and analyzed in a de-identified manner to ensure participant confidentiality.

- 33 references

Supplementary info

Grants and fundingExpand

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Ann Am Thorac Soc

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. 2026 Apr 1;23(4):597-606.

doi: 10.1093/annalsats/aaoaf043.

Mortality impact of long-term home non-invasive ventilation in COPD patients with versus without obstructive sleep apnea

Jean-Louis Pepin [1], Eleonore Herquelot [2], Hélène Denis [2], Maxime Patout [3 4], Anne Josseran [5], Florent Lavergne [5], Aurélie Schmidt [2], Atul Malhotra [6], Peter A Cistulli [7 8], Alain Palot [9], Arnaud Prigent [10]; medXcloud group

Collaborators, Affiliations Expand

- PMID: 41915562

- DOI: 10.1093/annalsats/aaoaf043

Abstract

Rationale: Coexisting chronic obstructive pulmonary disease (COPD) and obstructive sleep apnea (OSA) is known as overlap syndrome. This may represent a distinct clinical phenotype that shows different responses after being initiated on noninvasive ventilation (NIV) for hypercapnic chronic respiratory failure. However, current data remain scarce.

Objective(s): This study estimated the impact of overlap syndrome versus COPD without OSA on transitions between 3 states (without/recovery from severe exacerbation, severe exacerbation and death) in patients initiated on domiciliary NIV therapy.

Methods: Multistate model data came from the French national health insurance reimbursement system database for individuals with COPD aged ≥40 years and ≥1 NIV reimbursement in 2015-2019. Outcomes in the overlap syndrome and COPD without OSA groups were compared using a Cox model and inverse probability of treatment weighting analysis, adjusted for patient characteristics.

Results: Data from 54,545 patients were included (median age 70 years, 51.2% male). Probabilities of transitioning from severe exacerbation to death (10% vs. 22%) and without severe exacerbation to death (5% vs. 18%) were lower in the overlap syndrome versus COPD without OSA group. The rate of transition from severe exacerbation to without exacerbation/recovery was also higher in the overlap

syndrome group. After NIV initiation, the mortality rate was 33% lower in people with overlap syndrome vs COPD without OSA.

Conclusions: For people with COPD started on domiciliary NIV, those with overlap syndrome might benefit from NIV to a greater extent than those without OSA. This highlights the need for OSA screening in people with COPD.

Keywords: chronic obstructive pulmonary disease; mortality; noninvasive ventilation; obstructive sleep apnea; overlap syndrome.

3

Ann Am Thorac Soc

. 2026 Apr 1;23(4):637-640.

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The minimal important deterioration of the incremental shuttle walk test in chronic obstructive pulmonary disease: a prospective cohort study

Timothy O Jenkins [1 2], George D Edwards [1], Suhani Patel [1 3], Jane Canavan [1], Samanta Kon [4], Ruth E Barker [5], Sarah Jones [6], Jessica A Walsh [1 7], Karen Ingram [8], Claire M Nolan [1 2], William D-C Man [1 3 8 9]

Affiliations Expand

- PMID: 41915553

- DOI: 10.1093/annalsats/aaoaf060

No abstract available

Supplementary info

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Chronic Obstr Pulm Dis

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. 2026 Mar 30.

doi: 10.15326/jcopdf.2025.0687. Online ahead of print.

Implementation of 2023 Canadian Thoracic Society Guidelines for Single-Inhaler Triple Therapy Could Reduce Exacerbation and Mortality Rates in COPD: PROMETHEUS Canada

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Affiliations Expand

- **PMID: 41911567**

- **DOI: 10.15326/jcopdf.2025.0687**

Free article

Abstract

Background: Chronic Obstructive Pulmonary Disorder (COPD) is the fifth leading cause of death in Canada. The ETHOS (NCT02465567) and IMPACT randomized controlled trials (RCT) (NCT02164513) demonstrated reduced exacerbations and all-cause mortality for patients with COPD on single-inhaler triple therapy (SITT). The 2023 Canadian Thoracic Society (CTS) COPD Pharmacotherapy guidelines recommend triple therapy and preferably SITT use in patients with moderate/severe symptom burden and high future risk of exacerbations. The clinical impact of broader SITT use in Canada has not yet been studied.

Aim: To estimate the benefit of appropriate SITT use according to CTS COPD guidelines on mortality, exacerbations and their corresponding costs in Canada.

Methods: A stochastic model using literature-derived characteristics (e.g. incidence, changes in COPD severity, treatment, mortality, and exacerbations) simulated the Canadian COPD population. Patients were assigned % of FEV_1 predicted levels and their annual characteristics were modeled for 2025-2034 under 2 scenarios: "status quo" (current practice) and "increased SITT" (following CTS guidelines).

Results: Based on our simulated results for the flagged population, "Increased SITT" use over 10 years compared to current treatment reduced moderate and severe exacerbation rates by 23% and 12%, respectively, for a reduction of 159,000 severe and 2.81 million moderate exacerbations and reduced all-cause mortality rate by 22%. In the flagged population alone, this reduction in exacerbations would equate to a savings of CA$3.9 billion over 10 years.

Conclusion: Appropriate use of SITT informed by the 2023 CTS COPD guidelines could lower mortality, exacerbation frequency and their corresponding costs in patients with COPD.

Keywords: COPD; population model; single-inhaler triple therapy.

Supplementary info

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Review

Expert Rev Respir Med

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. 2026 Mar 30:1-15.

doi: 10.1080/17476348.2026.2651412. Online ahead of print.

Daytime sleepiness in patients with obstructive sleep apnea and associated comorbidities

Claudia Di Chiara [1], Giulia Sartori [1], Nadia Castaldo [2], Alberto Fantin [1 2], Marcello Ferrari [1], Ernesto Crisafulli [1]

Affiliations Expand

- PMID: 41883309

- DOI: 10.1080/17476348.2026.2651412

Abstract

Introduction: Obstructive sleep apnea (OSA) is characterized by recurrent upper-airway collapse during sleep, leading to ineffective respiratory efforts, intermittent hypoxia, and sleep fragmentation. Patients with OSA often have comorbid conditions. Excessive daytime sleepiness (EDS), defined as an inability to remain awake during the day, is common among patients with OSA; however, its perception may vary with comorbidities that affect autonomic and neuroendocrine regulation.

Areas covered: We reviewed studies examining the prevalence and clinical impact of EDS in patients with OSA and its main comorbidities, published between January 2000 and September 2025, and identified through Medline.

Expert opinion: EDS is highly prevalent in patients with OSA and arterial hypertension, cardiac arrhythmias, cerebrovascular comorbidities (particularly in those with thalamic or pontine lesions), diabetes mellitus, metabolic syndrome, asthma, chronic kidney disease, and cancer. By contrast, EDS appears less prevalent in patients with heart failure, treated cerebrovascular and neurodegenerative disease (particularly in those receiving levodopa, selective serotonin reuptake inhibitors, or bromocriptine), and chronic obstructive pulmonary disease (COPD).In conclusion, in patients with OSA, EDS is perceived differently depending on comorbidity. Consequently, EDS assessment should follow a personalized, multidimensional approach that recognizes its clinical relevance while accounting for variability across comorbid conditions.

Keywords: Obstructive sleep apnea; cardiovascular disease; chronic obstructive pulmonary disease; comorbidities; diabetes; excessive daytime sleepiness.

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doi: 10.1055/a-2837-8778. Online ahead of print.

Role of Vaccination in the Prevention of ECOPD

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Affiliations Expand

- PMID: 41871621

- DOI: 10.1055/a-2837-8778

Abstract

Exacerbations of chronic obstructive pulmonary disease (ECOPD) represent key events in the natural history of COPD and are associated with several adverse outcomes. Respiratory infections are major and potentially modifiable triggers of ECOPD, with viral pathogens such as the influenza virus, respiratory syncytial virus (RSV), and SARS-CoV-2, as well as bacterial infections caused by Streptococcus pneumoniae, playing a central role. This narrative review examines the current evidence supporting vaccination as a preventive strategy for ECOPD and discusses its translation into clinical practice. The biological rationale for vaccination in COPD is reviewed, including disease-related immune dysregulation, impaired mucociliary clearance, and increased susceptibility to respiratory pathogens. Evidence from randomized clinical trials, observational studies, meta-analyses, and real-world data is summarized for pneumococcal, influenza, SARS-CoV-2, and RSV vaccines. Pneumococcal vaccination has been shown to reduce the burden of community-acquired pneumonia and invasive pneumococcal disease, with conjugate and higher-valent vaccines providing enhanced immunogenicity in older and high-risk adults. Influenza vaccination consistently reduces severe exacerbations, hospitalizations, and mortality, with additional cardioprotective effects of relevance in COPD. SARS-CoV-2 vaccination markedly lowers the risk of severe COVID-19 and related respiratory deterioration in COPD, while recently licensed RSV vaccines offer a novel opportunity to prevent RSV-associated lower respiratory tract disease and potentially reduce exacerbation risk. Patient populations most likely to benefit from vaccination include frequent exacerbators, older adults, individuals with severe airflow limitation, multimorbidity, immune dysfunction, infection-prone phenotypes, and socially vulnerable groups. Future perspectives include precision vaccination strategies, novel vaccine platforms, coadministration approaches, and interventions to improve vaccine uptake. Vaccination emerges as a cornerstone of ECOPD prevention, with substantial potential to reduce exacerbation burden and improve long-term outcomes in COPD.

Conflict of interest statement

The authors declare that they have no conflict of interest.

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Editorial

Open Respir Arch

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. 2026 Feb 18;8(2):100599.

doi: 10.1016/j.opresp.2026.100599. eCollection 2026 Apr-Jun.

Triple Therapy in Routine Clinical Practice: Beyond Clinical Trials in COPD

Juan Marco Figueira-Gonçalves [1], Bernardino Alcázar-Navarrete [2 3 4], Javier de Miguel-Díez [5]

Affiliations Expand

- **PMID: 41858384**

- **PMCID: PMC12995886**

- **DOI: 10.1016/j.opresp.2026.100599**

No abstract available

- **21 references**

Supplementary info

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Semin Respir Crit Care Med

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doi: 10.1055/a-2826-5752. Online ahead of print.

Acute Exacerbation of Chronic Obstructive Pulmonary Disease: Pharmacological Treatment of AECOPD New Perspectives

Jonathan Calvello [1], Michael Avaricio [1], Paolo Ruggeri [2], Antonio M Esquinas [3], Bushra Mina [4]

Affiliations Expand

- PMID: 41844237

- DOI: 10.1055/a-2826-5752

Abstract

Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) are major drivers of morbidity, mortality, disease progression, and healthcare utilization worldwide. Evolving definitions of COPD and exacerbations, along with emerging evidence on risk stratification and treatment optimization, have prompted updates in clinical practice, most recently reflected in the 2026 Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines. This review summarizes contemporary perspectives on AECOPD, with a focus on updated definitions, epidemiology, predictors, clinical impact, and current pharmacological and nonpharmacological management strategies, including emerging preventive therapies. A narrative review of published literature, international guidelines, and major clinical trials was conducted, emphasizing evidence relevant to the assessment, treatment, and prevention of AECOPD. Particular attention was given to severity classification and guideline-directed therapeutic approaches. AECOPD is associated with substantial short- and long-term mortality, accelerated lung function decline, increased cardiovascular risk, and high readmission rates. The 2026 GOLD guidelines lower the threshold for high-risk classification, recognizing that even a single moderate exacerbation increases future risk. Acute management remains centered on short-acting bronchodilators, short courses of systemic corticosteroids, and antibiotics when indicated, with treatment of intensity guided by clinical severity and physiological derangements. Adjunctive supportive measures and early postdischarge interventions are critical to improving outcomes. While biologics, macrolides, Roflumilast, and Ensifentrine have no established role

in the acute setting, they play an important role in exacerbation prevention as part of individualized, biomarker-informed maintenance strategies. AECOPD should be viewed as a sentinel event that necessitates both effective acute management and reassessment of long-term therapy. Early intervention, severity-based treatment, and postexacerbation optimization of maintenance therapy are essential to reduce recurrence, limit disease progression, and improve survival and quality of life in patients with COPD.

Conflict of interest statement

The authors declare that they have no conflict of interest.

Cite

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Review

Respir Med

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. 2026 Apr-May:255:108778.

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A review of artificial intelligence-based research on chronic obstructive pulmonary disease

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Affiliations Expand

- PMID: 41839412

- DOI: 10.1016/j.rmed.2026.108778

Abstract

In recent years, with the rapid development of artificial intelligence (AI), Chronic Obstructive Pulmonary Disease (COPD), one of the world's three major chronic diseases, has achieved remarkable progress in diagnosis, grading, and prognosis, which is of great significance for promoting the clinical transformation of respiratory diseases. To deeply explore the application of AI in the diagnosis and management of COPD, this paper reviews recent studies based on machine learning and deep learning, covering screening and diagnosis, disease grading and assessment, disease management and monitoring, and treatment. First, the technical basis of COPD-related research is analyzed from five perspectives: traditional research methods, supervised learning, unsupervised learning, semi-supervised learning, and reinforcement learning. Then, the commonly used datasets and model evaluation metrics are summarized. Finally, the application scenarios of AI in COPD research are elaborated, focusing on three aspects: early screening and diagnosis, disease monitoring and risk prediction, and disease classification and risk stratification. This paper summarizes the main research hotspots at home and abroad over the past five years with representative models, and analyzes and compares the advantages and limitations of each type of model in specific COPD tasks through comparative experiments. The study also outlines prospects for the future development of this field, aiming to provide theoretical references and insights for subsequent research.

Keywords: Artificial intelligence; Breath sounds; Chronic obstructive pulmonary disease; Clinical decision support; Computer-aided diagnosis; Machine learning.

Conflict of interest statement

Declaration of competing interest The 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.

Supplementary info

Publication types, MeSH termsExpand

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Review

Respir Med

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. 2026 Apr:254:108761.

doi: 10.1016/j.rmed.2026.108761. Epub 2026 Mar 7.

COPD and kidney disease: not so far apart?

Iuliana Stratan [1], Serghei Covantsev [2], Alexander G Mathioudakis [3], Alexandru Corlateanu [4]

Affiliations Expand

- PMID: 41796819

- DOI: 10.1016/j.rmed.2026.108761

Free article

Abstract

This review synthesizes current literature on the epidemiology, mechanistic links, and clinical consequences of the COPD-CKD association. In addition to evaluating established risk factors such as age and tobacco smoking, we discuss emerging insights into the roles of novel biomarkers and epigenetic modifications in bridging the lung-kidney axis. The review further examines how the presence of CKD influences clinical management in COPD patients, including impacts on drug dosing, electrolyte balance, acid-base homeostasis, and outcomes during acute exacerbations, and conversely, how COPD can adversely affect renal function. Recognizing that the dual burden of COPD and CKD may worsen patient prognosis by increasing the risk for cardiovascular events, hospitalizations, and mortality, we advocate for early detection, routine screening using both creatinine and cystatin C measurements, and a multidisciplinary approach to care. Finally, we outline future research directions aimed at standardizing screening protocols, refining risk stratification, and developing targeted therapies that address shared pathogenic pathways.

Keywords: CKD; COPD; Chronic kidney disease; Chronic obstructive pulmonary disease; Hemodialysis; Renal transplant.

Conflict of interest statement

Declaration of competing interest The authors declare no conflicts of interest directly related to this work. SI, CS, AC report no conflicts of interest. AGM reports honoraria from GlaxoSmithKline and Sanofi, share options from Healthy Networks, and non-financial support from Verona Pharma, not related to this work.

Supplementary info

Publication types, MeSH terms, SubstancesExpand

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Acta Physiol (Oxf)

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Physical Inactivity Drives COPD Progression Beyond Airflow Limitation

Salvatore Fuschillo [1], Claudio Candia [1], Pasquale Ambrosino [2], Carmen Lombardi [1], Claudia Merola [1], Giuseppe Rengo [3 4], Mauro Maniscalco [1 5]

Affiliations Expand

- PMID: 41795793

- DOI: 10.1111/apha.70192

No abstract available

- 10 references

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12

J Cachexia Sarcopenia Muscle

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. 2026 Apr;17(2):e70244.

doi: 10.1002/jcsm.70244.

Sex Differences in Muscle-Respiratory Function Relationship in Lung Transplant Patients: A Longitudinal Study

Chiara Ceolin [1 2 3], Agnese Alessi [2], Anna Citron [2], Monica Loy [4], Mario Virgilio Papa [2], Carlotta Andaloro [2], Bruno Micael Zanforlini [2], Maria Devita [2 5], Sara Bertolino [2], Sara Gonnelli [2], Daniele Michele Seccia [2], Anna Bertocco [2], Federico Rea [4], Giuseppe Sergi [1 2], Marina De Rui [2]

Affiliations Expand

- PMID: 41782505
- PMCID: PMC12961349
- DOI: 10.1002/jcsm.70244

Abstract

Background: Lung transplant recipients are at increased risk of sarcopenia and osteoporosis, which may negatively influence respiratory outcomes. Although muscle health is known to affect lung function, little is known about the long-term interplay between muscle parameters and pulmonary volumes, especially across sexes. The objective of this study is to evaluate the longitudinal relationship between muscle mass and strength and respiratory function in lung transplant patients, with sex-specific analysis.

Methods: This prospective cohort included three assessments (baseline ≥ 3 months after transplant, ~1 year and 2-3 years). The primary outcome was the longitudinal change in pulmonary function (VC, FVC, FEV1 and TLC) in relation to appendicular skeletal muscle mass index (ASMMI) and handgrip strength (HGS). Associations at baseline were tested with multivariable linear regression. Analyses were performed with linear mixed-effects models (LMM) including random intercepts for subject, time as a fixed effect and interactions between time and muscle parameters, adjusted for age, ADL, corticosteroid dose, vertebral fractures, osteoporosis, comorbidities and time since transplant.

Results: We studied 155 recipients (43.2% women, age 48.7 ± 13.3 years). Primary indications were cystic fibrosis (30.1%), restrictive (22.2%), obstructive (15.7%), miscellaneous (26.8%) and vascular diseases (5.2%). At baseline, HGS was independently associated with higher VC (R^2: 0.63, β = 0.35, p = 0.001 in women; R^2: 0.58, β = 0.16, p < 0.001 in men) and FEV1 (R^2: 0.51, β = 0.08, p = 0.020 in women; R^2: 0.57, β = 0.19, p = 0.009 in men). ASMMI was independently associated with VC in both sexes (women: R^2: 0.58, β = 0.31, p = 0.003; men: R^2: 0.40, β = 0.16, p = 0.023). Longitudinally, LMMs showed that higher HGS was associated with more favourable trajectories of pulmonary function over follow-up. Specifically, among women with restrictive disease, lower ASMMI predicted higher FEV1 (β = -4.95, 95% CI -6.93 to -2.97, p = 0.007) and higher TLC (β = -2.22, 95% CI -4.56 to -1.12, p = 0.04) over time. In women with cystic fibrosis, stronger HGS was associated with improved TLC (β = 0.38, p = 0.04). All associations persisted after full adjustment.

