7th September 2026



ERS Highlights & Insights

INDIAN CONTEXT

DAILY NEWS UPDATES
FROM ERS CONGRESS 2026

5th – 9th September 2026



An educational initiative by

HALEON | Otrivin®

HALEON

An educational initiative by

Otrivin®



For the use of Registered Medical Practitioner, a Hospital and a Laboratory only. Images appearing herein are for illustration purposes only.
©2026 Haleon group of companies or its licensor. All rights reserved. Trademarks are owned by or licensed to the Haleon Group of Companies.
Haleon India Consumer Health Private Limited, 10th Floor, One Horizon Center, Sector 43, DLF Phase 5, Golf Course Road, Gurugram - 122002, Haryana, India.

7th September 2026

ERS Highlights & Insights

INDIAN CONTEXT

DAILY NEWS UPDATES FROM ERS CONGRESS 2026

THE EUROPEAN RESPIRATORY SOCIETY (ERS) CONGRESS 2026 | 5–9 SEPTEMBER | BARCELONA, SPAIN

Reduction in Exacerbations with Benralizumab in Severe Eosinophilic Asthma and Chronic Rhinosinusitis with Nasal Polyps, Even If Asthma Is Not Well-Controlled

Real-world Turkish data confirm that benralizumab, an anti-IL-5 receptor biologic that depletes eosinophils through antibody-dependent cell-mediated cytotoxicity, sharply reduces exacerbation rates and improves both asthma and sinonasal outcomes in patients with severe eosinophilic asthma, including those with concomitant chronic rhinosinusitis with nasal polyps (CRSwNP).

Study population

This retrospective study compared asthma exacerbation rates, systemic corticosteroid usage, asthma control, lung function and quality-of-life scores before and six months after starting benralizumab in 24 adults with severe eosinophilic asthma. The mean age was 53.92±12.98 years (range 33-77), and 62.5% (n=15) were female. Median eosinophil count was 540 (IQR 753) x10^6/L and median IgE was 248 (IQR 640) kIU/L; 58.3% (n=14) had concomitant CRSwNP. At baseline, 62.5% of patients were on systemic corticosteroids and 70.8% were on high-dose inhaled corticosteroid plus long-acting beta-agonist combinations.

Exacerbations fall, steroid doses drop

After six months of benralizumab therapy, the mean exacerbation rate fell markedly from 3.08±1.58 to 0.25±0.73, and 21 patients (87.5%) remained exacerbation-free. Among the 15 patients on systemic corticosteroids at baseline, treatment was discontinued entirely in 46.7% (n=7) and reduced in a further 33.3% (n=5); median systemic corticosteroid dose fell by 75% (p=0.002).

Control, quality of life and lung function all improve

Asthma Control Test (ACT) scores rose significantly from 11.79±4.80 to 18.33±5.55, and Asthma Quality of Life Questionnaire (AQLQ) scores rose from 3.27±0.93 to 4.73±1.37 (both p<0.001). Mean FEV1 and FVC increased significantly compared with baseline, although the FEV1/FVC ratio did not change significantly (p=0.164).

Nasal polyp symptoms also improve

Among the 14 patients with concomitant CRSwNP, the mean SNOT-22 sinonasal symptom score fell significantly from 35.21±11.98 to 19.11±11.96 at six months (p=0.001), indicating a marked improvement in nasal symptom burden alongside the pulmonary benefits.

What it means

The authors conclude that benralizumab effectively reduces asthma exacerbation rates, supports systemic corticosteroid dose reduction and/or discontinuation, improves asthma control, enhances asthma-related quality of life, leads to improvements in spirometric volumes, and provides beneficial effects on sinonasal symptoms — benefits observed even in patients whose asthma had not been well-controlled beforehand.

Reference: Dindar Çelik F, Aksu K, Göktürk Ö, Baştuğ İnan NB, Karahan Y, Yağdıran M, Telli O, Çelik E, Aksu F. Reduction in Exacerbations with Benralizumab in Severe Eosinophilic Asthma and Chronic Rhinosinusitis with Nasal Polyps, Even If Asthma Is Not Well-Controlled. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Beyond Fixed Dosing: Free IgE, FcεRI and Omalizumab Plasma Levels to Individualize Treatment in Severe Asthma

Omalizumab is an effective long-term treatment for severe asthma, traditionally dosed according to body weight and baseline IgE. New Spanish data suggest that, in patients who have achieved sustained clinical remission, tracking free IgE, FcεRI receptor expression and omalizumab plasma concentration (Cp) could help clinicians safely individualise dose-reduction strategies.