Conclusion: Muscle mass and strength are associated with lung function after lung transplantation. These findings underscore the clinical importance of muscle health and support its integration into post-transplant management.

Keywords: COPD; cystic fibrosis; lung transplant; sarcopenia; vertebral fractures.

Conflict of interest statement

The authors declare no conflicts of interest.

- **40 references**
- **1 figure**

Supplementary info

MeSH termsExpand

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13

Review

Respir Med

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. 2026 Apr:254:108746.

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A decade of treatable traits in patients with COPD: lessons learned about its application

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Affiliations Expand

- **PMID: 41780763**

- DOI: 10.1016/j.rmed.2026.108746

Free article

Abstract

Chronic obstructive pulmonary disease (COPD) is a common chronic respiratory condition with potentially major implications for the lives of those it affects. In the past decade, the identification of clinically relevant patient characteristics - 'treatable traits' (TTs) - has received wide support as an important means of improving care. These traits can be categorised into three domains: pulmonary, extra-pulmonary and behavioural. Many patients with COPD have multiple TTs in various combinations. For this reason, the successful application of a TT approach requires a thorough assessment of the patient's health status to elicit relevant TTs. Combined with the patient's preferences and priorities, these traits enable the formulation of an individualised treatment plan. Execution of this plan often requires interprofessional collaboration among relevant healthcare providers and case management. Although international consensus appears to exist concerning the benefits of providing care according to the TT concept, widespread implementation in care is slow. Although pulmonary rehabilitation programmes have adopted the concept by definition, it has yet to be applied widely in practice, especially in primary and secondary care. We have used the TT model to develop a comprehensive model of care that contains all three elements (assessment, treatment plan and execution) and that provides insight into considerations and decisions regarding where such care should be provided. This paper describes the process, results and challenges of our attempts to design and implement this integrated care concept.

Conflict of interest statement

Declaration of competing interest The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Martijn Spruit reports a relationship with CIRO that includes: consulting or advisory and funding grants. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary info

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14

Observational Study

Respir Med

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doi: 10.1016/j.rmed.2026.108743. Epub 2026 Feb 28.

BDP/FF/GB single-inhaler triple therapy in COPD: real-world effectiveness, safety, and adherence in a pooled analysis of 5,000 patients

Guy Brusselle [1], Petros Bakakos [2], Daiana Stolz [3], Alessio Piraino [4], Laura Franzini [4], Elena Nudo [4], Luca Di Palma [4], Paola Rogliani [5]

Affiliations Expand

- **PMID: 41771375**

- **DOI: 10.1016/j.rmed.2026.108743**

Free article

Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) represents a major global health challenge, frequently necessitating triple therapy for patients unresponsive to dual bronchodilation. Single-inhaler triple therapy (SITT) combining beclomethasone dipropionate/formoterol fumarate/glycopyrronium bromide (BDP/FF/GB) offers a practical and effective solution with potential adherence benefits in real-world settings.

Methods: This pooled analysis included patient-level data from six European prospective observational studies assessing the real-world effectiveness and safety of BDP/FF/GB SITT in COPD patients over 6 months. Outcomes included changes in health-related quality of life (HRQoL; measured by COPD Assessment Test [CAT] scores), lung function (FEV_1), and adherence, with safety and exacerbation rates assessed through 12 months. Subgroup comparisons based on prior therapy, exacerbation history, and disease severity were also performed.

Results: Among 5523 patients enrolled, 4541 were included in the full analysis set. Significant improvements in HRQoL were observed, with mean CAT score reductions of -3.8 and -4.7 at months 3 and 6, respectively ($p < 0.0001$). Patients with at least one exacerbation decreased from 93.1% (pre-treatment) to 35.7% at 12 months ($p < 0.0001$). FEV_1 improvements were observed up to 6 months, with over 40% of patients achieving ≥100 mL increase. Adherence significantly improved, with poor-adherence patients transitioning to higher categories over time. Logistic regression identified poor baseline adherence (vs. good), ex-smokers (vs non-

smokers), and experiencing two (vs. >2) exacerbations as predictors of treatment response. Adverse events were reported by 21.9% of patients.

Conclusion: Extrafine BDP/FF/GB SITT demonstrated significant real-world safety and effectiveness in improving symptoms, lung function, and adherence.

Keywords: Adherence; CAT score; COPD; Exacerbation; Extrafine BDP/FF/GB; Single-inhaler triple therapy.

Conflict of interest statement

Declaration of competing interest The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:Paola Rogliani reports financial support was provided by Chiesi Pharmaceuticals SpA. Guy Brusselle, Petros Bakakos, Daiana Stolz reports financial support was provided by Chiesi Pharmaceuticals SpA. Paola Rogliani reports a relationship with Arcede Pharma, AstraZeneca, Boehringer Ingelheim, Chiesi Farmaceutici, Sanofi, Verona Pharma and Zambon that includes: funding grants. Paola Rogliani reports a relationship with AstraZeneca, Boehringer Ingelheim, Chiesi Farmaceutici, GlaxoSmithKline, Menarini Group, Novartis, Pfizer, Recipharm, Regeneron, Roche and Sanofi that includes: consulting or advisory. Daiana Stolz reports a relationship with AstraZeneca, Berline-Chemie-Menarini, Boehringer Ingelheim, Chiesi, CSL Behring, GSK, Merck, MSD, Novartis, Sanofi, Vifor and Roche that includes: speaking and lecture fees. Daiana Stolz reports a relationship with AstraZeneca, Berlin-Chemie- Menarini, Boehringer Ingelheim, Chiesi, CSL Behring, GSK, Merck, MSD, Roche, Novartis, Sanofi and Vifor that includes: consulting or advisory. Guy Brusselle reports a relationship with AstraZeneca, Boehringer-Ingelheim, Chiesi, Glaxo Smith Kline, Merck Sharp & Dohme, Novartis, Sanofi and Regeneron that includes: consulting or advisory and speaking and lecture fees. Petros Bakakos reports a relationship with Menarini, GSK, AstraZeneca, Guidotti, and Chiesi that includes: consulting or advisory and speaking and lecture fees. Alessio Piraino, Laura Franzini, Elena Nudo, Luca Di Palma reports a relationship with Chiesi Pharmaceuticals SpA that includes: employment. DS is the GOLD representative for Switzerland; PB is unpaid member of the Hellenic Thoracic Society If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Open Respir Arch

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Impact-Driven Strategies for Optimizing Inhaled Therapy Adherence in COPD: The OPTIMO Delphi Consensus

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Affiliations Expand

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Abstract

in English, Spanish

Introduction: Medication adherence is an important challenge in the management of COPD, with poor adherence negatively impacting symptom control, disease progression, and healthcare resource utilization.

Objetives: This study aimed to achieve multidisciplinary consensus on practical, prioritized interventions for improving adherence and clinical results in stable COPD treatment.

Material and methods: A modified Delphi study was carried out by a panel of COPD management experts to evaluate suboptimal adherence. The Delphi questionnaire, completed in two rounds, comprised 84 statements across six domains: non-adherence, its impact, contributing factors, assessment methods, improvement strategies, and prioritization. Agreement was rated using a 9-point Likert scale and consensus considered as ≥70% agreement.

Results: Seventy-three multidisciplinary experts completed the questionnaire, reaching consensus on 74 of 84 statements. The panel identified high-impact, easy-to-implement interventions, culminating in a practical decalogue for optimizing inhalation therapy. Two key strategies were emphasized: simplifying and unifying inhalation regimens, and empowering patients. While all adherence assessment

tools were considered useful, only digital and automated methods were deemed feasible in routine practice. Treatment optimization was also associated with reducing inhalation frequency by using long-acting molecules, and tailoring device selection to patient capacity.

Conclusion: The resulting decalogue provides structured expert guidance for healthcare professionals, highlighting once-daily dosing, simplified devices, and patient engagement as the interventions considered most likely to improve adherence in COPD.

Keywords: Adherence assessment tools; COPD adherence; Delphi method; Inhaled therapy optimization; Once-daily dosing.

- 50 references
- 2 figures

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Clin Drug Investig

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Missed Opportunities in the Therapeutic Patterns of Chronic Obstructive Pulmonary Disease: A Real-World Italian Study on the Impact of Severe Exacerbation on Mortality and Hospital Re-admission

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Abstract

Background and objectives: Patients experiencing severe exacerbation (SE) of chronic obstructive pulmonary disease (COPD) face high re-hospitalization and mortality rates. Knowing their impact on health services is crucial for making decisions and improving the care pathway for these patients. To describe mortality, re-hospitalizations and therapeutic patterns of patients experiencing SE, this study used real-world Italian administrative data and examined pre- and post-SE treatment patterns.

Methods: From an Italian administrative database (4.6 million inhabitants), patients with COPD in 2022 were identified, and those experiencing SE requiring hospitalization were described in terms of demographics, comorbidities, 12-month mortality and re-hospitalization rates, and therapeutic patterns during 12 months pre- and post-SE. Treatment patterns included dispensations of single-inhaler (SI, i.e., fixed-dose combination) triple therapy (TT), multiple-inhaler (MI, i.e., open combination) TT, dual therapy (DT; based on combinations between inhaled corticosteroid/long-acting beta-agonist/muscarinic antagonist), other respiratory treatment strategies, and no treatment.

Results: Among 81,571 patients with COPD (32.1 per 1000 inhabitants aged ≥ 45 years), patients experiencing SE were 6.2% (5080 patients with COPD): mean age was 77 years, 63.4% (3220 patients with SE) were male and 68.2% (3467 patients with SE) had ≥ 3 comorbidities. Mean in-hospital length of stay was 11.9 days. One-year mortality rate was 25.6% (1302 patients with SE), mainly within the first month, 76.0% (989 deaths) of which occurred in hospital. Among 12-month analysable patients (3778), the 12-month re-hospitalization rate was 20.1% (761 patients with SE and alive) occurring, on average, at 155 days (16.4% within the first month). Pre/post SE, single-inhaler triple therapy (SI-TT) and multiple-inhaler triple therapy (MI-TT) increased from 1.4 to 23.4% and from 1.2 to 6.3% patients, respectively; DT, other strategies, and no treatment reduced from 24.5 to 22.5%, 35.7 to 23.8% and 37.1 to 24.0%, respectively. On average, TT was initiated at ≥ 31 days post-SE. Mean time to TT initiation was approximately 31-42 days depending on prior therapy, although most patients initiated TT within the first 30 days.

Conclusion: A substantial proportion of patients experiencing SE of COPD were re-hospitalised or remained inadequately treated or untreated, despite a high mortality rate. These findings underscore the necessity for a more appropriate and prompt therapeutic intervention.

Conflict of interest statement

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Conflict of interest: SC, GR, LD, LeonardoD, ID, CP and NM are employees of Fondazione ReS and have no competing interests with any financial organisations regarding the material discussed in the manuscript. OB has no competing interests with any financial organisations regarding the material discussed in the manuscript. FDM reports grants and personal fees from AstraZeneca, Boehringer Ingelheim,

GSK, Chiesi, Zambon, Novartis, Guidotti/Malesci, Menarini, Mundipharma, Sanofi, TEVA, Levante Pharma, Neopharmed Gentili, outside the submitted work. CM reports fees as a speaker in national and international congress from AstraZeneca, GSK, Sanofi, Novartis, Chiesi, Menarini, Guidotti, Berlin Chemie, Boehringer, Zambon, outside the submitted work. Ethics approval: No ethics approval was sought because it was not required for this retrospective analysis of anonymised administrative data according to the European Regulation 2014/536, and it is not expected by the most recent national legislation on observational studies. Consent to participate: Not applicable because the informed consent was waived according to the specific Italian Privacy Authority's provision. Consent for publication: Not applicable. Code availability: Not applicable. Availability of data and material: The datasets analysed during the current study are not publicly available and are not available from the corresponding author on reasonable request, because they are owned by the Italian Regional/Local Health Authorities who have not authorised Fondazione ReS to make them available. Author contributions: Conceptualisation, LD, GR, LeonardoD, SC; data curation, LD, GR; formal analysis, LD, LeonardoD; funding acquisition, CP, NM; investigation, LD, GR, LeonardoD, SC, ID, CP; methodology, LD, GR; project administration, SC, LD; software, LD, LeonardoD; supervision, CP, NM; validation, FDM, CM, OB; writing – original draft, SC, ID, FDM, OB, CM; writing – review and editing, SC, ID, CP, LD, GR, NM, FDM, OB, CM. All authors have read and approved the final version and agreed to be accountable for the work.

- 47 references

Supplementary info

MeSH terms, SubstancesExpand

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Randomized Controlled Trial

Physiother Res Int

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Acute Effect of Inspiratory Muscle Training on Peripheral Tissue Oxygenation Behavior in Individuals With COPD: A Randomized Crossover Study

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- **DOI: 10.1002/pri.70177**

Abstract

Background and purpose: Chronic Obstructive Pulmonary Disease (COPD) is a pulmonary condition characterized by airflow obstruction, which progresses with systemic alterations such as changes in muscle composition and metabolism, anticipating the activation of the inspiratory metaboreflex. This study aimed to analyze the acute effects of Inspiratory Muscle Training (IMT) on peripheral muscle metabolism in individuals with COPD, using near-infrared spectroscopy (NIRS).

Methods: This randomized, blinded, crossover study included 29 individuals with COPD who underwent three distinct sessions: high-load IMT (IMT-Strength, 60% of maximal inspiratory pressure-MIP), low-load IMT (IMT-Endurance, 30% of MIP), and a sham protocol. Tissue oxygenation of the gastrocnemius muscle was assessed using NIRS before and after each protocol.

Results: Differences in mean final tissue oxygen saturation were observed only during the IMT-Endurance protocol. The oxygen desaturation time was shorter during the IMT-Strength protocol compared with the other groups. Although not statistically significant, patients with more severe COPD (GOLD 3-4) exhibited an oxygen desaturation rate higher during the strength IMT compared with the endurance and sham protocols.

Conclusions: Acute high-intensity IMT may accentuate the reduction in peripheral perfusion, especially in patients with advanced COPD, suggesting possible metaboreflex activation. Conversely, endurance IMT may improve peripheral perfusion. These findings reinforce the need for careful and individualized prescription of IMT in the COPD population.

Trial registration: Clinical Trials number: NCT06827379 https://clinicaltrials.gov/study/NCT06827379.

Keywords: COPD; breathing exercises; muscle fatigue; near-infrared; respiratory muscles; spectroscopy.

Conflict of interest statement

The authors declare no conflicts of interest.

- **23 references**
- **3 figures**

Supplementary info

Publication types, MeSH terms, Substances, Associated data, Grants and fundingExpand

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Semin Respir Crit Care Med

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Epidemiology of COPD Exacerbations

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- **DOI: 10.1055/a-2818-1414**

Abstract

Chronic obstructive pulmonary disease (COPD) exacerbations represent the most common acute event and the one with the greatest medium- to long-term clinical and prognostic impact, acting as a key driver of functional decline, deterioration in

quality of life, and constituting a substantial share of the morbidity, mortality, and healthcare costs attributable to the disease. This review synthesizes the most relevant epidemiological evidence on the frequency, distribution, and environmental determinants of exacerbations, with particular emphasis on longitudinal trends, seasonal patterns, and economic burden. Overall, the last decades have seen a decline in exacerbation rates within the context of clinical trials; however, analyses based on hospital registries are constrained by methodological limitations, notably reliance on International Classification of Diseases-coded case identification and a predominant focus on severe events. Temporal trajectories also vary by region: in Spain, decrease followed by subsequent rebounds have been described, with a more pronounced increase among women, while other European and non-European settings report divergent patterns. Seasonality emerges as a robust feature in temperate climates, with winter peaks and a consistent association between low temperatures (and thermal variability) and higher admission rates and exacerbation severity. Ambient air pollution ($PM_{2.5}$/PM_{10}, NO_2, SO_2, O_3) is linked to an increased risk of exacerbation, potentially with lagged effects, through biologically plausible pathways mediated by oxidative stress and inflammation. Finally, we discuss the impact of exposures arising from environmental disasters (wildfires, volcanic eruptions, and oil spills), illustrated by recent events in Spain, and integrate the economic dimension, underscoring that exacerbations account for a large proportion of the total cost of COPD.

Conflict of interest statement

J.L.L.C. has received honoraria during the last 3 years for lecturing, scientific advice, participation in clinical studies, or writing for publications for (in alphabetical order): AstraZeneca, Bial, Boehringer, Chiesi, CSL Behring, Faes, Gebro, Grifols, GSK, Menarini, Sanofi, Zambon. The rest of the authors declare no conflicts of interest.

Cite

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Review

Respir Med

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Clinical protocol for the use of biologic therapy in COPD

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Free article

Abstract

Preventing exacerbations in chronic obstructive pulmonary disease (COPD) is a priority, as exacerbations accelerate progression, increase mortality, and impair quality of life. The use of biologics that act on specific inflammatory pathways through monoclonal antibodies in COPD offers a precision medicine strategy for high-risk patients who continue to experience frequent exacerbations despite optimal inhaled treatment. However, the use of these biologic therapies in COPD presents significant challenges, stemming from the clinical heterogeneity of the disease and the limited evidence available. Their indication and management must be personalized and precise, raising questions about how to implement a biologic therapy strategy in COPD in clinical practice. This clinical protocol focused on the use of biologic therapy in COPD sets out the objectives ("Why"), describes the selection of candidates ("In which patients"), the monitoring of response ("What should be measured") and how to implement it in clinical practice ("How") based on the available evidence and expert consensus.

Keywords: Biologics; COPD; Candidates; Clinical practice; Management; Monitoring of response.

Conflict of interest statement

Declaration of competing interest The 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.

Supplementary info

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Review

Drugs

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Mucus as a Treatable Trait in Chronic Airway Diseases

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Affiliations Expand

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- PMCID: PMC13005781
- DOI: 10.1007/s40265-026-02294-1

Abstract

Chronic airway diseases, including asthma, chronic obstructive pulmonary disease, bronchiectasis, and cystic fibrosis, are increasingly recognized as heterogeneous disorders characterized by overlapping pathophysiological mechanisms. Among these, abnormalities in mucus production, composition, and clearance have been identified as clinically significant contributors to symptoms, airflow limitation, exacerbations, and disease progression. Within the "treatable traits" framework, mucus-related abnormalities represent a distinct, modifiable phenotype that supports personalized management strategies. This narrative review explores mucus as a treatable trait across chronic airways diseases, integrating mechanistic insights with clinical assessment, biomarkers, and current and emerging therapeutic approaches. We discuss the role of mucus in disease phenotyping, its impact on morbidity, and the potential of targeted interventions to improve outcomes. Recognizing mucus as a treatable trait aligns with the principles of

precision medicine and offers a pathway toward individualized therapy beyond traditional diagnostic labels.