Tracking biomarkers through dose reduction

This secondary analysis of the multicentre OPT-OMA-ASTHMA clinical trial included severe asthma patients receiving stable omalizumab who had achieved sustained remission. Patients were assigned to a final dose reduction (FDR) of either 25% or 50%. Free IgE, FcεRI expression and omalizumab Cp were measured by ELISA and flow cytometry at baseline and at weeks 24, 48 and 96, alongside demographic, clinical and lung-function data.

Stable control, falling drug levels

Twenty-five patients were included (12 in the FDR-25% group, 13 in the FDR-50% group; mean age 49±18 years; 46% female), with no baseline differences between groups. Clinical and functional stability was maintained throughout, with no exacerbations recorded. Three patients discontinued the study due to loss of asthma control, two of whom had shown low omalizumab Cp.

Omalizumab plasma concentration fell progressively and in proportion to the dose reduction, from a baseline of 96±69 μg/mL to 63±37 μg/mL at 6 months, and to 44±16 μg/mL (FDR-50%) or 53±29 μg/mL (FDR-25%) by 12 months, reaching 36±18 μg/mL and 49±24 μg/mL respectively at 24 months.

By contrast, free IgE levels remained essentially stable, rising only slightly from 14±9 ng/mL at baseline to around 17-18 ng/mL across follow-up in both groups, and FcεRI receptor expression likewise stayed broadly stable, from 29,986±12,703 receptors/cell at baseline to a range of roughly 18,700-25,300 receptors/cell at 24 months across the two dose-reduction arms.

What it means

The authors conclude that the fall in plasma omalizumab concentration was consistent with the dose decrease, while the stability of free IgE levels and FcεRI expression suggests sustained functional blockade of the IgE pathway despite lower drug exposure. This, they argue, supports the potential role of free IgE, FcεRI expression and omalizumab Cp as pharmacodynamic biomarkers to guide individualised dose-reduction strategies in severe asthma, moving beyond fixed, weight- and IgE-based dosing.

Reference: Navarrete Rouco ME, Gimeno Martínez R, García Sanz A, Admetlló M, Sancho Muñoz A, Bravo D, Luengo O, Basagaña M, Soto Soto D, Ausin Herrero P. Beyond fixed dosing: free IgE, FcεRI and omalizumab plasma levels to individualize treatment in severe asthma. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Asthma Incidence and Rhinitis/Asthma Severity: A Pediatric SLIT vs SCIT Non-Inferiority Cohort Study

In one of the largest real-world comparisons to date, sublingual immunotherapy (SLIT) was found to be non-inferior to subcutaneous immunotherapy (SCIT) for preventing asthma in children with allergic airway disease, according to a Swedish nationwide register-based cohort study of more than 21,000 children.

A large register-based cohort

Both SLIT and SCIT allergen immunotherapy (AIT) are used in children with allergic airway disease, but comparative real-world evidence using standardised asthma and rhinitis outcomes has been limited. The study aimed to assess whether SLIT is non-inferior to SCIT for asthma incidence, asthma severity and rhinitis severity in children. Researchers analysed a

nationwide cohort of children aged 5-15 years initiating AIT in Sweden (n=21,014; SLIT n=10,087, 48.0%; SCIT n=10,927, 52.0%), assessing asthma incidence and asthma/rhinitis severity at 1, 3, 5 and 10 years using GINA treatment steps and EUFOREA rhinitis severity criteria, analysed with Cox proportional hazards models and ordinal logistic regression.

Lower asthma incidence with SLIT

Among children without asthma at baseline (n=6,848), SLIT was associated with a lower risk of incident asthma compared with SCIT, with an adjusted hazard ratio of 0.75 (95% CI 0.66-0.85). Among children with asthma at baseline (n=14,166), SLIT was associated with lower asthma severity, with similar effect estimates at 1, 3 and 5 years after initiation of allergen immunotherapy: adjusted odds ratios of 0.76 (95% CI 0.70-0.83) at year 1, 0.81 (95% CI 0.73-0.88) at year 3, and 0.76 (95% CI 0.68-0.85) at year 5, though the effect attenuated by year 10 (adjusted OR 0.89, 95% CI 0.73-1.08).