Conflict of interest statement

Declarations. Conflicts of interest: The Authors have no conflicts of interest that are directly relevant to the content of this article. Mario Cazzola and Luigino Calzetta are Editorial Board members of Drugs. Mario Cazzola and Luigino Calzetta were not involved in the selection of peer reviewers for the manuscript, nor in any of the subsequent editorial decisions. Ethics approval: Not applicable. Consent to participate: Not applicable. Consent for publication: Not applicable. Availability of data and material: Not applicable. Code availability: Not applicable. Author contributions: All authors contributed to the manuscript. Original draft preparation: Mario Cazzola. Writing, review, and editing: Paola Rogliani, Josuel Ora, Luigino Calzetta, and Maria Gabriella Matera. All authors read and approved the final manuscript.

- **129 references**
- **3 figures**

Supplementary info

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Lancet Reg Health Eur

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors) showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma, and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA

and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this work. S Principe reports grants from Innovative Medicines Initiative 2 Joint Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards

(payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-chair of the Nordic severe asthma network; member of the steering committee of SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand,

Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

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Am Heart J

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Large-bore mechanical thrombectomy vs standard of care for acute high-risk pulmonary embolism: Rationale and design of the PERSEVERE randomized controlled trial

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Free article

Abstract

Background: Catheter-directed therapies are increasingly used to treat acute pulmonary embolism (PE). However, randomized data on reperfusion treatments, including large-bore mechanical thrombectomy (LBMT), for patients with High-Risk PE are lacking.

Methods: PERSEVERE (NCT06588634) is a multinational randomized controlled trial comparing the FlowTriever LBMT system vs. standard of care (SoC) in patients with High-Risk PE, with the modified intention-to-treat population planned for 200 patients from 40 sites in Europe and the US. Patients are randomized 1:1 to LBMT or SoC (systemic thrombolysis [ST], surgical embolectomy, extracorporeal membrane oxygenation [ECMO], or anticoagulation alone). Key inclusion criteria are the presence of proximal pulmonary thrombus on computed tomography plus ≥1 of the following: (1) systolic hypotension or need for vasopressors, (2) venous lactate ≥4 mmol/L with clinical signs suggesting obstructive shock, (3) need for mechanical circulatory support, (4) resuscitated cardiac arrest. Exclusion criteria include known chronic thromboembolic pulmonary hypertension and key absolute contraindications to ST. Patients are followed for 3 months. The primary endpoint is a composite of events through hospital discharge or 7 days post randomization, whichever occurs first: (1) all-cause death, (2) cardiac arrest requiring cardiopulmonary resuscitation, (3) bailout to rescue treatment, (4) major bleeding, and (5) ECMO in place on day 7. Secondary endpoints include a broad spectrum of functional and patient-reported outcomes (quality of life, functional status and healthcare resource utilization) at 3 months. The trial is funded by Inari.

Conclusion: The PERSEVERE study will assess the potential superiority of LBMT over SoC for the treatment of High-Risk PE.

Clinicaltrials: gov Identifier: NCT06588634.

Conflict of interest statement

Conflict of interest S. Barco reports honoraria of low amount from Penumbra, Inari, Concept Medical, and Boston Scientific, and institutional grants from Penumbra and Boston Scientific; Sripal Bangalore reports lecture/consultant fees from Inari, Imperative Care, Argon Medical and Jupiter; Romain Chopard reports lecture/consultant fees from MSD, BMS/Pfizer, Bayer Healthcare, Braun Medical, Edwards Life Science, and Inari Medical, outside the submitted work; Jay Giri reports serving as an advisor to and receiving research funds to the institution from

Inari Medical, Boston Scientific, and Endovascular Engineering; Lukas Hobohm reports lecture/consultant fees from Boston Scientific, Inari Medical, MSD and Johnson&Johnson, outside the submitted work; Stavros Konstantinides reports research grants or contracts to his institution from Bayer AG, Boston Scientific, Daiichi Sankyo, Inari Medical, and Penumbra and personal consulting fees from Boston Scientific, Daiichi Sankyo, Inari Medical, and Penumbra; Felix Mahfoud has been supported by Deutsche Forschungsgemeinschaft (SFB TRR219, Project-ID 322900939), and Deutsche Herzstiftung. Saarland University has received scientific support from Ablative Solutions, Medtronic and ReCor Medical. Until May 2024, FM has received speaker honoraria/consulting fees from Ablative Solutions, Astra-Zeneca, Inari, Medtronic, Merck, Novartis, Philips and ReCor Medical; John Moriarty reports consultant fees from Inari Medical, AngioDynamics, Penumbra, Argon Medical, Pavmed, Auxetics, Innova Vascular, Retriever Medical, Truvic, Inquis Medical, and Imperative Care and is president of the PERT Consortium; Nicolas Meneveau reports consultancy agreements with Inari, Abbott, Boston Scientific, Edwards Lifesciences, and Terumo as well as speaker fees from Astra Zeneca, BMS-Pfizer, and Servier; Stephan Rosenkranz reports remunerations for lectures and/or consultancy from Abbott, Acceleron, Actelion, Aerovate, AOP, AstraZeneca, Bayer, BMS, Boehringer-Ingelheim, Edwards, Ferrer, Gossamer, Inari, Janssen, Lilly, Liquidia, MSD, OMT, Pfizer, and United Therapeutics and grants to institution from Actelion, AstraZeneca, Bayer, MSD, and Janssen; Andrew Sharp reports Consultancy/Speaker's Fees from Philips, Medtronic, Boston Scientific, Penumbra, AngioDynamics and Recor Medical; Holger Thiele reports having no conflicts of interest; Catalin Toma reports a consultancy agreement with Inari and SAB membership for Jupiter Endovascular.

Supplementary info

Publication types, MeSH terms, Substances, Associated dataExpand

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Comparative Study

Int J Clin Pharm

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doi: 10.1007/s11096-025-02037-4. Epub 2025 Nov 7.

Comparative effectiveness and safety of fluticasone-based versus beclometasone-based single-inhaler triple therapies in patients with chronic obstructive pulmonary disease: a population-based cohort study

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- PMCID: PMC12992448
- DOI: 10.1007/s11096-025-02037-4

Abstract

Introduction: There is a paucity of comparative real-world evidence for fluticasone-based and beclometasone-based single-inhaler triple therapies in patients with chronic obstructive pulmonary disease (COPD).

Aim: To compare clinical outcomes of fluticasone/umeclidinium/vilanterol (a once-daily dry powder inhaler) and beclometasone/glycopyrrolate/formoterol (a twice-daily metered dose inhaler) in patients with COPD.

Method: This population-based cohort study enrolled patients with COPD who initiated fluticasone/umeclidinium/vilanterol or beclometasone/glycopyrrolate/formoterol from a nationwide Taiwanese database between 2019 and 2022. The effectiveness outcomes included severe and moderate exacerbations and the safety outcomes were pneumonia and composite cardiovascular events. Patients were followed from the first day after cohort entry to the earliest of each outcome occurrence, study treatment discontinuation or change, death, end of data (2022/12/31), or the 365th day after cohort entry. Cox regression models were employed to estimate hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) for each outcome comparing fluticasone/umeclidinium/vilanterol versus beclometasone/glycopyrrolate/formoterol after high-dimensional propensity score matching.

Results: There were 12,971 initiators included in the high-dimensional propensity score matched cohort. The HR suggested a lower risk of severe and moderate exacerbations (0.80 [95% CI 0.69-0.93] and 0.80 [95% CI 0.74-0.87], respectively) and a marginally non-significant decreased risk of pneumonia (0.85 [95% CI 0.70-1.02]) associated with fluticasone/umeclidinium/vilanterol. However, both treatments showed a similar risk of composite cardiovascular events (0.96 [95% CI 0.69-1.35]). The results were generally consistent across several pre-specified sensitivity and subgroup analyses. Of note, among patients treated for ≥ 90 days (nearly 73% of the initiators), the differences in clinical outcomes of both treatments tended to be minimal, with an HR of 0.98 (95% CI 0.78-1.23) for severe exacerbations, 0.93 (95% CI 0.72-1.20) for pneumonia, and 1.07 (95% CI 0.64-1.77) for composite cardiovascular

events. Nevertheless, fluticasone/umeclidinium/vilanterol remained having a lower risk of moderate exacerbations (0.86 [95% CI 0.74-0.98]).

Conclusion: This cohort study conducted in an Asian COPD population suggests that fluticasone/umeclidinium/vilanterol may be a preferred initial treatment option over beclometasone/glycopyrrolate/formoterol. While among patients who are able to maintain their therapies for ≥ 90 days, both treatments may demonstrate more comparable effectiveness and safety profiles.

Keywords: Adrenergic beta-2 receptor agonists; Cohort study; Glucocorticoids; Muscarinic antagonists; Pulmonary disease, chronic obstructive; Single-inhaler triple therapies.

Conflict of interest statement

Declarations. Competing interests: The authors declare no competing interests. Ethics approval: The National Yang-Ming Chiao Tung University Research Ethics Committee approved the study on 2023/05/30 (ID: NYCU112102AE). Informed consent was waived given the retrospective nature of the study and the analysis of anonymous data.

- 45 references
- 2 figures

Supplementary info

Publication types, MeSH terms, Substances, Grants and fundingExpand

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Review

Ann Pharmacother

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Novel Maintenance Therapies for Chronic Obstructive Pulmonary Disorder

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Affiliations Expand

- PMID: 41190453

- DOI: 10.1177/10600280251382202

Abstract

Objective: To summarize the Global Initiative for Chronic Obstructive Lung Disease (GOLD) updates and data for novel therapies/interventions.

Data sources: Articles gathered from MEDLINE, Cochrane Reviews, and PubMed databases. Package inserts for novel agents were utilized. Search terms included chronic obstructive pulmonary disorder (COPD), inhaled corticosteroids (ICS), inhaled long-acting muscarinic antagonists (LAMA), inhaled long-acting beta-2 agonists (LABA), dupilumab, ensifentrine, icenticaftor, and itepekimab.

Study selection and data extraction: English-language primary literature and review articles evaluated. GOLD guidelines from 2001 to 2025 were utilized.

Data synthesis: Six meta-analyses and one randomized controlled trial (RCT) favor initial LAMA/LABA combination over monocomponent agents. Three RCTs note increased ICS efficacy with blood eosinophils >300 cells/μL. One meta-analysis and 8 RCTs address vaccinations, specifically RSV and pneumococcal. Novel medications ensifentrine, dupilumab, and icenticaftor each have 2 RCTs in patients with persistent symptoms despite optimized LAMA/LABA and ICS. Itepekimab is in phase 3 studies. The CAPTURE tool has 2 RCTs validating screening patients with unrecognized COPD.

Relevance to patient care and clinical practice: Identification of notable changes to optimal COPD management, including LABA/LAMA therapy, judicious ICS use, updated vaccine recommendations, and potential roles for ensifentrine and dupilumab.

Conclusion and relevance: Evidence supports LABA/LAMA with nuanced use of ICS. Several novel therapies are approved or are being studied for patients suboptimally controlled. Pharmacists can assist in medication optimization and access; however, patient-specific factors like exacerbation history, blood eosinophils, and COPD endotype must be considered. Implementing early identification screening tools such as CAPTURE should factor into patient assessment.

Keywords: bronchodilators; chronic obstructive pulmonary disease; inhaled corticosteroids; pharmaceutical care; vaccines.

Conflict of interest statement

Declaration of Conflicting InterestsThe authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Supplementary info

Publication types, MeSH terms, SubstancesExpand

Full text links

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Cite

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Ann Am Thorac Soc

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Pulmonary vascular features on chest computed tomography differentially associate with adverse outcomes in smokers in COPDGene

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Affiliations Expand

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- DOI: 10.1513/AnnalsATS.202507-800OC

Abstract

Rationale: In smokers with and without chronic obstructive pulmonary disease (COPD), the differential strengths of association between chest computed tomography (CT)-based metrics of pulmonary vascular disease and adverse outcomes are unknown.

Objectives: We aimed to quantify the differential strengths of association of CT features, from the distal pulmonary arteries to the central great vessels and cardiac chambers, with acute respiratory exacerbations (AREs) and mortality in smokers with and without COPD.

Methods: Smokers with and without COPD with pulmonary vascular morphology and outcomes data were identified in COPDGene. Negative binominal and multivariable Cox proportional hazard models were used to investigate the

association of CT features, including volume of the distal pulmonary arterial vasculature or pruning (<5 mm2 normalized to total arterial blood vessel volume [aBV5/aTBV]), preacinar vessels (5-20 mm2), and pulmonary artery to aorta (PA/Ao) and right to left ventricular epicardial volume (RV/LV) ratios, with outcomes. Kaplan-Meier curves were used to describe pruning risk on mortality.

Results: A total of 3169 smokers with COPD and 2530 smokers without COPD were analyzed. Among smokers with COPD, PA/Ao was the only imaging feature significantly associated with AREs (incidence rate ratio, 1.08 [95% CI, 1.04-1.12]), even after adjusting for aBV5/aTBV. Conversely, pruning demonstrated the strongest association with mortality, even in smokers without COPD (hazard ratio, 1.22 [95% CI, 1.14-1.30] and 1.26 [95% CI, 1.11-1.42], respectively). The association of preacinar vessels with mortality in smokers with COPD and in those without COPD, but with significant emphysema on imaging (≥5%), was novel.

Conclusions: Pruning is significantly associated with mortality risk in smokers with and without COPD; however, PA/Ao selectively associates with AREs in COPD, even when accounting for distal vasculopathy.

Keywords: COPD; chest computed tomography; pulmonary vasculature.

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Affiliations Expand

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- DOI: 10.1080/00207454.2025.2577353

Abstract

Background: Chronic obstructive pulmonary disease (COPD) and stroke are leading contributors to mortality and disability. We analyzed national trends in deaths attributed to co-occurring stroke and COPD in US adults from 1999-2023.

Methods: Using CDC WONDER multiple cause-of-death data for adults aged ≥25 years, we identified decedents with co-existent stroke (ICD-10 I60.x, I61.x, I63.x, I64, I69.0, I69.1, I69.3, I69.4) and COPD (J40-J44). Crude and age-adjusted mortality rates (AAMRs) per 100,000 population were calculated using the 2000 US standard population. Joinpoint regression estimated annual percent changes (APCs) and identified trend inflection points. Analyses were stratified by sex, race/ethnicity, metropolitan status, and state.

Results: From 1999 to 2023, 311,375 deaths involved concurrent stroke and COPD. Overall AAMRs declined from 8.04 per 100,000 in 1999 to 5.17 in 2009 (APC -4.40%), with continued decline to 2018 (APC -1.34%). Rates then increased through 2020-2021 (APC +7.33%) before a modest decline toward 2023. Males consistently exhibited higher AAMRs than females. Non-Hispanic White adults had the highest AAMRs, whereas Hispanic and Asian/Pacific Islander groups had lower rates. Non-metropolitan areas experienced consistently higher AAMRs than metropolitan areas. State-level analyses identified the highest burdens in Appalachian and Deep South regions.

Conclusion: Mortality from coexisting stroke and COPD declined for two decades but rose around the COVID-19 period, revealing significant sex, racial/ethnic, and geographic disparities. These findings highlight the need for targeted prevention, improved access to care for high-risk populations, and further research into mechanisms driving recent inflection points. Data and methods are detailed in the manuscript.

Keywords: CDC WONDER; Chronic obstructive pulmonary disease; stroke.

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J Nurs Care Qual

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A Qualitative Study of the Lived Transition in Care Experiences of Patients With COPD Over the Age of 65

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Affiliations Expand

- PMID: 40658919

- DOI: 10.1097/NCQ.0000000000000893

Abstract

Background: The management of chronic obstructive pulmonary disease (COPD) is complex due to comorbidities, symptom variability, and the need for long-term care, which can lead to high 30-day hospital readmission rates.

Purpose: The purpose of this study was to explore the lived experiences of patients with COPD discharged from the hospital setting, focusing on the patients' perspectives on discharge planning to enhance care transitions and reduce readmissions.

Methods: A semi-structured qualitative design was employed over 3 months, involving 10 patients with COPD.

Results: Key areas for improving care transitions include anxiety management, enhancing nursing education, strengthening support for patients and care partners, preventing functional decline, standardizing care, and increasing patient engagement in discharge planning.

Conclusions: Targeting these opportunities may enhance patient outcomes and optimize care transitions.

Keywords: COPD; anxiety management; care transitions; education; functional decline; mobility.

Conflict of interest statement

The authors declare no conflicts of interest.

- 11 references

Supplementary info

MeSH termsExpand

"Multimorbidity"[Mesh Terms] OR Multimorbidity[Text Word]

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Review

Expert Opin Drug Saf

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Safe prescribing of antihypertensive drugs in the elderly and managing the risk of adverse events

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Abstract

Introduction: Older patients with hypertension have unique clinical challenges based on age-related physiological changes, multimorbidity, and vulnerability to drug side effects. The 2017 American College of Cardiology/American Heart Association guidelines propose initiating antihypertensive therapy at a systolic blood pressure threshold of 130 mm Hg for non-institutionalized ambulatory adults over the age of 65. They also recommended individualized treatment for adults with advanced frailty or high risk of adverse events.

Areas covered: This article reviews evolving paradigms and strategies in blood pressure therapy in elderly individuals, based on shifting the approach from

stringent treatment targets toward individualized management. Step-care management is one such strategy that involves prudent introduction of low doses of antihypertensives with regular follow-up to avoid side effects. Deprescribing is increasingly being promoted as a key intervention in reducing polypharmacy and maximizing safety in at-risk groups of patients.

Expert opinion: There is an increased need to incorporate frailty and functional assessments into routine practice and clinical trials. Despite growing evidence, there are still implementation challenges like insufficient robust long-term data for the frail populations. Pragmatic trials, adoption of digital solutions, and implementation of individualized goals must be the target of future research.

Keywords: Hypertension; adverse drug events; de-prescription; elderly; frailty; shared decision making.

Supplementary info

Publication typesExpand

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Semin Respir Crit Care Med

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Role of Vaccination in the Prevention of ECOPD

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Abstract

Exacerbations of chronic obstructive pulmonary disease (ECOPD) represent key events in the natural history of COPD and are associated with several adverse outcomes. Respiratory infections are major and potentially modifiable triggers of ECOPD, with viral pathogens such as the influenza virus, respiratory syncytial virus (RSV), and SARS-CoV-2, as well as bacterial infections caused by Streptococcus pneumoniae, playing a central role. This narrative review examines the current evidence supporting vaccination as a preventive strategy for ECOPD and discusses its translation into clinical practice. The biological rationale for vaccination in COPD is reviewed, including disease-related immune dysregulation, impaired mucociliary clearance, and increased susceptibility to respiratory pathogens. Evidence from randomized clinical trials, observational studies, meta-analyses, and real-world data is summarized for pneumococcal, influenza, SARS-CoV-2, and RSV vaccines. Pneumococcal vaccination has been shown to reduce the burden of community-acquired pneumonia and invasive pneumococcal disease, with conjugate and higher-valent vaccines providing enhanced immunogenicity in older and high-risk adults. Influenza vaccination consistently reduces severe exacerbations, hospitalizations, and mortality, with additional cardioprotective effects of relevance in COPD. SARS-CoV-2 vaccination markedly lowers the risk of severe COVID-19 and related respiratory deterioration in COPD, while recently licensed RSV vaccines offer a novel opportunity to prevent RSV-associated lower respiratory tract disease and potentially reduce exacerbation risk. Patient populations most likely to benefit from vaccination include frequent exacerbators, older adults, individuals with severe airflow limitation, multimorbidity, immune dysfunction, infection-prone phenotypes, and socially vulnerable groups. Future perspectives include precision vaccination strategies, novel vaccine platforms, coadministration approaches, and interventions to improve vaccine uptake. Vaccination emerges as a cornerstone of ECOPD prevention, with substantial potential to reduce exacerbation burden and improve long-term outcomes in COPD.