Rhinitis severity also favours SLIT — with caveats at 10 years

SLIT was also associated with lower rhinitis severity at 1, 3 and 5 years, with adjusted odds ratios of 0.78 (95% CI 0.73-0.84), 0.87 (95% CI 0.81-0.94) and 0.86 (95% CI 0.78-0.94) respectively. However, at 10 years of follow-up, inferiority of SLIT compared with SCIT with respect to both asthma and rhinitis severity could not be excluded, with adjusted odds ratios crossing 1.0 (asthma severity 0.89, 95% CI 0.73-1.08; rhinitis severity 1.07, 95% CI 0.92-1.24).

What it means

The authors conclude that this study, based on data from routine clinical practice, indicates SLIT is non-inferior to SCIT for the prevention of asthma, and that children treated with SLIT did not experience more severe rhinitis or asthma after treatment — though longer-term data beyond 10 years warrant continued monitoring

Reference: Konradsen JR, Putri R, Hedman A, Dahl Å, Brew B, Ingemansson M, Lundholm C, Almqvist C. Asthma Incidence and Rhinitis/Asthma Severity – a pediatric SLIT vs SCIT non-inferiority cohort study. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

The Relationship Between Concomitant Allergic Rhinitis With Serum Leptin and IL-6 Levels in Asthmatic Patients

Asthma patients with concomitant allergic rhinitis (AR) have significantly higher serum leptin levels than those without rhinitis — independent of the frequency or severity of their rhinitis — according to a Turkish study that also found AR to be linked with markedly poorer quality of life, regardless of asthma severity or control status.

Background and study population

It has been suggested that asthma and allergic rhinitis may be associated with high serum leptin levels independent of obesity, and that leptin levels may correlate with disease severity and outcomes. The study aimed to investigate the relationship between the presence of concomitant AR and asthma control, quality-of-life parameters, and serum leptin and IL-6 levels in non-obese asthmatic patients. The study included 176 asthma patients (118 female, 58 male); 91 of these patients (52%) were also diagnosed with AR, assessed using the Total Nasal Symptom Score (TNSS), reflective TNSS and a visual analogue scale (VAS). Patients were categorised into Group 1 (with AR) and Group 2 (without AR).

Distinct inflammatory profile in patients with rhinitis

Patients with concomitant AR (Group 1) showed statistically significant increases in eosinophil count (p=0.025), eosinophil-to-basophil ratio (p=0.003) and total IgE (p<0.001), while their neutrophil-to-lymphocyte ratio was significantly lower (p=0.048) — a laboratory pattern consistent with a more Th2-skewed, allergic inflammatory profile.

Poorer quality of life across multiple domains

Group 1 patients also had statistically significantly poorer scores across several quality-of-life domains, including emotional role difficulty (p<0.001), mental health (p=0.021), social functioning (p=0.001) and pain (p<0.001).

Leptin elevated regardless of rhinitis severity

Group 1 patients had significantly higher blood leptin levels than Group 2 (p=0.037). Notably, there was no

significant correlation between the frequency or severity of allergic rhinitis and the levels of blood leptin or IL-6, suggesting that the mere presence of concomitant AR — rather than its severity — is what distinguishes the leptin-elevated phenotype.

What it means

The authors conclude that asthma patients with AR had significantly elevated serum leptin levels regardless of the frequency and severity of their rhinitis, and that AR accompanying asthma has a negative effect on quality-of-life parameters regardless of asthma severity or control status. Leptin, they suggest, may serve as a discriminator in asthma patients, particularly in those with concomitant allergic rhinitis.

Reference: Kulaksız B, Eröz B, Kulaksız Y, Yıldız BP. The relationship between concomitant allergic rhinitis with serum leptin and IL-6 levels in asthmatic patients. Abstract 57535. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Association Between Habitual Snoring and Nasal IL-1β Levels in Children: Overall and Stratified by Allergic Rhinitis Status

Habitual snoring is significantly associated with elevated nasal inflammatory biomarker levels in children — but, strikingly, only in those who also have allergic rhinitis (AR), a Hong Kong study has found, suggesting that AR may amplify snoring-related nasal inflammation rather than acting independently of it.