Conflict of interest statement

The authors declare that they have no conflict of interest.

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Lancet Reg Health Eur

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors)

showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for

lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma, and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this

work. S Principe reports grants from Innovative Medicines Initiative 2 Joint Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards (payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-

chair of the Nordic severe asthma network; member of the steering committee of SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand, Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

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Clinical outcomes and safety of SGLT2 inhibitors in the older population with heart failure: A systematic review and meta-analysis

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Abstract

Background: Heart failure (HF) predominantly affects older adults, yet this group remains underrepresented in sodium-glucose cotransporter-2 inhibitor (SGLT2i) trials. Given their frailty and multimorbidity, clarifying the potential benefit and safety of SGLT2i in older HF patients is essential.

Methods: PubMed, ScienceDirect, and Cochrane Central were searched through August 2025 for randomized controlled trials (RCTs) and observational studies comparing SGLT2i with control in patients aged ≥65 years with HF. Hazard ratios (HRs), risk ratios (RRs), and mean differences (MDs) were pooled using random-effects models.

Results: Ten studies (4 RCTs, 6 cohorts; n = 20,844) were included. SGLT2i was associated with a lower hazard of all-cause mortality (HR 0.81 [95% CI 0.72-0.90]; p < 0.001), cardiovascular (CV) death (HR 0.83 [0.74-0.94]; p = 0.004), first HF hospitalization (HR 0.73 [0.66-0.80]; p < 0.001), composite CV death or HF hospitalization (HR 0.78 [0.70-0.87]; p < 0.001), and rehospitalization (HR 0.60 [0.53-0.69]; p < 0.001). SGLT2i lowered serious adverse events (RR 0.92 [0.89-0.95]; p < 0.001) and slowed renal function decline (MD 1.86 [1.15-2.58] mL/min/1.73 m^2 per year; p < 0.001). An increase was observed in genital (RR 3.07 [2.03-4.64]; p < 0.001) and urinary tract infections (RR 1.19 [1.03-1.38]; p = 0.02).

Conclusions: In older patients with HF, SGLT2i was associated with lower mortality and HF hospitalizations and with a slower renal decline, while largely maintaining a favorable safety profile. These findings support the consideration of SGLT2i as an important therapeutic option for older adults with HF.

Keywords: Aged; Cardiovascular mortality; Heart failure; Hospitalization; Meta-analysis; Renal outcomes; Safety; Sodium–glucose cotransporter-2 inhibitors.

Conflict of interest statement

Declaration of competing interest The 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.

Supplementary info

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J Affect Disord

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Multimorbidity and memory-related disorders in older adults: A cross-national study based on three large longitudinal cohorts

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Affiliations Expand

- **PMID: 41448400**
- **DOI: 10.1016/j.jad.2025.120995**

Abstract

Background: Memory-related disorders pose a growing burden on ageing populations, yet the global understanding of how multimorbidity links to such cognitive decline remains limited. This study aimed to examine the exposure-response relationship between multimorbidity-including its specific patterns-and memory impairment in older adults, and identify key mediating mechanisms.

Methods: A total of 20,242 participants aged 50 years old and above from three prospective and representative cohorts were included. Memory-related disorders were assessed via self-reported physician diagnoses, and multimorbidity was defined as the coexistence of two or more chronic diseases in the same individual.

Time-dependent Cox proportional hazards regression models evaluated the association between multimorbidity and memory-related disorders, and identified high-risk multimorbidity patterns. Random forest-based double machine learning was used to identify mediating mechanisms.

Results: Multimorbidity was associated with a 111.8 % higher risk of memory-related disorders. Mental multimorbidity (HR = 2.664) and cardiometabolic multimorbidity (HR = 1.656) conferred the highest risk, while pulmonary multimorbidity showed moderate risk (HR = 1.227). Transition to multimorbidity correlated with increased vision loss (risk increment = 0.183), hearing loss (risk increment = 0.199), and worse BADL and IADL conditions. Vulnerable subgroups included older adults aged 50-64 years (HR = 2.620), females (HR = 2.114), those with lower educational attainment (HR = 2.216), non-retirees (HR = 2.557), smokers (HR = 2.296), drinkers (HR = 2.178), and those without investments (HR = 2.137).

Conclusion: Multimorbidity elevates memory-related disorder risk in older adults, with sensory and functional impairment as key mediators. Health policies should integrate standardized cognitive check-ups, sensory and functional support, community anti-isolation interventions, and optimized electronic health records into national ageing frameworks.

Keywords: Healthy ageing; Memory-related disorders; Multimorbidity; Older adults.

Conflict of interest statement

Declaration of competing interest The 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.

Supplementary info

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J Adv Nurs

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Gender Differences in Disease Burden, Symptom Burden, and Quality of Life Among People Living With Heart Failure and Multimorbidity: Cross-Sectional Study

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- PMCID: PMC12312650
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Abstract

Aim: Heart failure is a leading cause of hospitalisation and often coexists with seven comorbid conditions on average. This study aimed to examine the gender differences in disease burden, symptom burden, and quality of life among older adults with heart failure and multimorbidity.

Design: Cross-sectional study.

Methods: This study utilised a baseline survey from an ongoing cohort study in 2022-2023. Adults aged ≥ 50 years with heart failure and more than one chronic condition were recruited from a university-affiliated hospital using an electronic patient portal. Disease burden was measured using a modified Disease Burden Impact Scale. The Edmonton Symptom Assessment Scale and EuroQoL-5D-5L assessed symptom burden and quality of life. Gender differences in baseline outcomes were examined using Pearson's Chi-square tests, Welch's t-tests, and multiple linear regressions.

Results: Among 353 participants who completed the baseline survey, the mean (±SD) age was 70 (±9.5) years, and 50.1% were women (mean age: 67 ± 9 vs. men: 72 ± 10). In adjusted models, women had 4.9 points higher disease burden (p = 0.003) and reported higher symptom scores of pain (p = 0.018), tiredness (p = 0.021), nausea (p = 0.007), and loss of appetite compared to men (p = 0.036). Women had significantly more moderate/severe problems in usual activities and pain/discomfort and 0.07 points lower EuroQoL index than men (p = 0.010).

Conclusions: There were gender differences in disease/symptom burdens and quality of life. Women living with heart failure and multimorbidity had higher burdens but lower quality of life.

Impact: Identifying gender differences among people with heart failure and multimorbidity can be the first step to explaining health disparities. Research should take more inclusive and equitable approaches to address these differences. Healthcare providers, including nurses, should implement targeted strategies for effective multimorbidity management by considering these differences and disparities in clinical settings.

Reporting method: STROBE checklist, cross-sectional.

Patient or public contribution: No patient or public contribution.

Keywords: aged; chronic illness; gender; heart failure; multimorbidity; nursing; quality of life; symptom burden.

Conflict of interest statement

Conflict of interest disclosure

No conflict of interest has been declared by the authors.

- **Cited by 3 articles**
- **41 references**

Supplementary info

MeSH terms, Grants and fundingExpand

"asthma"[MeSH Terms] OR asthma[Text Word]

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Nat Med

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Benralizumab versus placebo for hypereosinophilic syndrome: a randomized, placebo-controlled phase 3 trial

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Affiliations Expand

- **PMID: 41917160**

- DOI: 10.1038/s41591-026-04315-8

Abstract

Benralizumab, an eosinophil-depleting anti-IL-5 receptor α antibody, has demonstrated efficacy in severe eosinophilic asthma and eosinophilic granulomatosis with polyangiitis and shown promising results in hypereosinophilic syndrome (HES). NATRON was a randomized, double-blind placebo-controlled phase 3 study evaluating the efficacy and safety of benralizumab in FIP1L1::PDGFRA-negative HES. The primary endpoint was time to first HES flare. In total, 133 patients (median (range) age 51 (14-87) years, 62% female) were randomized (1:1) to receive benralizumab 30 mg every 4 weeks or placebo for 24 weeks, in addition to background therapy. Benralizumab significantly reduced the risk of first flare versus placebo (hazard ratio 0.35, 95% CI 0.18 to 0.69, P = 0.0024). Adverse events occurred in 64.2% and 66.7% of benralizumab- and placebo-treated patients, respectively. Benralizumab's safety was consistent with its known profile. These results demonstrate the efficacy and safety of add-on benralizumab in the treatment of HES. ClinicalTrials.gov identifier: NCT04191304 .

Conflict of interest statement

Competing interests: P.U.O. has received grant funding from AstraZeneca, Blueprint, DBV Technologies and GSK, as well as consulting fees from AstraZeneca, Novartis and BioCryst. F.R. has received consulting fees from GSK, AstraZeneca and Merck; honoraria from AstraZeneca and GSK; and royalties from UpToDate. P.A. has received grant funding from AstraZeneca, Sanofi and Recode, as well as consulting fees from AstraZeneca, Connect Biopharma, Sanofi, Regeneron, GSK, Amgen, Vida Ventures and Enveda. P. Kuna has received honoraria from Adamed, Angellini, AstraZeneca, Berlin Chemie Menarini, Glenmark, Chiesi, GSK, Celon Pharma, Polpharma, Sanofi and Teva. M.G. has received consulting fees from AstraZeneca and GSK. A.R. has received grant funding from Abbvie, AOP, AstraZeneca, BMS, Blueprint Medicines, GSK and Incyte; consulting fees from Abbvie, AOP, AstraZeneca, BMS, Blueprint Medicines, GSK, Incyte and Novartis; and honoraria from AOP, AstraZeneca, BMS, Blueprint Medicines, GSK and Novartis. S.H.S. has received consulting fees from AstraZeneca, GSK, Areteia Therapeutics, Sanofi and Chiesi; honoraria from AstraZeneca, GSK, Chiesi and Medscape; and has a patent in breath analysis and biomarkers submitted at the time of writing. P.G.N.J.M. has received grant funding from AstraZeneca. L.M.B. is employed by AstraZeneca and may hold stock in AstraZeneca. A.B. is employed by AstraZeneca and holds stock in AstraZeneca, Pfizer and GSK. G.B. is employed by AstraZeneca and may hold stock in AstraZeneca. L.G.B. is employed by AstraZeneca and may hold stock in AstraZeneca. J.F. is employed by AstraZeneca and may hold stock in AstraZeneca. H.G. is employed by AstraZeneca and may hold stock in AstraZeneca. C.N.H. is employed by AstraZeneca and may hold stock in AstraZeneca. P.J. is employed by AstraZeneca and may hold stock in AstraZeneca. R.L.P. is employed by AstraZeneca and may hold stock in AstraZeneca. M.L.J. is employed by AstraZeneca and may hold stock in AstraZeneca. A.D.K. has received royalties from UpToDate. The other authors declare no competing interests.

- 29 references

Supplementary info

Associated data, Grants and fundingExpand

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Pediatr Rev

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. 2026 Apr 1;47(4):234-236.

doi: 10.1542/pir.2025-006812.

Single Maintenance and Reliever Therapy for the Pediatric Patients With Asthma

Mary Nguyen Jacobson [1] [2], **Bridgette L Jones** [1] [2]

Affiliations Expand

- **PMID: 41916547**

- **DOI: 10.1542/pir.2025-006812**

No abstract available

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Cite

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Paediatr Perinat Epidemiol

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. 2026 Mar 31.

doi: 10.1111/ppe.70141. Online ahead of print.

The Effectiveness of the Palivizumab Programme in Reducing the Risk of Paediatric Asthma: A Population-Based Study in Ontario, Canada

Kimberley A Foley [1 2], Sarah A Buchan [3 4 5 6 7], Dayre McNally [8], Michelle Dimitris [9], Sarah Swayze [6], Jeffrey C Kwong [3 4 5 6 10 11], Steven Hawken [6 12], Sonia Saxena [1], Dougal Hargreaves [2 13], Tiffany Fitzpatrick [3 4 5 6 7]

Affiliations Expand

- PMID: 41914371

- DOI: 10.1111/ppe.70141

Abstract

Background: Palivizumab was introduced in Canada in 1998 as a publicly funded programme to reduce respiratory syncytial virus (RSV) disease in high-risk children. Severe early-life RSV infections are associated with increased asthma risk. Thus, palivizumab may also indirectly reduce paediatric asthma.

Objective: To evaluate the effectiveness of Ontario, Canada's palivizumab programme in decreasing paediatric asthma.

Methods: We used multiple linked population-based administrative databases to identify all children born in Ontario between 1993 and 2013, with follow-up through March 2020. Our primary outcome was physician-diagnosed asthma by age 7. Controlled interrupted time-series analysis was used to compare changes in annual asthma incidence (by birth year) before and after palivizumab's introduction, according to programme eligibility (clearly, possibly, or ineligible). Socio-demographic differences were explored via stratification.

Results: Nearly 3 million children were included in this study, including 406,596 (14.6%) diagnosed with asthma by age 7. Asthma incidence substantially declined over the study period, with the greatest declines among palivizumab-ineligible children (35.3%, 95% confidence interval [CI] 28.3, 42.6, versus 18.1%, 95% CI 13.9, 22.9 among clearly eligible children). However, relative to ineligible children, post-palivizumab declines were most apparent among possibly eligible children, with an additional annual decline of 2.0% (95% CI 0.3, 3.7) in asthma. Socio demographic differences in asthma incidence and post-palivizumab declines were noted. Particularly, incidence was higher among children born to teenage mothers than among those aged 19+ years; this gap narrowed over time, especially among possibly eligible children.

Conclusions: Asthma incidence declined over this 20-year study; however, smaller declines were observed among children clearly eligible for palivizumab relative to ineligible children. However, exploratory evidence was suggestive of reduced social inequities in asthma post-palivizumab, particularly among possibly eligible children. Larger reductions in asthma might be realized with the introduction of population-

based RSV immunization programmes through broader programme eligibility and reductions in severe RSV disease.

Keywords: asthma; child health; interrupted time series analysis; palivizumab; respiratory syncytial virus.

- 62 references

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4

Review

Aust N Z J Obstet Gynaecol

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. 2026 Apr;66(2):e70120.

doi: 10.1111/ajo.70120.

Asthma Control During Pregnancy and Adverse Perinatal Outcomes: A Systematic Review

Emilie Johanne Vedtofte Haastrup [1], Esther Bay Smedegaard [1], Anne Vejen Hansen [1], Charlotte Suppli Ulrik [1 2]

Affiliations Expand

- PMID: 41913695
- PMCID: PMC13036474
- DOI: 10.1111/ajo.70120

Abstract

Background and aim: Poor control of maternal asthma during pregnancy may increase the risk of pregnancy complications and adverse perinatal outcomes. The present review provides an update on the current knowledge of the association between asthma control during pregnancy and pregnancy complications, which are pre-eclampsia, low birth weight (LBW), pre-term birth, and small for gestational age (SGA).

Methods: This review was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.

Results: A total of 14 studies fulfilled the predefined criteria for inclusion in the present review. The included studies revealed that the presence of maternal asthma per se is associated with an increased risk of SGA, whereas the observations were contradictory in relation to pre-eclampsia, LBW and pre-term birth. Asthma exacerbations and uncontrolled asthma during pregnancy were associated with an increased risk of pre-eclampsia. The studies did, however, not consistently suggest an increased risk of LBW, pre-term birth or SGA associated with asthma exacerbations during pregnancy.

Conclusions: Apart from small for gestational age, maternal asthma itself is not associated with a higher risk of pregnancy complications and adverse perinatal outcomes. However, asthma exacerbations and uncontrolled asthma during pregnancy increase the risk of pre-eclampsia.

Keywords: asthma; asthma control; pregnancy complications.

Conflict of interest statement

E.J.V.H., E.B.S. and A.V.H. have no conflicts of interest to declare. C.S.U. has received personal fess from AstraZeneca, GSK, TEVA, Chiesi, Sanofi Genzyme, Boehringer-Ingelheim, Orion Pharma, Novartis, ALK-Abello, Mundipharma, Berlin Chemie, Pfizer, TFF Pharmaceuticals, Covis Pharma Hikma Pharmaceuticals, Novo Nordisk, Roche and Actelion outside the submitted work.

- **25 references**
- **1 figure**

Supplementary info

Publication types, MeSH termsExpand

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Cite

5

Respiration

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. 2026 Mar 30:1-12.

doi: 10.1159/000551651. Online ahead of print.

Biologic Therapy Timing and Severe Asthma Progression: Insights from the Italian Registry on Severe Asthma

Matteo Martini, Lorenzo Cecchi, Maria Aliani, Leonardo Antonicelli, Francesco Bini, Fausto De Michele, Nicola Cosimo Facciolongo, Rosa Fasano, Francesco Menzella, Antonino Musarra, Adriano Vaghi, Giuseppe Valenti, Claudio Micheletto, Maria Beatrice Bilò

- **PMID: 41911079**

- **DOI: 10.1159/000551651**

Abstract

Introduction: Timely initiation of biologic therapies is crucial in the management of severe asthma. However, delays in diagnosis and access to advanced treatments are common in real-life settings. We investigated disease progression and trajectories, with a focus on the impact of treatment latency among patients with severe asthma.

Methods: This longitudinal observational analysis included adult patients with severe asthma enrolled in the Italian Registry on Severe Asthma (IRSA). Data were stratified by use and timing of biologic therapy. Two core analyses were conducted: (1) characterization of patients not receiving biologics; (2) evaluation of disease progression and the relationship with the timing of biologic initiation.

Results: At baseline, 43.7% of the 2,114 patients were not receiving biologics, despite having severe disease and a T2-high phenotype. The average time of progression from initial diagnosis to severe asthma was 17 years. The median time from severe asthma diagnosis to biologic initiation was 2 years (IQR: 0.9-4.2). At the 1-year follow-up, patients who initiated biologics after enrollment showed the most significant clinical improvements compared with patients without biologic treatment or already on biologics at baseline, including a 64.8% reduction in the number of exacerbations ($p=0.019$), a mean reduction in annual exacerbation rate of 4.1 events ($p<0.001$), and an increase of 6.3 points in ACT score ($p<0.001$).

Conclusion: Severe asthma often develops after a prolonged progression from initial symptom onset. Structured referral and earlier biologic initiation may be associated with better outcomes and potentially influence the clinical trajectory of severe asthma.

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J Investig Allergol Clin Immunol

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. 2026 Mar 30:0.

doi: 10.18176/jiaci.1156. Online ahead of print.

Real-World Experience With Dupilumab in Severe Asthma: The Spanish Multicenter Study DUPImpact

Hemily Izaguirre-Flores [1], Ebymar Arismendi [2 3], Elena Martín-Ruiz de la Rosa [4], Berta Román-Bernal [5], Irene De Lorenzo-García [4], Inmaculada Plasencia-García [6], Annety Juanes-Olite [7], Candelaria Martín-García [1], Yurena Hernández Galván [7], Cristina Bellver-Asperilla [8 9], Alberto García-De la Fuente [3], Carlos Cabrera-López [10], Mariana Muñoz-Esquerre [8 9]

Affiliations Expand

- PMID: 41906934

- DOI: 10.18176/jiaci.1156

Abstract

Background and objective: Dupilumab has proven effective in asthma in clinical trials, although real-world evidence remains limited. While tools such as FEOS and EXACTO may aid in evaluating effectiveness, evidence supporting their use remains scarce. Objective: To evaluate the real-world effectiveness of dupilumab in a Spanish cohort of patients with uncontrolled severe type 2 (T2) asthma.

Methods: DUPImpact was a retrospective, multicenter study including adults with severe uncontrolled T2 asthma treated with dupilumab in 7 Spanish hospitals. Clinical outcomes were evaluated at 6 and 12 months and included the annual exacerbation rate (AER), asthma control (Asthma Control Test [ACT]), lung function (FEV1), and maintenance oral corticosteroid (OCS) use. Response was quantified using the FEOS and EXACTO scales.

Results: The study population comprised 94 patients, of whom most had previously received biologics and 16% were OCS-dependent. After 12 months, AER decreased significantly, asthma control improved for most patients (ACT ≥20), and the mean increase in FEV1 was >300 mL. The number of patients receiving maintenance OCS decreased from 15 to 5, and the mean daily dose tended to decrease (not significant). According to the FEOS and EXACTO scales, over 70% of patients were classified as good responders, with 31% classified as super-responders.

Conclusions: The DUPImpact study confirms the real-world effectiveness of dupilumab in reducing exacerbations and OCS use and improving asthma control and lung function. FEOS and EXACTO enable a multidimensional assessment beyond individual measures.

Keywords: Asthma; Dupilumab; EXACTO; FEOS; Real-world evidence.

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Otolaryngol Head Neck Surg

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. 2026 Mar 29.

doi: 10.1002/ohn.70227. Online ahead of print.

FDA Approval of Respiratory Biosimilars-Turning the Tide of Cost-Effectiveness?

Ashley L Miller [1 2], Charles A Elmaraghy [1 2], Casey D Curtis [2 3], Vinay K Rathi [2 4]

Affiliations Expand

- **PMID: 41904981**

- **DOI: 10.1002/ohn.70227**

Abstract

In March 2025, the Food and Drug Administration (FDA) approved the first respiratory biosimilar: omalizumab-igec. FDA designated omalizumab-igec as "interchangeable" with the anti-IgE antibody omalizumab, which will permit pharmacists to perform substitution without consulting providers. This biosimilar will compete with blockbuster biologics indicated for the treatment of chronic

rhinosinusitis with nasal polyposis (CRSwNP) and other inflammatory airway diseases. Biologics are presently not cost-effective for CRSwNP and should be reserved for patients who have not responded to conventional therapy (eg, nasal steroids and endoscopic sinus surgery) or are unfit for surgery. However, market entry of omalizumab-igec and other omalizumab biosimilars in late-stage clinical development may spur substantial price reductions if biosimilar manufacturers are able to overcome barriers such as patient and physician skepticism and restrictive state pharmacy laws (eg, requiring patient consent for biosimilar substitution). Comparative cost-effectiveness research is necessary to inform coverage policies and treatment decisions by patients and clinicians.

Keywords: FDA; allergy; asthma; biologic; biosimilar; chronic sinusitis; cost-effectiveness; nasal polyps; omalizumab; omalizumab-igec.

- 10 references

Full text links

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8

Review

Expert Rev Respir Med

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. 2026 Mar 30:1-15.

doi: 10.1080/17476348.2026.2651412. Online ahead of print.

Daytime sleepiness in patients with obstructive sleep apnea and associated comorbidities

Claudia Di Chiara [1], Giulia Sartori [1], Nadia Castaldo [2], Alberto Fantin [1 2], Marcello Ferrari [1], Ernesto Crisafulli [1]

Affiliations Expand

- PMID: 41883309

- DOI: 10.1080/17476348.2026.2651412

Abstract

Introduction: Obstructive sleep apnea (OSA) is characterized by recurrent upper-airway collapse during sleep, leading to ineffective respiratory efforts, intermittent hypoxia, and sleep fragmentation. Patients with OSA often have comorbid conditions. Excessive daytime sleepiness (EDS), defined as an inability to remain awake during the day, is common among patients with OSA; however, its perception may vary with comorbidities that affect autonomic and neuroendocrine regulation.

Areas covered: We reviewed studies examining the prevalence and clinical impact of EDS in patients with OSA and its main comorbidities, published between January 2000 and September 2025, and identified through Medline.

Expert opinion: EDS is highly prevalent in patients with OSA and arterial hypertension, cardiac arrhythmias, cerebrovascular comorbidities (particularly in those with thalamic or pontine lesions), diabetes mellitus, metabolic syndrome, asthma, chronic kidney disease, and cancer. By contrast, EDS appears less prevalent in patients with heart failure, treated cerebrovascular and neurodegenerative disease (particularly in those receiving levodopa, selective serotonin reuptake inhibitors, or bromocriptine), and chronic obstructive pulmonary disease (COPD).In conclusion, in patients with OSA, EDS is perceived differently depending on comorbidity. Consequently, EDS assessment should follow a personalized, multidimensional approach that recognizes its clinical relevance while accounting for variability across comorbid conditions.

Keywords: Obstructive sleep apnea; cardiovascular disease; chronic obstructive pulmonary disease; comorbidities; diabetes; excessive daytime sleepiness.

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Review

J R Soc N Z

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. 2026 Mar 8;56(2):e70015.

doi: 10.1002/snz2.70015. eCollection 2026 Apr.

Anti-Inflammatory Reliever Therapy in Asthma: A Review

Richard Beasley [1 2], Mitesh Patel [3], Karen Oldfield [1], Lee Hatter [1 2], Pepa Bruce [1 2], Jonathan Noble [1 2], Allie Eathorne [1], Mark Weatherall [4]

Affiliations Expand

- PMID: 41804386
- PMCID: PMC12967592 (available on 2027-03-08)
- DOI: 10.1002/snz2.70015

Abstract

Anti-inflammatory reliever (AIR) therapy is the use of a combination 2-in-1 inhaler which includes a corticosteroid and a fast-onset beta$_2$-agonist, as a reliever therapy in asthma. The clinical research of its use to date has primarily involved the inhaled corticosteroid (ICS) budesonide together with the long-acting beta$_2$-agonist (LABA) formoterol, and budesonide with the short-acting beta$_2$-agonist (SABA) albuterol (salbutamol). Based on high-quality evidence from randomised controlled trials of a substantial reduction in the risk of severe exacerbations, New Zealand and international guidelines recommend ICS/formoterol as the preferred reliever inhaler for use by adolescents and adults with asthma, across the range of asthma severity, rather than SABA reliever-based regimens. Recently, international guidelines have also recommended ICS/SABA reliever as an alternative to a SABA reliever. These recommendations represent the most important paradigm change in asthma management for decades.

Keywords: albuterol; anti-inflammatory reliever therapy; asthma; formoterol; inhaled corticosteroid; salbutamol; short-acting beta2-agonist.

Conflict of interest statement

RB has received institutional research funding and personal fees from AstraZeneca and Teva, and personal fees from Avillion; is Chair of the Asthma and Respiratory Foundation of New Zealand Adolescent and Adult asthma guidelines; and reviewer for the Global Initiaitve for Asthma (GINA) strategy.

- 70 references

Supplementary info

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Multicenter Study

Respir Med

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. 2026 Apr:254:108750.

doi: 10.1016/j.rmed.2026.108750. Epub 2026 Mar 3.

Real-world, long-term effectiveness of dupilumab treatment in patients with severe asthma: 2 years of the ProVENT study

Marek Lommatzsch [1], Olaf Schmidt [2], Hartmut Timmermann [3], Monika Gappa [4], Henrik Watz [5], Jason Kwah [6], Olivier Ledanois [7], Nicole Nischan [8], Matthias Hahn [9], Andreas Heimann [8], Stephanie Korn [10]

Affiliations Expand

- PMID: 41785977

- DOI: 10.1016/j.rmed.2026.108750

Free article

Abstract

Background: Randomized controlled trials have shown clinical efficacy of dupilumab in patients with severe asthma. However, the long-term effectiveness of dupilumab treatment for asthma in real-life is incompletely understood.

Methods: ProVENT (NIS-Nr.: 514; study code: OBS16379) is a non-interventional, prospective, 3-year study in patients aged ≥12 years with severe asthma receiving dupilumab per routine clinical care in Austria, Germany, and Switzerland. This interim analysis of ProVENT assessed lung function, asthma control, quality of life, biomarkers, and clinical remission, over the first 2 years in the study. Safety outcomes will be presented in the final analysis.

Results: 421 patients were screened, and of the 399 patients enrolled in ProVENT, 259 had ≥1 post-baseline assessments and 100 had documented data after 24 months of treatment. At month 24, mean (standard deviation [SD]) improvement from baseline was 0.24 L (0.46) for pre-bronchodilator forced expiratory volume in 1 s (FEV_1); 10.10% (15.80) for pre-bronchodilator percent predicted FEV_1;-0.96 (1.24)

for 5-item Asthma Control Questionnaire score (ACQ-5); 4.4 (5.4) for Asthma Control Test score (ACT); and 0.61 [1.3] for Standardized Asthma Quality of Life Questionnaire overall score (AQLQ [S]). Blood eosinophils, fractional exhaled nitric oxide (FeNO), and total serum immunoglobulin E (IgE) decreased by month 24. Among patients with available data, clinical remission rates were 55.9% at year 1 and 58.0% at year 2.

Conclusion: In real-world patients with severe asthma, long-term dupilumab treatment is associated with sustained improvements in lung function, asthma control, and quality of life. Nearly 60% of patients achieved clinical asthma remission after 2 years.

Keywords: Asthma; Asthma control; Biomarkers; Clinical remission; Dupilumab; Lung function; Real-world study.

Conflict of interest statement

Declaration of competing interest The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: ML reports receiving honoraria for lectures and/or consultant fees from ALK, Allergopharma, APONTIS PHARMA, AstraZeneca, Bencard Allergie, Berlin-Chemie, Boehringer Ingelheim, Bosch, Chiesi, Circassia, GSK, HAL Allergy, Janssen-Cilag, MSD, Mundipharma, Novartis, Nycomed/Takeda, Sanofi, Stallergenes Greer, Teva, and UCB; reimbursement of attendance fees for conferences and educational events and that of travel and accommodation costs from AstraZeneca and Novartis; research support from AstraZeneca, DFG, and GSK; and funding for performing clinical studies from AstraZeneca and Sanofi. OS reports receiving honoraria for lectures and/or consultant fees from AstraZeneca, Boehringer Ingelheim, Chiesi, GSK, Novartis, Sanofi, and Teva; research support from AstraZeneca, Boehringer Ingelheim, GSK, Novartis, and Sanofi. HT reports receiving consultant fees from AstraZeneca, Almirall, Astellas Pharma, Bayer, Berlin-Chemie, Boehringer Ingelheim, GSK, LETI Pharma, Meda, Mundipharma, Novartis, Nycomed, Pfizer, Sanofi, Takeda, and Teva. MG reports receiving clinical trial fees, advisory board and/or, lecture fees from ALK, AstraZeneca, DBV, Evangelisches Krankenhaus (Düsseldorf, Germany), InfectoPharm, Klinik für Kinder-und Jugendliche, Novartis, OM Pharma, Sanofi/Regeneron Pharmaceuticals Inc. HeHWreports consultancy, travel, and speaker fees from AstraZeneca, Bayer, Boehringer Ingelheim, Chiesi, GSK, Novartis, Sanofi, and Takeda. JHK is an employee and shareholder of Regeneron Pharmaceuticals Inc. OL, NN, MH, and AH are employees of Sanofi and may hold stock and/or stock options in the company. SK receives grants/funds, personal fees for lectures and advisory boards from AstraZeneca, GSK, Novartis, Sanofi, and Teva.

Supplementary info

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11

Review

Respir Med

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. 2026 Apr:254:108736.

doi: 10.1016/j.rmed.2026.108736. Epub 2026 Feb 27.

Treatable traits and treatment options in asthma

Shivani Setur Kanabar [1], Ian Douglas Pavord [1], Timothy Stopford Christopher Hinks [2]

Affiliations Expand

- PMID: 41763276

- DOI: 10.1016/j.rmed.2026.108736

Free article

Abstract

In the era of personalised medicine, approaches to asthma assessment and management have shifted significantly. Asthma is no longer viewed as a homogeneous disease; rather, it arises from multiple biological processes with complex inter-relationships with other diseases. This conceptual shift renders difficult and severe asthma highly tractable therapeutically. Effective management therefore relies on the identification and management of these processes. This review discusses these treatable traits in asthma, their pathology, diagnosis and management, providing a structured approach for the holistic care of patients with severe asthma. Current evidence gaps are surveyed and future avenues for targeted treatment are highlighted.

Keywords: Asthma; Asthma assessment; Asthma management; Treatable traits.

Conflict of interest statement

Declaration of competing interest Shivani S Kanabar reports no relevant conflicts of interest. Ian D Pavord reports consulting fees from GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Amgen, Areteia, Kymera, and Pfizer; payments or honoraria for lectures, presentations, speakers bureaus, manuscript writing or educational events from GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Menarini, Chiesi, and Aerocrine outside the submitted work within the last 36 months. Timothy SC Hinks reports grants from The Wellcome Trust, grants from The Guardians of the Beit Fellowship, grants from Pfizer, grants from NIHR Oxford BRC, grants from University of Oxford, grants from Kymab, grants from Arcturis, grants from Asthma + Lung UK during the conduct of the study; personal fees from Astra Zeneca, personal fees from TEVA, personal fees from Peer Voice, personal fees from AZ Pieris, outside the submitted work.

Supplementary info

Publication types, MeSH terms, SubstancesExpand

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12

Respir Med

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. 2026 Apr:254:108731.

doi: 10.1016/j.rmed.2026.108731. Epub 2026 Feb 25.

Anxiety is associated with asthma severity in adolescents: a school-based epidemiological study

Cristiane Ribeiro Veloso da Silva [1], Emília Chagas Costa [2], Emanuel Sarinho [3], Murilo Gominho Antunes Correia Júnior [4], Caroline Ramos de Moura Silva [4], Marcos Andre Moura Dos Santos [5], Mauro Virgílio Gomes Barros [6], Nadia Gaua [7], João Francisco Lins Brayner Rangel Junior [8], Marco Aurélio de Valois Correia Júnior [9]

Affiliations Expand

- **PMID: 41759833**

- **DOI: 10.1016/j.rmed.2026.108731**

Free article

Abstract

Background: Asthma is one of the most prevalent noncommunicable chronic diseases worldwide. Adolescents' emotional state has been associated with asthma symptoms and disease severity. Given the epidemiological relevance of anxiety and asthma during adolescence, it is important to investigate the association between these conditions. The objective of this study is to analyze the association between social anxiety and asthma in adolescents.

Methods: This was a cross-sectional school-based study conducted among adolescents aged 14 to 19 years. Data were collected using the adapted Global School-based Student Health Survey (GSHS), the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire, and the Social Anxiety Scale for Adolescents (SAS-A). Logistic regression analysis was performed to assess the association between asthma and the following variables: social anxiety, age, sex, physical activity level, maternal education, parental asthma, parental smoking, and municipality.

Results: A total of 4514 adolescents were assessed, 54.6% of whom were female. Active asthma was present in 26.5% of participants, while severe asthma was reported by 10%. Mild anxiety affected 46.7%, and moderate to intense anxiety was reported by 27.2% of participants. Adolescents with moderate to intense anxiety had a higher likelihood of active asthma (OR = 1.6; 95%CI: 1.3-2.0) and more than twice the risk of severe asthma (OR = 2.1; 95%CI: 1.5-3.0).

Conclusion: An association was found between asthma and social anxiety symptoms, with increased anxiety levels correlating with greater asthma severity. These findings highlight the potential relevance of considering mental health in asthma management strategies.

Keywords: Adolescents; Anxiety; Asthma; Epidemiology.

Conflict of interest statement

Declaration of competing interest The 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.

Supplementary info

MeSH termsExpand

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Cite

13

Am J Physiol Lung Cell Mol Physiol

. 2026 Apr 1;330(4):L379-L389.

doi: 10.1152/ajplung.00295.2025. Epub 2026 Feb 23.

The impact of estrogen replacement during perimenopause on lung function and airway inflammation in the VCD mouse model

William P Pederson [1 2], Laurie Michelle Ellerman [1], Riley D Hellinger [1 3], Joselyn Joanna Rojas Quintero [4], Francesca Polverino [4], John P Konhilas [2], Julie G Ledford [1 3]

Affiliations Expand

- PMID: 41729584

- DOI: 10.1152/ajplung.00295.2025

Free article

Abstract

Menopause associated asthma impacts a subset of women and is less responsive to current treatments. Mechanisms driving this late-onset asthma are unknown. We recently developed a mouse model of menopause associated asthma using a combination of 4-vinylcyclohexene diepoxide (VCD) and house dust mite (HDM) exposures. The goal of this study was to determine how hormone replacement therapy during perimenopause impacts lung function and inflammation. The experimental groups included menopausal mice (VCD) with and without exposure to HDM (to model allergic airways disease) and menopausal mice with and without hormone replacement therapy (HRT; via estrogen pellet implantation). Lung function during methacholine challenge was assessed by flexiVent. Serum, bronchoalveolar lavage fluid (BALF), and histological samples were collected for assessment. Mice that received HRT during perimenopause had enhanced airway hyperresponsiveness (AHR) detected by total airway resistance (R_{rs}), tissue damping (G), and downward shifts in pressure-volume (PV) curves compared with controls, independent of HDM challenge. Although HRT in perimenopause resulted in decreased eosinophils in the HDM model, neutrophil levels and mucus production were unchanged. Mice receiving HRT in perimenopause also had significantly increased collagen production and inflammation associated with large and small airways, independent of HDM challenge. HRT given during perimenopause may be detrimental to lung responses, including increased AHR and decreased lung function, as well as increased tissue inflammation and airway remodeling.NEW & NOTEWORTHY Menopause-associated asthma is a subtype of asthma that is still largely unexplored and difficult to manage. Women experiencing menopause-associated asthma often have more severe exacerbations, higher rates of exacerbation, and most importantly, poor response to standard treatments. This study examined the impact of estrogen replacement given during the

perimenopause phase on lung inflammation and function after menopause. While decreasing eosinophil recruitment, estrogen replacement actually led to worse lung function and more airway remodeling.