Rationale and methods

Habitual snoring (HS) is a cardinal feature of paediatric sleep-disordered breathing, linked to upper airway inflammation via mechanical vibration stress. Allergic rhinitis, a prevalent comorbidity of sleep-disordered breathing, also contributes independently to nasal inflammation. Although interleukin (IL)-1β is a reliable inflammatory biomarker, evidence directly linking HS to nasal IL-1β — particularly in children with AR — has remained limited. The cross-sectional study enrolled primary school-aged children who underwent overnight polysomnography (PSG) and clinical assessment. Habitual snoring was defined as snoring three or more nights per week, and AR as a caregiver-reported physician diagnosis. Nasal epithelial lining fluid was collected using nasal strips, and IL-1β was measured by cytometric bead array; generalised linear models assessed associations between HS and log-transformed nasal IL-1β, stratified by AR status.

A significant overall association

The study included 116 children with a mean age of 8.70±1.70 years; 81.0% (n=94) had habitual snoring and 73.3% (n=85) had allergic rhinitis. Overall, HS was significantly associated with higher nasal IL-1β levels, with an adjusted beta coefficient of 0.853 (95% CI 0.210 to 1.496, p=0.011).

The effect is driven entirely by the AR subgroup

When stratified by rhinitis status, the association between HS and nasal IL-1β was significant only in children with AR: adjusted beta 1.03 (95% CI 0.12 to 1.95, p=0.030) in the AR group (n=85), compared with a non-significant adjusted beta of 0.48 (95% CI -0.35 to 1.32, p=0.270) in the non-AR group (n=31). In other words, in the AR subgroup, habitual snoring was significantly associated with higher nasal IL-1β levels, indicating an increased localised inflammatory response, while this relationship was not observed in children without AR.

What it means

The authors suggest that AR may potentiate snoring-related upper airway inflammation, possibly through pre-existing mucosal immune activation, rendering children with AR more susceptible to snoring-induced nasal inflammation — pointing to phenotypic heterogeneity in upper airway inflammation among snoring children. They conclude that habitual snoring was associated with increased nasal mucosal inflammation specifically in children with AR, and that children with both habitual snoring and allergic rhinitis may benefit from closer airway inflammatory monitoring and targeted anti-inflammatory strategies.

Reference: Yang M, Tsun JGS, Chan KYY, Jan ETY, Au CT, Yuen NTK, Chen TT, Leung NMW, Siu AKY, Li AM, Chan KCC. Association Between Habitual Snoring and Nasal IL-1β Levels in Children: Overall and Stratified by Allergic Rhinitis Status. Poster presented at: ERS Congress 2026; Barcelona, Spain.

IL-33 Enhances Disulfidptosis Sensitivity in Activated Eosinophils via SLC7A11 to Limit Eosinophilic Airway Inflammation

Researchers have identified a metabolic vulnerability in activated eosinophils that could be exploited as a novel, steroid-sparing treatment strategy for eosinophilic asthma. The study shows that IL-33 sensitises eosinophils to disulfidptosis — a form of cell death triggered by metabolic stress — via upregulation of the cystine transporter SLC7A11, and that a pharmacological agent exploiting this pathway alleviates eosinophilic airway inflammation in mice.

Rationale

Asthma is one of the most common chronic airway diseases worldwide and imposes a substantial disease burden in China; in type 2 asthma, eosinophils act as key effector cells. The study rested on three linked observations: IL-33 activates eosinophils, SLC7A11 imports cystine into cells, and metabolic stress can trigger disulfidptosis. Primary eosinophils were isolated from IL-5 transgenic (NJ.1638) mice and stimulated with IL-33 in vitro, with transcriptomics integrated with public asthma datasets, plus qPCR, Western blotting, immunofluorescence, cystine uptake assays and cell-death assays following treatment with the SLC7A11-pathway-targeting compound BAY-876, with or without the antioxidants TCEP or NAC. In vivo, wild-type mice underwent house dust mite (HDM) challenge on days 0, 7 and 14, with BAY-876 or vehicle given on days 14-16 and analysis on day 17.

SLC7A11 rises with IL-33 exposure and disease severity

Transcriptomic screening and protein validation confirmed robust upregulation of SLC7A11 in IL-33-activated eosinophils. SLC7A11 was also markedly increased in eosinophils from HDM-challenged lungs and in human asthma samples, where its expression was associated with greater disease severity.

A targetable metabolic vulnerability

IL-33 enhanced cystine uptake in eosinophils and sensitised them to BAY-876-induced cell death, an effect that was reversed by the antioxidants TCEP or NAC — confirming that the cell death occurred via oxidative, disulfidptosis-related mechanisms rather than off-target toxicity. In HDM-challenged mice, BAY-876 selectively reduced eosinophilic airway inflammation and ameliorated airway pathology, and combining low-dose BAY-876 with dexamethasone achieved greater suppression of airway inflammation than either treatment alone.

What it means

The authors conclude that IL-33 upregulates SLC7A11 in activated eosinophils, increasing their susceptibility to disulfidptosis, and that BAY-876 exploits this metabolic vulnerability to alleviate eosinophilic airway inflammation. They suggest that exploiting disulfidptosis may offer a selective therapeutic strategy for eosinophilic asthma with potential steroid-sparing benefits, given the enhanced effect seen when combined with low-dose glucocorticoids.

Reference: Lou J, Li W. IL-33 enhances disulfidptosis sensitivity in activated eosinophils via SLC7A11 to limit eosinophilic airway inflammation. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Air Pollution and Allergen Co-Exposure in Mice Induces T-Cell Expansion in Lymph Nodes and Local Lung Pathology With T2-Inflammation and Mucus Hypersecretion

Combining fine particulate air pollution with house dust mite (HDM) allergen exposure produces markedly worse airway inflammation in mice than either exposure alone, together with systemic immune activation in the lymph nodes — new Swedish research shows, underscoring how environmental pollution and allergen exposure can

interact to raise the risk of asthma exacerbations.

Setting up a co-exposure model

Air pollution is increasingly recognised as a key factor in lung health, yet its interplay with chronic inflammatory diseases such as asthma remains poorly studied. The researchers aimed to set up an experimental model to study the key cellular mechanisms induced by the combination of fine particulate matter (PM2.5) and house dust mite exposure in mice. Five-week-old C57BL/6J male mice were exposed to inhaled PM2.5 on four occasions, with or without HDM exposure, over three weeks. Mice were terminated 24 hours after the last exposure; bronchoalveolar lavage fluid (BALF) was collected and analysed for leukocytes, lung tissue was assessed for cytokine mRNA expression and histology, and lymph nodes were analysed by flow cytometry for lymphocyte counts.

Combined exposure worsens local and systemic inflammation

Mice exposed to HDM alone showed significant BAL eosinophilia and increased total cell infiltrates compared with controls, and these increases were further amplified in mice exposed to the combination of HDM and PM2.5, which also showed an additional increase in lymphocytes. The combination of PM and HDM aggravated lung gene expression of the chemotactic mediators KC ($p<0.05$) and IL-1β ($p<0.04$), as well as the type-2 transcription factor GATA3 ($p<0.04$), compared with HDM exposure alone. Comparing the effects of PM alone versus combined PM plus HDM exposure, significant gene upregulation was found for MCP-1 ($p<0.001$) and IL-1β ($p<0.04$).

Histological analysis using H&E and mucus staining showed increased tissue leucocyte infiltration and mucus hypersecretion in HDM-plus-PM-exposed mice, together with increased T-cell expansion in the draining lymph nodes — evidence of a coordinated local (lung) and systemic (lymph node) inflammatory response.

What it means

The authors conclude that this study highlights the importance of co-exposure models, capturing both local lung and systemic lymph-node effects. PM2.5 combined with HDM enhances the type-2 immune response, they note, thus increasing the exacerbation risks caused by environmental factors in airway diseases.

Reference: Mahmutovic Persson I, Ramu S, Vázquez Mera S, Malm Tillgren S, Menzel M, Drelciuc M, Lundgren M, Gribonika I, Uller L. Air pollution and allergen co-exposure in mice induces T-cell expansion in lymph nodes and local lung pathology with T2-inflammation and mucus hypersecretion. Presented at: ERS International Congress 2026; Barcelona, Spain.

Intranasal OM-85 Attenuates Allergen- and Virus-Induced Airway Inflammation by Restoring Epithelial–Macrophage Homeostasis

A bacterial lysate given intranasally can both dampen allergen-driven airway inflammation and blunt virus-triggered exacerbations, according to Swiss preclinical research that points to restoration of epithelial-macrophage cooperation as the underlying mechanism — with a durable protective effect lasting well beyond the treatment period.