Keywords: asthma; estrogen; lung function; menopause; remodeling.

Supplementary info

MeSH terms, Substances, Grants and fundingExpand

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14

Review

Drugs

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Mucus as a Treatable Trait in Chronic Airway Diseases

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Affiliations Expand

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Abstract

Chronic airway diseases, including asthma, chronic obstructive pulmonary disease, bronchiectasis, and cystic fibrosis, are increasingly recognized as heterogeneous disorders characterized by overlapping pathophysiological mechanisms. Among

these, abnormalities in mucus production, composition, and clearance have been identified as clinically significant contributors to symptoms, airflow limitation, exacerbations, and disease progression. Within the "treatable traits" framework, mucus-related abnormalities represent a distinct, modifiable phenotype that supports personalized management strategies. This narrative review explores mucus as a treatable trait across chronic airways diseases, integrating mechanistic insights with clinical assessment, biomarkers, and current and emerging therapeutic approaches. We discuss the role of mucus in disease phenotyping, its impact on morbidity, and the potential of targeted interventions to improve outcomes. Recognizing mucus as a treatable trait aligns with the principles of precision medicine and offers a pathway toward individualized therapy beyond traditional diagnostic labels.

Conflict of interest statement

Declarations. Conflicts of interest: The Authors have no conflicts of interest that are directly relevant to the content of this article. Mario Cazzola and Luigino Calzetta are Editorial Board members of Drugs. Mario Cazzola and Luigino Calzetta were not involved in the selection of peer reviewers for the manuscript, nor in any of the subsequent editorial decisions. Ethics approval: Not applicable. Consent to participate: Not applicable. Consent for publication: Not applicable. Availability of data and material: Not applicable. Code availability: Not applicable. Author contributions: All authors contributed to the manuscript. Original draft preparation: Mario Cazzola. Writing, review, and editing: Paola Rogliani, Josuel Ora, Luigino Calzetta, and Maria Gabriella Matera. All authors read and approved the final manuscript.

- 129 references
- 3 figures

Supplementary info

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15

Lancet Reg Health Eur

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema

plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors) showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated

with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma, and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi;

support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this work. S Principe reports grants from Innovative Medicines Initiative 2 Joint Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards (payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and

Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-chair of the Nordic severe asthma network; member of the steering committee of SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand, Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

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Ovarian hormones and obesity drive Th17-mediated airway inflammation through estrogen receptor-α signaling

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Affiliations Expand

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- PMCID: PMC13023106
- DOI: 10.1152/ajplung.00400.2025

Abstract

Obesity is a risk factor for increased prevalence and severity of asthma, particularly in females. As adults, females have an increased prevalence of asthma compared with males. Yet, the mechanisms remain unclear on how sex hormones and obesity increase airway inflammation. We hypothesize that estrogen signaling through estrogen receptor-alpha (ER-α) in T cells increased airway inflammation in the context of obesity. To test our hypothesis, we utilized a high-fat diet on female and male mice that underwent ovariectomy or gonadectomy or in *Esr1*$^{fl/fl}$ *X Cd4*$^{Cre+}$ male and female mice. As controls, mice in similar groups were fed normal chow. After 8-12 wk on diets, house dust mite sensitization and challenge occurred in all mice. Lungs and bronchoalveolar lavage fluid were harvested 24 h after the last challenge. Ovarian hormones and ER-α signaling in T cells increased eosinophils, neutrophils, and Th17-mediated airway inflammation in the lungs of obese female mice. In addition, using peripheral blood mononuclear cells (PBMCs) from a well-characterized cohort of asthmatic participants, we determined that women with asthma and obesity had increased Th17 cells compared with men with asthma and obesity. Our results show that ER-α signaling in T cells increases Th17-mediated airway inflammation in obese mice and that Th17 cells circulate at higher frequencies in women with asthma compared with men with asthma. Further research into the interplay between hormonal signaling and immune responses in asthma is essential for developing personalized treatments.NEW & NOTEWORTHY Estrogen receptor-alpha (ER-α) signaling increased obesity and allergen-induced airway inflammation in mice. In addition, women with obesity and asthma had increased circulating Th17 cells compared with men with obesity and asthma. These findings provide mechanistic insights into the intersection of obesity, sex hormones, and airway inflammation-underscoring the importance of personalized approaches to managing individuals with obesity and asthma.

Keywords: CD4+ T cells; airway inflammation; asthma; estrogen receptor-alpha; obesity.

Conflict of interest statement

Disclosures: No conflicts of interest are declared by the authors

Update of

- **Ovarian Hormones and Obesity Drive Th17-mediated Airway Inflammation through Estrogen Receptor Signaling.**

Henriquez-Pilier E, Cephus JY, Kuehnle S, Tannous E, Tomasello A, McKernan K, Peebles RS, Cahill KN, Rathmell JC, Newcomb DC.bioRxiv [Preprint]. 2025 Dec 5:2025.12.02.691857. doi: 10.64898/2025.12.02.691857.Update in: Am J Physiol Lung Cell Mol Physiol. 2026 Apr 1;330(4):L267-L273. doi: 10.1152/ajplung.00400.2025.PMID: 41573940 Free PMC article. Preprint.

- **21 references**
- **4 figures**

Supplementary info

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17

Review

Am J Emerg Med

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. 2026 Apr:102:206-208.

doi: 10.1016/j.ajem.2026.01.010. Epub 2026 Jan 10.

Practice changing articles: Efficacy of albuterol-budesonide inhaler compared with albuterol alone in mild asthma

Rachel E Bridwell [1], Ali Pourmand [2], Michael Gottlieb [3], Brit Long [4]

Affiliations Expand

- PMID: 41571468

- DOI: 10.1016/j.ajem.2026.01.010

No abstract available

Keywords: Albuterol; Asthma; Asthma exacerbation; Evidence-based medicine; Pulmonary; Respiratory; Steroids.

Conflict of interest statement

Declaration of competing interest None.

Supplementary info

Publication typesExpand

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18

Emerg Med J

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. 2026 Mar 30;43(4):266.

doi: 10.1136/emermed-2025-215141.

Correspondence on "Are acute asthma presentations to the emergency department an opportunity for optimising long-term management? A qualitative study on beliefs and behaviours of healthcare professionals" by Skene *et al*

Eva Tadros [1]

Affiliations Expand

- PMID: 41125336

- DOI: 10.1136/emermed-2025-215141

No abstract available

Keywords: Emergency Medicine; asthma; emergency department; emergency department management.

Conflict of interest statement

Competing interests: None declared.

Supplementary info

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Geroscience

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. 2026 Apr;48(2):2603-2616.

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Ambient air pollution exposure accelerates the occurrence of 78 non-communicable chronic diseases: an accelerated failure time analysis of a nationwide cohort

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- **PMCID: PMC12972164**
- **DOI: 10.1007/s11357-025-01806-3**

Abstract

Ambient air pollution is a well-established risk factor for chronic diseases, but its impact on disease onset age remains unclear. This study systematically evaluated the acceleration effect of air pollutants on the onset of 78 chronic diseases using over 900,000 hospitalization records from 396,000 UK Biobank participants. Both particulate matter and nitrogen oxides were associated with accelerated onset of 46

out of 78 diseases (9 cardiovascular diseases, 7 respiratory diseases, 14 psychological/neurological disorders, 3 digestive diseases, 2 cancers, and 11 other chronic diseases). Significant associations including those for common chronic diseases were observed. Each interquartile range (IQR) increase in $PM_{2.5}$ was strongly associated with a 0.93% (95% CI-0.86%, 1.00%) decrease in age at onset (AAO) of hypertension. Similarly, NO_x was associated with a 0.96% (95% CI-0.82%, 1.09%) decrease in AAO of COPD, PM_{10} with a 0.95% (95% CI-0.81%, 1.09%) decrease in AAO of diabetes, and NO_2 with a 0.88% (95% CI-0.77%, 1.00%) decrease in AAO of dementia. Notably, we observed that neurological/psychological disorders were observed to be mostly affected, including schizophrenia, dystonia, polyneuropathies, and migraine, with 1 ~ 3% reduction in the AAO. On a population level, $PM_{2.5}$ overexposure (exceeding the WHO guideline of 5 $\mu g/m^3$) accounted for 539,320 person-years of accelerated AAO across 78 chronic conditions, with hypertension (18.10%), asthma (6.03%), and diabetes (5.39%) contributing the most. This study provides the first evidence that air pollutants could accelerate onset of common chronic diseases. Findings highlight the urgent need for measures to improve air quality to slow progression of disease development.

Keywords: Accelerated failure time; Acceleration; Air pollution; Chronic diseases; Prospective cohort.

Conflict of interest statement

Declarations. Competing interests: The authors declare no competing interests. Disclosures: None.

- 44 references
- 5 figures

Supplementary info

MeSH terms, Substances

"rhinitis"[MeSH Terms] OR rhinitis[Text Word]

1

Review

Pediatr Allergy Immunol

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doi: 10.1111/pai.70329.

Update on the treatment of allergic rhinitis in children

Eleni Anastasiou [1], Paraskevi Xepapadaki [1], Julijana Asllani [2], Foteini Kapsouli [3], Aspasia Karavelia [4], Aikaterini Petta [1], Nikolaos G Papadopoulos [1 5]

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- PMID: 41918374

- DOI: 10.1111/pai.70329

Abstract

Allergic rhinitis (AR) is the most common chronic condition from childhood to adulthood and remains a major, often underestimated, contributor to impaired quality of life, school performance, and healthcare use. Although symptoms frequently begin early in life, pediatric AR is still underdiagnosed and inadequately treated, with important consequences for physical, emotional, and cognitive development. In this narrative review, we summarize recent evidence on the treatment of AR in children, highlighting age-specific challenges and evolving treatment concepts. International recommendations, particularly those from ARIA, support a stepwise, patient-centred approach focused on symptom control, safety, and long-term outcomes. Intranasal corticosteroids remain the cornerstone of therapy for moderate-to-severe disease, while second-generation antihistamines and intranasal antihistamines provide effective options for milder or intermittent symptoms. Fixed-dose intranasal steroid-antihistamine combinations are highly effective, providing options for children with more severe or uncontrolled AR. Allergen immunotherapy is the only disease-modifying intervention. Emerging strategies, including biologics and novel immunotherapy approaches, are promising but currently limited to specific contexts. Alongside pharmacotherapy, education, adherence support, and pragmatic environmental measures are essential to achieve sustained disease control. Ongoing gaps in the pediatric evidence-base highlight the need for age-adapted algorithms and long-term studies focusing on early intervention and disease modification.

Keywords: allergen immunotherapy; allergic rhinitis; children; pharmacotherapy; treatment of allergic rhinitis.

- 142 references

Supplementary info

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Lancet Reg Health Eur

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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Affiliations Expand

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Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and

their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors) showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were

all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma, and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca;

payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this work. S Principe reports grants from Innovative Medicines Initiative 2 Joint Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards (payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended

advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-chair of the Nordic severe asthma network; member of the steering committee of SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand, Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

- **37 references**
- **4 figures**

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Comparative Study

Otolaryngol Head Neck Surg

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. 2026 Apr;174(4):954-962.

doi: 10.1002/ohn.70156. Epub 2026 Feb 6.

Radiofrequency Ablation Versus Laser Neurolysis of the Posterior Nasal Nerve in Patients With Chronic Rhinitis

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Affiliations Expand

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Abstract

Objective: To compare the clinical outcomes of radiofrequency ablation of the intraturbinate segment of the posterior nasal nerve (RAPN) alone versus combined RAPN with CO_2 laser posterior nasal nerve neurolysis (RPN3) in patients with chronic rhinitis refractory to medical therapy.

Study design: Retrospective cohort study.

Setting: Single tertiary care center.

Methods: Adult patients with chronic rhinitis unresponsive to medical treatment for over 6 months who underwent either RAPN or RPN3 between February 2023 and May 2024 were included. Inclusion criteria were 24-hour reflective total nasal

symptom score (rTNSS) ≥ 3, rhinorrhea score ≥1, and NOSE score ≥11. Exclusion criteria included chronic rhinosinusitis, nasal polyps, and coagulopathy. Demographics and comorbidities were documented. Primary endpoints were changes in rTNSS, NOSE scores, and response rate (≥30% improvement from baseline) at 1-, 3-, and 6-months posttreatment.

Results: A total of 101 patients were analyzed (RPN3: 76; RAPN: 25). Baseline rTNSS and NOSE scores were comparable between the two groups (P = .144 and .414, respectively). Both groups demonstrated significant improvements in rTNSS and NOSE scores at all follow-up points (P < .001). From 1 to 6 months, RPN3 achieved significantly greater improvements compared to RAPN (P = .039-.045), particularly in the rhinorrhea and nasal itching sub-scores (P = .003-.039). Response rates for rTNSS ranged from 91% to 100% in the RPN3 group and 84% to 96% in the RAPN group.

Conclusion: Both RAPN and RPN3 are effective in treating chronic rhinitis unresponsive to medication. The addition of CO_2 laser posterior nasal nerve neurolysis (RPN3) provides superior symptomatic relief, particularly for rhinorrhea and nasal itching.

Keywords: allergy; posterior nasal nerve neurolysis; radiofrequency; rhinitis.

Conflict of interest statement

None.

- **31 references**
- **4 figures**

Supplementary info

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Allergol Int

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. 2026 Apr;75(2):319-326.

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Subclinical pulmonary abnormalities on chest CT in patients with eosinophilic chronic rhinosinusitis

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- DOI: 10.1016/j.alit.2025.12.001

Free article

Abstract

Background: Chronic rhinosinusitis (CRS) often coexists with lower respiratory tract diseases, and such cases tend to be more refractory. However, few studies have specifically investigated chest computed tomography (CT) findings in patients with CRS. This study analyzed chest CT findings in patients with CRS and investigated their associations with CRS phenotypes and clinical characteristics.

Methods: We retrospectively analyzed 278 patients with CRS who underwent preoperative chest CT prior to endoscopic sinus surgery. Patients were stratified based on the presence or absence of abnormal chest CT findings. Clinical parameters related to upper and lower airway inflammation, including CRS phenotypes, were compared between the groups. The prognosis for ECRS was evaluated using the systemic steroid dose as a longitudinal outcome variable in a linear mixed-effects model.

Results: Among the 278 patients, 174 were diagnosed with eosinophilic CRS (ECRS) and 104 with non-eosinophilic CRS (NECRS). Ground-glass attenuation (GGA) and bronchial wall thickening (BWT) were observed in 35 and 27 patients (12.6 %, 9.7 %), respectively. The frequencies of GGA and BWT were significantly higher in the ECRS group than in the NECRS group. Among patients with ECRS, those with GGA had significantly higher tissue eosinophil counts and Lund-Mackay CT scores, as well as significantly lower olfactory function. Steroid dose reduction was significantly slower in the GGA group.

Conclusions: The presence of GGA on chest CT in patients with CRS is associated with the CRS phenotype and greater disease severity, suggesting that chest imaging findings could serve as potential indicators of systemic disease burden in CRS.

Keywords: Bronchial wall thickening; Chest CT findings; Chronic rhinosinusitis; Ground-glass attenuation; United airways disease.

Conflict of interest statement

Conflict of interest TNa reports lecture fees a research grant from Sanofi. YM received an honorarium from Olympus. The rest of the authors have no conflict of interest.

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5

Immunology

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Microplastics in Allergic Rhinitis: Multimechanistic Drivers of Barrier Disruption and Immune Dysregulation

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Affiliations Expand

- **PMID: 41360054**

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No abstract available

Supplementary info

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6

Review

J Pharm Pract

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. 2026 Apr;39(2):117-139.

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Revisiting Rhinitis Medicamentosa: Examining the Evidence on Topical Nasal Decongestants

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Affiliations Expand

- PMID: 40580050

- DOI: 10.1177/08971900251350510

Abstract

Background: Over-the-counter topical nasal decongestants are effective and well-tolerated treatments for the temporary relief of nasal congestion, a symptom that can impair quality of life. Their duration of use is limited owing to potential for rhinitis medicamentosa (RM) or rebound congestion (RC), despite uncertainties around the clinical occurrence or onset of these phenomena. Objective: To investigate the clinical occurrence and onset of RM, RC or tolerance with topical nasal decongestants to inform evidence-based recommendation practices for pharmacists and ensure patients do not forego potentially beneficial treatments. Methods: A literature search was conducted with ProQuest to identify and synthesize evidence on RM, RC or tolerance with nasal decongestant sprays or drops. A respiratory specialist and community pharmacist provided clinical perspectives. Results: Eighteen articles were assessed, reporting 13 studies with oxymetazoline, five studies with xylometazoline. There was no evidence of RM or RC after 7 days with oxymetazoline (up to 400 µg total daily dose) or up to 10-days with xylometazoline (840 µg total daily dose). Well-designed studies suggested no occurrence of RM, RC or tolerance with up to 4 weeks of oxymetazoline. No studies evaluating naphazoline, phenylephrine or ephedrine were identified. Conclusion: Oxymetazoline and xylometazoline are highly effective at rapidly improving nasal congestion and have well-established safety profiles. Well-designed studies yielded no evidence of RM, RC or tolerance when used short-term at commonly recommended dosing and frequency. Since some patients may exceed

the duration of use in the label, pharmacists can play a vital role in counseling patients on proper intranasal decongestant use and treatment duration.

Keywords: intranasal decongestant; nasal congestion; oxymetazoline; rebound congestion; xylometazoline.

Plain language summary

A blocked or stuffy nose is a common symptom of hayfever and colds that can make daily activities and sleeping difficult. Over-the-counter nasal decongestant sprays or drops work within a few minutes to ease a blocked nose and provide symptom relief. Some studies from around 30 years ago suggested that, when these medicines were used regularly for weeks or months, they might cause a condition called rhinitis medicamentosa, also known as rebound congestion (RC). This is where the lining of the nose swells leading to the nose feeling blocked when the medicines are stopped. We looked for other studies that investigated this effect to see when it might occur and if people developed tolerance to the medicines, meaning they needed to take more for them to work. We included information to help pharmacists treat patients with RC. We found 18 studies that looked at medicines containing xylometazoline or oxymetazoline. When people used these medicines for 7 days (oxymetazoline) or up to 10 days (xylometazoline) there was no evidence of RC. Some well-designed studies found no evidence for RC or tolerance with up to 4-weeks of oxymetazoline use. No studies were found for other medicines in nasal decongestants. When people use xylometazoline- or oxymetazoline-containing medicines as described in the patient information leaflet there is no evidence for RC or tolerance. Pharmacists can play an important role in making sure that patients know how to use the medicines properly, at the correct dose and for no longer than stated in the instructions.

Conflict of interest statement

Declaration of Conflicting InterestsThe author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: MH, EM and MBF are employees of Haleon. GV is a former employee of Haleon.

- **Cited by 1 article**

Supplementary info

Publication types, MeSH terms, SubstancesExpand

chronic cough

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ERJ Open Res

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. 2026 Mar 30;12(2):00342-2025.

doi: 10.1183/23120541.00342-2025. eCollection 2026 Mar.

The therapeutic effects of labetalol in chronic stress- and immuno-agonist-induced cough hypersensitivity and lung inflammation involve reducing interferon-γ-producing T lymphocytes

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Abstract

Background: Psychological stress correlates with chronic cough, which is often triggered by respiratory viruses. Chronic stress promotes sympathetic nerves to release norepinephrine and neuropeptide Y. Labetalol blocks sympathetic adrenergic receptors. Norepinephrine and neuropeptide Y can upregulate interferon-γ (IFN-γ)-producing lymphocytes and increase IFN-γ levels *in vivo*. IFN-γ-producing lymphocytes and IFN-γ contribute to chronic stress-induced colonic inflammation and influenza virus-caused pulmonary inflammation. IFN-γ can also enhance cough sensitivity. Administration of haemagglutinin and R848 (immuno-agonists) can induce a virus-infection-like illness in animal models.

Methods: We investigated the effects and underlying mechanisms of labetalol on chronic stress and immuno-agonist-induced cough hypersensitivity and lung inflammation.

Results: Chronic stress and immuno-agonists induced cough hypersensitivity and pulmonary inflammation in guinea pigs, which were alleviated by labetalol. In mice, chronic stress triggered the release of norepinephrine and neuropeptide Y, leading to IFN-γ-producing T lymphocytosis and pulmonary inflammation. Additionally, chronic stress and immuno-agonists increased IFN-γ in the lung of guinea pigs and mice. Labetalol effectively reduced chronic stress-induced and immuno-agonist-induced IFN-γ-producing T lymphocytosis and lung inflammation in mice. Labetalol dose-dependently inhibited the increase in IFN-γ-producing T lymphocytes induced by immuno-agonists and norepinephrine *in vitro*.

Conclusions: Chronic stress can stimulate the release of sympathetic neurotransmitters, which may increase IFN-γ-producing T lymphocytes and lead to cough hypersensitivity and pulmonary inflammation by activating the adrenergic receptors. Immuno-agonists may increase IFN-γ-producing T lymphocytes, exacerbating chronic stress-induced cough hypersensitivity and pulmonary inflammation. The therapeutic effects of labetalol in chronic stress and immuno-

agonist-induced cough hypersensitivity and pulmonary inflammation possibly occur by reducing IFN-γ-producing T lymphocytes.

Conflict of interest statement

Conflict of interest: The authors declare that there are no conflicts of interest.

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Mayo Clin Proc Innov Qual Outcomes

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. 2026 Mar 2;10(2):100707.

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When Refractory Is Not the End: Redefining Chronic Cough Through Tertiary Center Evaluation

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- PMCID: PMC12969109
- DOI: 10.1016/j.mayocpiqo.2026.100707

Abstract

Objective: To describe the clinical outcomes of refractory chronic cough (RCC) diagnosis and management at a tertiary care chronic cough clinic.

Patients and methods: This is a prospective study of all eligible patients referred to the Mayo Clinic Chronic Cough Clinic between October 6, 2020 and April 9, 2021. All adult patients aged 18 years or older with cough persisting beyond 8 weeks in accordance with the American College of Chest Physicians definition were

screened. Only participants with research authorization and consent were included in this study.

Results: A total of 278 patients were screened; 100 patients were enrolled in this study. On the initial visit, 56 patients who were considered to have RCC were invited for a 3-month follow-up survey. Of those 56 patients, electronic health record review clarified that 35 (62%) had etiology-confirmed chronic cough 21 (37%) patients had unexplained chronic cough.

Conclusion: This study shows that RCC needs further attentiveness toward diagnosis and trials of therapy to truly ascertain those who have unexplained chronic cough such as at specialized chronic cough centers.

Conflict of interest statement

The authors report no competing interests.

- **48 references**
- **1 figure**

Full text links

Cite

3

Review

Respir Med

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doi: 10.1016/j.rmed.2026.108729. Epub 2026 Feb 23.

From symptom to disease: Reclassifying chronic cough through the treatable traits framework?

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- PMID: 41740803

- DOI: 10.1016/j.rmed.2026.108729

Abstract

Chronic cough has traditionally been defined by its duration and regarded mainly as a symptom of underlying diseases. However, growing evidence suggests that in a substantial proportion of individuals, chronic cough persists despite adequate treatment of comorbid conditions and may reflect an underlying neuro-pathophysiological process, particularly cough reflex hypersensitivity. The treatable traits framework offers a structured and personalized approach by identifying clinically relevant, measurable, and modifiable traits to guide therapy. Recognition of cough hypersensitivity as a key treatable trait has been pivotal in shifting the perspective toward chronic cough as a distinct disease entity. This review summarizes the clinical relevance, diagnostic tools, therapeutic strategies, and supporting evidence for treatable traits in chronic cough and proposes a stratified application of the treatable traits model, prioritizing actionable traits based on evidence and resources. Beyond guiding treatment, this framework serves as a lens through which to interpret the multifactorial nature of chronic cough. We advocate for moving away from potentially misleading terminology such as "idiopathic", "unexplained", or "refractory". Instead, we propose reclassifying chronic cough based on identifiable traits. This shift supports the recognition of chronic cough as a distinct disease entity defined by specific mechanisms (e.g., neural dysregulation), as well as a symptom secondary to other conditions. Such an evolution is essential to align clinical practice and nosology with current scientific understanding.

Keywords: Chronic cough; Classification; Treatable traits.

Conflict of interest statement

Declaration of competing interest AO is supported by FCT - Fundaçãao para a Ciência e Tecnologia, I.P. by project reference UID 4501- Instituto de Biomedicina - Universidade de Aveiro. HC reports grant from the Basic Science Research Program of the Korean Ministry of Education (grant no. 2021R1I1A3052416); consulting fees from Gilead, Boehringer Ingelheim, and Abbott; and lecture fees from Kolong, Boryung, Abbott, Otsuka, and Handok. WJS declares grants from Merck Sharp & Dohme Corp., Daewoong Pharmaceutical, and AstraZeneca, consulting fees from Merck, Bellus, AstraZeneca, Shionogi, and GSK, and lecture fees from Thermo Fischer/Immunotek, Celltrion, Merck, AstraZeneca, GSK, Sanofi, and Novartis. Other authors have none to declare.

Supplementary info

Publication types, MeSH termsExpand

Full text links

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Lancet Reg Health Eur

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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- PMCID: PMC12906202

- DOI: 10.1016/j.lanepe.2026.101600

Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and

their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors) showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were

all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma, and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca;

payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this work. S Principe reports grants from Innovative Medicines Initiative 2 Joint Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards (payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended

advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-chair of the Nordic severe asthma network; member of the steering committee of SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand, Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

- **37 references**
- **4 figures**

"bronchiectasis"[MeSH Terms] OR bronchiectasis[Text Word]

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J Mol Med (Berl)

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. 2026 Apr 1;104(1):59.

doi: 10.1007/s00109-026-02662-0.

Neutrophil-related gene expression profile is associated with future paediatric bronchiectasis exacerbations

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- PMID: 41917171

- DOI: 10.1007/s00109-026-02662-0

Abstract

Acute respiratory exacerbations in bronchiectasis are important as they impair quality of life and are associated with accelerated lung function decline. Yet, no validated methods exist to identify children at increased risk of exacerbations. We therefore determined if peripheral blood gene expression (GE) signatures can identify those at risk of an impending exacerbation. Thirty-one children with bronchiectasis had RNA extracted from peripheral blood collected whilst they were clinically stable, with 22 having an exacerbation during the next 3 months. Microarray assays using the HumanHT-12 v4.0 Expression BeadChip identified differentially expressed genes (p value ≤ 0.05, fold change > 1.5). The top targets were verified using real-time quantitative polymerase chain reaction (rt-qPCR) assays, and receiver operating characteristics and area under the curve (AUC) were assessed. Functional analysis of these genes was performed using Ingenuity Pathway Analysis. Overall, 647 entities were significantly dysregulated (p < 0.05) in the exacerbation group (n = 22), and pathway analysis identified neutrophil degranulation as the dominant affected pathway, which was also significantly inhibited (p < 0.001). Forty entities (32 genes) were associated with a future exacerbation (p ≤ 0.05, fold change ≥ 1.5) and six genes (ANXA3, ALAS2, DEFA1, ALPL, SNCA, PROK2) were verified using RT-qPCR (all p < 0.04) as the most discriminatory. DEFA1 and ANXA3 had the highest AUC (0.92, 95% confidence

interval [CI] 0.82-1.00, and 0.87, 95% CI 0.73-1.00, respectively). We identified neutrophil-associated genes from peripheral blood that could be potential biomarkers for children with bronchiectasis at increased risk of exacerbations during the next 3 months. These GE signatures warrant further investigation and validation in larger, independent cohorts. KEY MESSAGES: Exacerbations in paediatric bronchiectasis are important. Peripheral blood gene expression may help identify children at risk of exacerbations. Six neutrophil-associated genes were associated with a future exacerbation. Identifying predictive gene expression signatures warrants further investigation.

Keywords: Bronchiectasis; Gene expression; Microarray; Paediatrics.

Conflict of interest statement

Declarations. Ethics approval: All parents/guardians provided written informed consent to participate in the BEST studies and consented to the storage of samples for future research use. Competing interests: The authors declare no competing interests. Clinical trial number: Not applicable.

- 48 references

Supplementary info

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Randomized Controlled Trial

Pediatr Pulmonol

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. 2026 Apr;61(4):e71602.

doi: 10.1002/ppul.71602.

Does Addition of Oscillatory Positive Expiratory Pressure (OPEP) Device to a Chest Physiotherapy Program Provide Further Benefits in Children With Bronchiectasis?: A Randomized Trial

Melih Zeren [1], Meral Barlik [2], Beyza Nur Caglar Tosun [3], Esen Demir [4], Figen Gulen [4]

Affiliations Expand

- PMID: 41914274

- DOI: 10.1002/ppul.71602

Abstract

Background: Airway clearance is major part of care for children with bronchiectasis (BE) and oscillatory positive pressure (OPEP) devices are widely used, yet their added value beyond a multicomponent chest physiotherapy (CPT) program remains unclear and device access often entails out of pocket costs. Aim of this study was to evaluate whether adding an OPEP device to a multicomponent CPT program provides additional benefits in exercise capacity and dynamic ventilatory responses in children with BE.

Methods: A randomized trial involving 42 children with non-cystic fibrosis BE was conducted. Children were allocated to either CPT or CPT + OPEP group. Both groups trained at home twice daily for 8 weeks. 6-min walk test (6MWT) with Spiropalm® device, spirometry, quadriceps strength measurement and Leicester Cough Questionnaire (LCQ) were applied at baseline and after training.

Results: 6MWT distance, change in inspiratory capacity during exertion (ΔIC), lowest SpO_2, the time spent with a drop of ≥ %4 in SpO_2 [T ($dSpO_2$ ≥ %4)], FVC%, FEV_1%, PEF%, significantly improved in both groups, with no between-groups difference. Program adherence (%) was significantly higher in CPT + OPEP group (85 ± 15) compared to CPT group (74 ± 18) ($p < 0.05$).

Conclusion: In children with BE, a multicomponent CPT program improved exercise capacity, dynamic ventilatory responses and spirometry, while adding an OPEP device primarily increased adherence and conferred no additional benefit. The decision to add an OPEP device may be individualized to address engagement or feasibility rather than further clinical benefit.

Trial registration: The study was prospectively registered to ClinicalTrials. gov on September 1, 2021, under the registration number NCT05034900.

Keywords: airway clearance; bronchiectasis; chest physiotherapy; children; oscillatory positive expiratory pressure.

- 31 references

Supplementary info

Publication types, MeSH terms, Associated data, Grants and fundingExpand

Full text links

UNIMORE

3

Review

J Sleep Res

. 2026 Mar 29:e70333.

doi: 10.1111/jsr.70333. Online ahead of print.

Obstructive Sleep Apnea in Bronchiectasis: Prevalence, Risk Factors and Clinical Implications-A Systematic Review and Meta-Analysis

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- PMID: 41905893

- DOI: 10.1111/jsr.70333

Abstract

Obstructive sleep apnea (OSA) has been poorly characterised in patients with bronchiectasis despite potential bidirectional clinical impacts. This systematic review and meta-analysis determined the prevalence of OSA confirmed by polysomnography in cystic fibrosis and non-cystic fibrosis bronchiectasis patients. A comprehensive search of MEDLINE, EMBASE and Cochrane databases through September 2025 identified observational studies reporting polysomnography-confirmed OSA prevalence in adult bronchiectasis patients. Five studies encompassing 256 patients met inclusion criteria, with data extracted and quality assessed using the Newcastle-Ottawa Scale. The pooled obstructive sleep apnea prevalence was 50.5% (95% confidence interval: 42.3%-58.9%) with low heterogeneity, ranging from 40.4% to 64.3% in cystic fibrosis patients and 40.8% to 55.8% in non-cystic fibrosis bronchiectasis patients. Traditional risk factors including obesity and excessive daytime sleepiness showed weak associations, with a mean body mass index of 23.8-24.2 kg/m^2. Male gender, longer disease duration, and corticosteroid use emerged as significant risk factors. Nocturnal hypoxemia independent of obstructive sleep apnea occurred in 16.7% of patients. Only 41% of diagnosed patients received positive airway pressure therapy. OSA affects approximately half of all bronchiectasis patients regardless of aetiology, substantially exceeding general population rates. The clinical presentation differs

from classical OSA, with weaker associations with obesity and daytime sleepiness, suggesting distinct pathophysiological mechanisms. Systematic screening should be incorporated into bronchiectasis management, particularly for male patients with longer disease duration or corticosteroid use.

Keywords: bronchiectasis; meta-analysis; obstructive sleep apnea; polysomnography; prevalence; systematic review.

- 30 references

Supplementary info

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4

Review

Pediatr Pulmonol

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. 2026 Apr;61(4):e71577.

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The Diagnosis of Primary Ciliary Dyskinesia: Putting The European Respiratory/American Thoracic Guideline Into Practice

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Abstract

Primary ciliary dyskinesia (PCD) is a rare inherited disease which is characterised by progressive lung disease, chronic rhinosinusitis, repeated middle ear infections, laterality defects, and reduced fertility. An early diagnosis is critical to reduce morbidity, however diagnosis is often delayed due to the heterogeneity in clinical presentation. Diagnosis relies on multiple tests, and the American Thoracic (ATS) and European Respiratory (ERS) societies have previously developed separate diagnostic guidelines. Both differed in recommendations and approach. The recently published joint ERS/ATS guidelines for the diagnosis of PCD is the first evidence-based guidelines that unifies the approach recommendations between both societies. These guidelines were formulated by a task force (TF) comprised of experts in the field, and were guided by a systematic reviews and GRADE (Grading of Recommendations, Assessment, Development and Evaluation) approach. The TF formulated three 'Patients, Intervention, Comparison, Outcomes' (PICO) questions to determine the accuracies of (1) nasal nitric oxide (nNO) (2) high-speed video microscopy (HSVM) and 3) immunofluorescence (IF) when compared to a reference test of either transmission electron microscopy (TEM) and/or genetics. There were also three narrative questions which sought to determine (1) what clinical presentation would support a clinician to refer a patient for PCD diagnostic testing (2) what additional diagnostic tests could be useful and (3) how to overcome PCD diagnostic challenges in resource limited settings. This review presents example cases that highlight the recommendations of the clinical practice guidelines.

Conflict of interest statement

Amelia Shoemark reports grants from AstraZeneca and Lifearc; consultancy fees from Spirovant, Translate Bio and ReCode Therapeutics; payment or honoraria for lectures, presentations, manuscript writing or educational events from Translate Bio, Ethris and Insmed; a leadership role as Chair BEAT-PCD ERS CRC; and involvement in European Respiratory Society Clinical Research Collaborations (EMBARC and AMR Lung). Amjad Horani reports leadership roles with the PCD Foundation and PCD Research. Katharine Harman has no potential conflicts of interest to disclose.

- **32 references**

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Review

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Bronchiectasis: Past insights, present clinical evidence, and future research pathways

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Free article

Abstract

Bronchiectasis is a chronic disease characterized by irreversible airway dilation, persistent inflammation, and recurrent infections. Its global prevalence has risen significantly, now surpassing 1400 cases per 100,000 in certain regions, likely due to increased awareness over the last several decades. Bronchiectasis exhibits wide heterogeneity in etiology and clinical phenotype. Advances in imaging, microbiology, and biomarker profiling have refined diagnostic accuracy and revealed distinct endotypes, including those characterized by neutrophilic and eosinophilic inflammation. Innovations in therapy, ranging from airway clearance and inhaled antibiotics to emerging biologics and neutrophil-targeted therapies, underscore a shift toward individualized treatment. In this review, we provide a comprehensive and up-to-date discussion of the pathophysiological mechanisms, etiologies, and evolving management strategies in bronchiectasis.

Keywords: Airway infection; Bronchiectasis; DPP1 inhibitors; Exacerbations; Neutrophilic inflammation; Precision medicine; Pseudomonas aeruginosa; Type 2 inflammation.

Conflict of interest statement

Declaration of competing interest All the authors of this review article report NO financial or personal relationships with other people or organizations that could inappropriately influence or bias our work. Including all the following potential

competing interests: Employment. Consultancies. Stock ownership. Honoraria. Paid expert testimony. Patent applications or registrations. Grants or any other funding Affiliation with the journal as an Editor or Advisory Board Member.

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Review

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Mucus as a Treatable Trait in Chronic Airway Diseases

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Abstract

Chronic airway diseases, including asthma, chronic obstructive pulmonary disease, bronchiectasis, and cystic fibrosis, are increasingly recognized as heterogeneous disorders characterized by overlapping pathophysiological mechanisms. Among these, abnormalities in mucus production, composition, and clearance have been identified as clinically significant contributors to symptoms, airflow limitation,

exacerbations, and disease progression. Within the "treatable traits" framework, mucus-related abnormalities represent a distinct, modifiable phenotype that supports personalized management strategies. This narrative review explores mucus as a treatable trait across chronic airways diseases, integrating mechanistic insights with clinical assessment, biomarkers, and current and emerging therapeutic approaches. We discuss the role of mucus in disease phenotyping, its impact on morbidity, and the potential of targeted interventions to improve outcomes. Recognizing mucus as a treatable trait aligns with the principles of precision medicine and offers a pathway toward individualized therapy beyond traditional diagnostic labels.

Conflict of interest statement

Declarations. Conflicts of interest: The Authors have no conflicts of interest that are directly relevant to the content of this article. Mario Cazzola and Luigino Calzetta are Editorial Board members of Drugs. Mario Cazzola and Luigino Calzetta were not involved in the selection of peer reviewers for the manuscript, nor in any of the subsequent editorial decisions. Ethics approval: Not applicable. Consent to participate: Not applicable. Consent for publication: Not applicable. Availability of data and material: Not applicable. Code availability: Not applicable. Author contributions: All authors contributed to the manuscript. Original draft preparation: Mario Cazzola. Writing, review, and editing: Paola Rogliani, Josuel Ora, Luigino Calzetta, and Maria Gabriella Matera. All authors read and approved the final manuscript.