Rationale

The rising burden of allergic airway diseases highlights the need for therapies that restore mucosal immune balance and limit infection-driven exacerbations. The researchers examined whether intranasal administration of OM-85, a bacterial lysate, reduces allergen- and viral-induced airway inflammation by modulating epithelial-macrophage interactions.

Study design

Female Balb/cJ mice received intranasal OM-85 (1 mg) or vehicle every 48-72 hours, beginning five days before and continuing through intranasal house dust mite (HDM, 25 μg) sensitisation and challenge. A subset of mice was infected with mouse-adapted influenza virus (75 PFU, intranasal) seven days after the final HDM exposure. Inflammatory profiles, mucus production and histopathology were assessed 96 hours after the last HDM exposure. Flow cytometry quantified epithelial and immune cell subsets, lung mechanics were measured using FlexiVent, and bulk RNA sequencing of CD45-positive and CD45-negative lung cells identified transcriptomic changes.

Reduced eosinophilia, improved lung mechanics

OM-85 reduced HDM-induced eosinophilia and mucus production, and improved lung compliance while reducing airway resistance — an effect that was sustained 14 days after allergen cessation. OM-85 also increased alveolar macrophage and

type II alveolar epithelial (ATII) cell numbers, suggesting strengthened epithelial-macrophage coordination and repair potential involving the signalling molecules BMP3 and Grem1. Transcriptomic profiling showed immune-regulatory reprogramming, with enrichment of epithelial-supportive and resolution-associated pathways and suppression of type 2 inflammation.

Antiviral benefit as well

In the influenza-challenged subset, OM-85 reduced viral load following flu exacerbation and dampened virus-exacerbated airway inflammation, indicating a dual protective effect against both allergen- and infection-driven airway disease.

What it means

The authors conclude that intranasal OM-85 mitigates allergen-induced airway inflammation by enhancing epithelial-macrophage interactions and promoting resolution. Its antiviral activity, they suggest, supports its potential as a mucosal immunotherapy for preventing allergic airway disease exacerbations.

Reference: Kouklas A, Trompette A, Guney S, Stickley L, Schiavetti F, Wieser N, Pasquali C, Baulier E, von Garnier C, Ubags N. Intranasal OM-85 Attenuates Allergen- and Virus-Induced Airway Inflammation by Restoring Epithelial–Macrophage Homeostasis. Presented at: ERS International Congress 2026; Barcelona, Spain.

Clinical Characteristics and Longitudinal Lung Function in People With Cystic Fibrosis and Allergic Bronchopulmonary Aspergillosis: A Multicenter Case-Control Study

Allergic bronchopulmonary aspergillosis (ABPA), a well-recognised complication of cystic fibrosis (CF), was linked to pancreatic insufficiency but not to faster lung function decline in a large Israeli multicentre study — a somewhat counterintuitive finding that highlights the complexity of tracking disease progression in this population.

Study design

ABPA is an important complication of CF associated with pulmonary morbidity and reduced lung function, though only a subset of people with CF develop the condition. The study aimed to compare clinical characteristics of people with CF with and without ABPA, and to evaluate longitudinal differences in lung function between the two groups. Researchers conducted a retrospective multicentre case-control study of patients aged six years and older followed between 2012 and 2023. ABPA was defined using international consensus criteria; multivariable logistic regression assessed associations with ABPA status, and longitudinal analyses compared FEV1% trajectories between groups.

Pancreatic insufficiency linked to ABPA

A total of 497 people with CF were included, of whom 57 had ABPA. In multivariable logistic regression, pancreatic insufficiency was independently associated with ABPA (odds ratio 2.51, 95% CI 1.19-5.30, p=0.016), whereas age, chronic Pseudomonas aeruginosa (PSA) infection, and baseline FEV1% were not significantly associated with ABPA status.

No clear link between ABPA and faster lung function decline

Surprisingly, unadjusted longitudinal analysis showed a greater decline in FEV1% in patients without ABPA than in those with ABPA (16% vs 9% between 2012 and 2023; p=0.12). However, in multivariate analysis controlling for age, sex, pancreatic insufficiency and PSA infection, ABPA status was not associated with differences in FEV1% trajectories over time (Pillai's Trace = 0.037, $F(12, 249) = 0.80$, p=0.65) — indicating that the unadjusted difference likely reflected other factors rather than a true protective or harmful effect of ABPA itself.