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Multimorbidity phenotypes and associated characteristics in severe asthma: an observational study of European severe asthma registries

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Abstract

Background: The phenotypic nature of multimorbidity in severe asthma is poorly understood. Our aims in this study were to define multimorbidity phenotypes and their characteristics in severe asthma across Europe by identifying and characterising co-aggregation of comorbidities.

Methods: Cross-sectional patient data were analysed from the pan-European Severe Heterogenous Asthma Research Collaboration: Patient Centred (SHARP) Central database of national severe asthma registries. Patients were grouped by four European regions (North, South, East, and West). Hierarchical clustering of comorbidities was applied to characterise the correlation structure of the ten commonest comorbidities within these geographical regions. Subsequent multimorbidity phenotypes (MMP) and their clinical features were then defined.

Findings: Data were available for 2690 severe asthma patients and 23 comorbidities from 11 countries. Three comorbidity clusters were consistently seen across the four European regions: 1) osteoporosis plus steroid-induced weight gain, 2) eczema plus rhinitis, and 3) chronic sinusitis plus nasal polyps. Four further comorbidities (obesity, bronchiectasis, gastro-oesophageal reflux disease, psychological factors) showed variable clustering. Multimorbidity was ubiquitous. Patients were assigned

multimorbidity phenotypes (MMP) according to comorbidity cluster alignment. MMP sn (sinonasal-associated) and MMP u (no specific cluster alignment) were commonest. MMP ster (steroid-associated multimorbidity) had highest maintenance oral steroid (m-OCS) use, and Body Mass Index, plus worst lung function, asthma control, and asthma exacerbation frequency. MMP max (maximal multimorbidity) showed high prevalence of variably assigned comorbidities, higher m-OCS and biologic treatment needs.

Interpretation: Multimorbidity is common in severe asthma and can be classified into replicable novel phenotypes with characteristic clinical traits and outcomes. Recognising these phenotypes can guide better care of the 'whole patient' with severe asthma. Future clinical guidance should promote such understanding in order to support delivery of more effective personalised asthma care.

Funding: European Respiratory Society, pharmaceutical industry partners (Sanofi, TEVA, Novartis, GlaxoSmithKline, Chiesi).

Keywords: Cluster; Multimorbidity; Phenotype; Severe asthma.

Conflict of interest statement

RJ Kurukulaaratchy co-holds a methods patent outside the submitted work on the cellular profiles of Tissue Resident Memory T-cells and their use in asthma. B Ainsworth is a member of the UK Taskforce for Lung Health, has received honoraria for educational talks from AstraZeneca, and sits on advisory boards for the Medito Foundation and earGym. All of these are unrelated to this work. R Djukanovic is a Past co-Chair of the European Respiratory Society's Clinical collaboration on severe asthma ∗SHARP. He is also a co-founder and shareholder of and consultant to Synairgen, has received funding for lectures from GlaxoSmithKline, and has been on advisory boards of GlaxoSmithKline, Celltrion, ALK Abello and ZenasBio, all unrelated to this work. S Hromis reports speakers fees from AstraZeneca, Berlin Chemie Menarini, Takeda, Providens, Amicus Therapeutics; support for attending meetings from AstraZeneca, Chiesi (Providens), Hemofarm and payment for advisory boards from AstraZeneca, BerinChemie Menarini, Providens. These were all unrelated to this work. HM Haitchi co-holds a method patent on Anti-ADAM33 oligonucleotides and related methods, which is unrelated to the current work. I Adcock reports institutional grants from GlaxoSmithKline, MRC, Sanofi, and EPSRC; consulting fees from GlaxoSmithKline and Kinaset; funding for lectures from AstraZeneca and GlaxoSmithKline, and has served on advisory boards for GlaxoSmithKline, Sanofi, Chiesi and Kinaset. All unrelated to this work. M Florin has received funding for lectures with presentations from AstraZeneca, Sanofi, Pfizer, and Angelini, unrelated to this work. B Gemicioglu is Chair of the Turkish Board of Pulmonology and GARD Turkey Coordinator (unpaid); reports institutional Honoria for lectures from Abdi Ibrahim, AstraZeneca, Daeva, and GlaxoSmithKline; support for attending meetings from AstraZeneca and GlaxoSmithKline and participation on advisory boards of GlaxoSmithKline. Unrelated to this work. B Dahlén reports personal Honoria for lectures from AstraZeneca, GlaxoSmithKline, and Sanofi, as well as payment for participation on advisory boards of Affibody and is associated with the Swedish Medical Products Agency, all unrelated to this work. P Kuna reports grants for investigator led academic study from AstraZeneca, payment for lectures from Adamed, GlaxoSmithKline, AstraZeneca, Glenmark, Teva, Polpharma,

and Berlin Chemie Menarini, and conference travel grants from AstraZeneca and Berlin Chemie Menarini, all unrelated to this work. E Damadoglu is Chair of the Turkish Thoracic Society Asthma Section (unpaid), unrelated to this work. M Caminati reports consulting fees from AstraZeneca and Sanofi and speaker fees from GlaxoSmithKline, AstraZeneca, and Sanofi, all unrelated to this work. AT Brinke reports research grants from AstraZeneca, GlaxoSmithKline and TEVA, consulting fees/advisory board from AstraZeneca, GlaxoSmithKline, Novartis and TEVA and payment for lectures from AstraZeneca, GlaxoSmithKline, Novartis, TEVA and SanofiGenzyme. all unrelated to this work. A Egesten participated on advisory boards for BioCryst, and CSL-Behring, unrelated to this work. C Chaves Loureiro received grants for investigator led study from AstraZeneca and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, and Sanofi; payment for lecture presentations from GlaxoSmithKline, AstraZeneca, TEVA, and Sanofi; support for attending conferences from AstraZeneca, Sanofi and Vivisol, and served on advisory boards for AstraZeneca, GlaxoSmithKline, Sanofi, and Menarini. All unrelated to this work. GAML Costanzo received support for conference attendance from Chiesi, Sanofi, GlaxoSmithKline, and AstraZeneca, unrelated to this work. G Roberts is the Past president of the British Society of Allergy and Clinical Immunology and reports grants from the EU 3TR Consortium and consulting fees from AstraZeneca, not related to this work. G Brusselle is President of the Belgian Respiratory Society (BeRS) and reports speaker fees from AstraZeneca, Boehringer-Ingelheim, Chiesi, GSK, Novartis, Merk Sharp & Dohme and Sanofi Regeneron. All unrelated to this work. J Varkonyi-Sepp reports research grants from GlaxoSmithKline, separate to this work. K Bieksiene received lecture fees from AstraZeneca, Berlin Chemie, and Chiesi and support for conference and scientific meeting attendance from Chiesi, AstraZeneca, and Berlin Chemie, all not related to this work. M Zappa reports payment for lectures from AstraZeneca, unrelated to this work. O Goksel received grants for investigator led study from AstraZeneca, GlaxoSmithKline, and Sanofi as well as payment for lectures from GlaxoSmithKline not related to this work. P Kopač reports consulting fees from AstraZeneca, Berlin-Chemie Menarini, Medis, Swixx Biopharma; speaker fees from AstraZeneca, Berlin-Chemie Menarini, Medis, and Swixx Biopharma; and support for attending conferences from AstraZeneca, and Berlin-Chemie Menarini, all unrelated to this work. R Chaudhuri received grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, Teva, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi, and GlaxoSmithKline; and participation on advisory boards of GlaxoSmithKline, AstraZeneca, and Celltrion. All unrelated to this work. S Dimic-Janjic reports payment for lectures from Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, Takeda, and Providens and participation on advisory boards of Boehringer Ingelheim, AstraZeneca, Berlin Chemie Menarini, and Takeda. All unrelated to this work. C Porsbjerg reports institutional grants from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; consulting fees and payment for lectures (paid to institution and personal Honoria) from AstraZeneca, GlaxoSmithKline, Novartis, TEVA, Sanofi, Chiesi, and ALK; and participation on advisory boards of AstraZeneca, Novartis, TEVA, Sanofi, ALK. All unrelated to this work. A Spanevello reports grants from GlaxoSmithKline, Sanofi, and Menarini; consulting fees from Chiesi and Sanofi; payment for lectures from GlaxoSmithKline, AstraZeneca, Chiesi, and Sanofi; support for attending conferences from Chiesi, Sanofi and GlaxoSmithKline; and sits on advisory board meetings of GlaxoSmithKline and Sanofi. All unrelated to this work. S Principe reports grants from Innovative Medicines Initiative 2 Joint

Undertaking (JU) and the European Union's HORIZON Research and Innovation programme, and is associated with the Young Investigator Board—Netherlands Respiratory Society (unpaid) All unrelated to this work. V Kalinauskaite-Zukauske reports payment for lectures from AstraZeneca, Chiesi, Sanofi, and Medison as well as support for attending conferences and scientific meetings from Chiesi, AstraZeneca, and Medison. All unrelated to this work. V Yasinska reports grants for investigator led study from AstraZeneca; payment for lectures from GlaxoSmithKline, AstraZeneca, and Sanofi; and participation on Advisory boards (payment to institution) of GlaxoSmithKline and AstraZeneca, all unrelated to this work. Z Csoma reports grants for investigator led study from AstraZeneca; payment for lectures from AstraZeneca, Chiesi, Sanofi, and Belin Chemie; support for attending scientific meetings from Orion Pharma and participation on Chiesi and AstraZeneca advisory boards, all unrelated to this work. AM Pereira received support for attending scientific meetings from Menarini and Roxall, not related to this work. A Štajduhar received support for attending scientific meetings from AstraZeneca unrelated to this work. D Paróczai reports personal university research grants from the University Research Fellowship Program (EKÖP) of the Ministry for Culture and Innovation from the source of the National Research, Development and Innovation Fund (EKÖP-24-4–SZTE-376) and ÚNKP-23-4-New National Excellence Program of the Ministry for Culture and Innovation, the National Research, Development, and Innovation Fund (ÚNKP-23-4-SZTE-380), unrelated to this work. E Heffler reports grants from Chiesi paid to his institution; personal consultancy fees from Chiesi, GlaxoSmithKline, Sanofi, AstraZeneca, Regeneron, Almirall, Apogee Therapeutics, Celltrion Healthcare, and Bosch; payment for lectures from Chiesi, GlaxoSmithKline, AstraZeneca, Sanofi, Regeneron, Novartis, Lofarma, and Firma; and participation on GlaxoSmithKline, AstraZeneca, and Sanofi advisory boards. All unrelated to this work. R Hou participates on advisory boards for the Medical Research council, ECNP and AAIC NPI (programme chair). All unrelated to this work. LG Heaney has received institutional project grant funding from AstraZeneca and GlaxoSmithKline and has been involved in asthma clinical trials with GlaxoSmithKline, AstraZeneca and Roche/Genentech for which his institution was renumerated. He has given lectures supported by AstraZeneca, Sanofi, Circassia, GlaxoSmithKline, and Teva; received travel support to attend international respiratory meetings from AstraZeneca, Sanofi, Teva and GSK; and attended advisory boards/lectures of GlaxoSmithKline, AstraZeneca, and Celltrion. Unrelated to this work. A Bourdin reports grants from AstraZeneca, Boeringher Ingelheim, and GlaxoSmithKline; consulting fees from AstraZeneca, GlaxoSmithKline, Sanofi, Chiesi, Celltrion, Boeringher Ingelheim, and Novartis; speaker fees from Sanofi Regeneron, AstraZeneca, GlaxoSmithKline, Boeringher Ingelheim, and Novartis; support for attending scientific meetings from AstraZeneca, and Sanofi; and participates on the AB science advisory board, all unrelated to this work. I Horvath has received personal grants from AstraZeneca and Boeringher Ingelheim; consulting fees from AstraZeneca, Boeringher Ingelheim, Sanofi, and Chiesi; payment for lectures from Sanofi, AstraZeneca, Chiesi, Berlin-Chemie Menarini, and Boeringher Ingelheim; and travel fees from AstraZeneca, Chiesi, Boeringher Ingelheim, and MSD, all unrelated to this work. S Popović-Grle has received personal consultancy fees from AstraZeneca, Pliva Hrvatska, and Providens as well as payment for lectures from AstraZeneca, Berlin-Chemie, Pliva Hrvatska, and Providens, all unrelated to this work. A Bossios is Head of Assembly 5 (Airway diseases, asthma, COPD, and chronic cough), European Respiratory Society; co-chair of the Nordic severe asthma network; member of the steering committee of

SHARP, ERS severe asthma Clinical Research Collaboration; member of the steering committee of the Swedish National Airway Register. He reports a grant from AstraZeneca as well as Honoraria and lecture fees from Chiesi, GlaxoSmithKline, and AstraZeneca, paid to institution outside of the submitted work. All unrelated to this work. D Lúdvíksdóttir reports Honoraria for lectures from GlaxoSmithKline, Sanofi and Chiesi; and travel fees from Chiesi, all unrelated to this work. M Bonini has received research grants and advisory board/speaker fees from AstraZeneca, Boehringer Ingelheim, Chiesi, Grifols, GlaxoSmithKline, Lallemand, Lusofarmaco. Menarini, Omron, and Sanofi, all unrelated to this work. F Schleich received grants from AstraZeneca, Sanofi, GlaxoSmithKline and Chiesi to give lectures and perform research activities. All unrelated to this work. JK Sont received Institutional Grants from: AstraZeneca, Dutch RAPSODI severe asthma registry, Care Research Netherlands (ZonMW). All unrelated to this work. M Hyland declares grants from GlaxoSmithKline and from AstraZeneca outside the submitted work. GW Canonica reports research or clinical trials grants paid to his Institution from Menarini, AstraZeneca, GlaxoSmithKline, Sanofi Genzyme and fees for lectures or advisory board participation from Menarini, AstraZeneca, CellTrion, Chiesi, Faes Farma, Firma, Genentech, Guidotti-Malesci, GlaxoSmithKline, HAL Allergy, Innovacaremd, Novartis, OM-Pharma, Red Maple, Sanofi-Aventis, Sanofi-Genzyme, Stallergenes-Greer and Uriach Pharma. All unrelated to this work. S Škrgat reports Honoraria for Educational events, invited lectures and presentations supported by Sanofi, AstraZeneca, Medis, Berlin Chemie, and Chiesi, as well as participation on a local advisory board for AstraZeneca. All unrelated to this work. S Siddiqui has received fees for advisory service from AstraZeneca, GlaxoSmithKline, Chiesi, Sanofi, Areteia. Speaker fees from AstraZeneca, GlaxoSmithKline, Chiesi, Areteia & Medscape, and is the ERS Clinical Research Collaborations Director. All unrelated to this work. S Rink has received honoraria for lectures and educational events from Berlin Chemie, Chiesi and Medis and support for attending meetings from AstraZeneca, GlaxoSmithKline, Chiesi and Berlin Chemie. All unrelated to this work. All other authors have no conflicts of interests to declare.

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Anti-inflammatory therapies for bronchiectasis

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Abstract

Purpose of review: Bronchiectasis is a condition that is characterized by chronic airway inflammation and usually infection, resulting in progressive airway damage. It results in a significant symptom burden and impacts both clinical and socioeconomic aspect of a patient's life. This review will detail anti-inflammatory treatment options for bronchiectasis, their benefits and possible side effects.

Recent findings: Inflammation is the main driver of bronchiectasis disease progression and has been the focus of research for many years. Inhaled corticosteroids, though not routinely recommended in guidelines, are still commonly used even in patients without other indication for treatment such as asthma, chronic obstructive pulmonary disease (COPD) and allergic bronchopulmonary aspergillosis (ABPA), despite their association with some worsened outcomes. Macrolides, on the other hand, have been shown to decrease frequency of exacerbation and improve quality of life. The dipeptidyl peptidase-1 inhibitor, brensocatib, inhibits activation of neutrophil serine proteases, including neutrophil elastase. In a phase 3 trial, brensocatib decreased the frequency of exacerbation, delayed time to first exacerbation, slowed the decline in lung function and improved quality of life. It recently became the first pharmacologic treatment approved by the U.S. Food and Drug Administration (FDA) for the treatment of bronchiectasis. Other treatment options are being investigated.

Summary: Anti-inflammatory therapy is one of the integral components of treatment for people with bronchiectasis.

Keywords: bronchiectasis; dipeptidyl peptidase-1 inhibitor; macrolides; neutrophilic inflammation.

- 41 references

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Multicenter Study

Infection

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Sex disparities in tuberculosis outcomes: evidence from a multicenter Italian cohort (Italian South TB Network (ISTB-Net)

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Abstract

Background: Sex disparities in tuberculosis (TB) outcomes are not well characterized, especially in high-income countries where social vulnerability and migration influence access to care. Although men globally experience a higher TB burden, the interaction between sex, migration, and social determinants is complex and extends beyond biological factors. This study evaluated sex differences in clinical and programmatic TB outcomes in a high-income European country with a significant substantial migrant population.

Methods: A retrospective multicentre cohort study was conducted across 16 Infectious Diseases Units in seven Italian regions from (January 2021 to September 2025). Outcomes included time to sputum conversion (in pulmonary TB), length of hospital stay (LOS), adverse events (AEs) and their severity, incomplete treatment (defined as failure, death, or loss to follow-up), and loss to follow-up (LTFU). Mixed-effects models were applied using two prespecified adjustment sets: sex, centre, and core confounders (Model A); and sex, centre, and clinically relevant baseline imbalances (Model B). Sub-analyses examined the impact of migration status.

Results: Of 982 TB patients, 229 (23.3%) were women and 753 (76.7%) were men. Women exhibited lower rates of smoking (24.4% vs 36.7%), diabetes (7.9% vs 15.8%), and COPD/bronchiectasis (4.5% vs 10.3%). The median sputum conversion time was 21 days for both sexes. Adjusted analysesindicated shorter LOS among women (Model A: - 22% [95%CI - 32 to - 10]; Model B: - 19% [95%CI - 28 to - 9]). Time to sputum conversion was slightly shorter in women in Model A (- 13%; 95%CI -23% to -1%) but not in Model B (- 9%; 95%CI -17% to 1%). The risk and severity of AEs were similar between sexes. In Model B, women had lower odds of incomplete treatment (OR 0.64 [95%CI 0.41 to 0.99]) and LTFU (OR 0.62 [95%CI 0.38 to 0.99]). Migrants experienced worse overall outcomes, but the effect of sex did not differ by migration status.

Conclusion: Women had consistently shorter hospital stays and greater treatment continuity without increased toxicity, indicating that sex differences in TB outcomes are likely attributable to social and behavioural factors rather than biological differences. Supportive associative networks and non-governmental organisations may help reduce sex disparities, underscoring the importance of sex- and migration-responsive TB care models in Europe.

Keywords: Adherence; Health equity; Hospital stay; Italy; Migration; Sex; Treatment outcomes; Tuberculosis.

Conflict of interest statement

Declarations. Conflict of Interests: The authors declare no competing interests.

- **24 references**
- **3 figures**

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