What it means

The authors conclude that, in this multicentre study, ABPA in CF was associated with pancreatic insufficiency, while adjusted longitudinal analyses showed no association between ABPA and FEV1% decline — highlighting the complexity of disease progression in this population and cautioning against over-interpreting simple unadjusted comparisons of lung function trajectories.

Reference: Dotan M, Tolkin S, Yaacoby-Bianu K, Bar-Yoseph R, Gur M, Golan Tripto I, Cohen-Cymberknoh M, Avneri L, Heching M, Mussaffi H, Gendler Y, Prais D. Clinical characteristics and longitudinal lung function in people with cystic fibrosis and allergic bronchopulmonary aspergillosis: a multicenter case-control study. Presented at: ERS International Congress 2026; Barcelona, Spain.

Lower Airway Immunophenotyping in Recurrent Severe Preschool Wheeze Reveals Clusters Distinguished by Infection and Neutrophil Phenotype

Bronchoscopy-based immune profiling of preschool children with recurrent severe wheeze has identified three distinct inflammatory clusters defined chiefly by lower airway infection and neutrophil activation status — rather than by wheeze severity itself or allergic sensitisation — a UK study reports, potentially reshaping how treatment-resistant wheeze is approached.

Background and rationale

About a third of preschool children with recurrent severe wheeze (RSW) have aeroallergen sensitisation and/or blood eosinophilia and tend to respond to inhaled corticosteroids, but most do not. Previous studies identified neutrophilia in bronchoalveolar lavage (BAL) and suggested that infection and/or neutrophilia may represent treatable traits. Since neutrophil subtypes with functional heterogeneity have been described in the adult lung, the researchers hypothesised that lower airway neutrophil subtype and function would relate to infection, allergic sensitisation and prescribed treatments in young children.

Methods

Children aged 1-5 years with RSW (n=106) and non-wheeze respiratory illness (NWRI, n=53) controls underwent clinical phenotyping, bronchoscopy, flow cytometric characterisation of BAL leukocytes, and assessment of lower airway infection.

Infection drives a distinct, activated neutrophil profile

Positive BAL bacterial culture was associated with BAL neutrophilia, more activated neutrophils (low CD62L and low CXCR2 expression), and higher soluble activation markers — including CD62L, myeloperoxidase and dsDNA — in BAL. RSW and NWRI groups showed the same bacterial species distribution, predominantly Moraxella catarrhalis, Haemophilus influenzae and Streptococcus pneumoniae.

Three immune clusters, independent of wheeze status

Unsupervised analysis revealed three distinct clusters: a neutrophilic, airway-infection-predominant cluster with low-CD62L neutrophils; an eosinophil/lymphocyte-rich cluster with high-CD62L neutrophils; and a pauci-granulocytic cluster characterised by low infection burden, high inhaled corticosteroid use, and high-CD62L neutrophils. Children with RSW and those with NWRI were evenly distributed across all three clusters, which were also independent of allergic sensitisation status.

What it means

The authors conclude that these clusters were differentiated by lower airway infection and neutrophil CD62L expression rather than by the presence of wheeze. Targeting underlying infection in children with RSW who fall into the neutrophilic, infection-predominant cluster may be more effective than escalating asthma therapies, they suggest, and this approach merits further investigation.

Reference: Bonner K, Fontanella S, Gore M, Cook J, Hoffland A, Scotney E, Tian K, Bush A, Custovic A, Lloyd C, Saglani S. Lower airway immunophenotyping in recurrent severe preschool wheeze reveals clusters distinguished by infection and neutrophil phenotype. Presented at: ERS International Congress 2026; Barcelona, Spain.

For the use of a Registered Medical Practitioner or a Hospital or a Laboratory only.

Conceptualized, Edited & Designed by Klub Class

Disclaimer: This newsletter contains information compiled by the Klub Class editorial team, Bangalore for the benefit of the medical profession. The references have been quoted to the extent possible; omissions if any are unintentional. While the attempt is to provide information that is in line with the evolving trends, M/s. Klub Class, Bangalore assumes no responsibility for the validity, completeness and correctness of the information provided.