9th September 2026



ERS Highlights & Insights

INDIAN CONTEXT

DAILY NEWS UPDATES
FROM ERS CONGRESS 2026

5th – 9th September 2026



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

Systemic and Airway Immunomodulatory Effects of Anti–IL-5 and Anti–IL-5Rα Therapy in Severe Eosinophilic Asthma

A detailed Greek immunophenotyping study is beginning to map exactly what anti-IL-5 and anti-IL-5Rα biologics do to the immune system beyond simply depleting eosinophils, revealing broad, coordinated suppression of inflammatory monocytes, basophils, dendritic cells, and a wide network of circulating cytokines, alongside a selective rise in regulatory T cells with IL-5Rα blockade.

Rationale and design

Severe eosinophilic asthma is driven by type 2-high inflammation, with IL-5 playing a central role in eosinophil differentiation, activation and survival. While biologics targeting IL-5 or IL-5Rα effectively reduce exacerbations and improve lung function, the precise underlying immunological mechanisms have not been fully elucidated. This study aimed to characterise airway and systemic immunological changes following anti-IL-5 or anti-IL-5Rα therapy and to correlate these with clinical outcomes.

Twenty-nine patients have been enrolled to date, with the protocol targeting a 12-month treatment course. At the time of reporting, 13 patients had completed at least six months of therapy; among them, nine had already achieved the full one-year regimen (benralizumab n=5, mepolizumab n=4), three (all mepolizumab) were ongoing towards the 12-month target, and one benralizumab patient discontinued at the six-month milestone. Patients were evaluated at baseline (T0), 6 months (T1) and 12 months (T2), with bronchoalveolar lavage (BAL), bronchial biopsies and peripheral blood collected at T0 and T2, and peripheral blood additionally collected at T1. Samples were analysed by spectral flow cytometry, ELISA and Luminex, and bronchial biopsies were assessed histologically for inflammation and remodeling; immunological findings were correlated with asthma control (ACT, RCAT, SNOT-22), exacerbation frequency and lung function.

A biologic-naive, comorbidity-heavy population

Of the 13 patients analysed, five were male (38%) and eight female (62%), with a mean age of 55.7±8.8 years; 85% were biologic-naive. The mean annual exacerbation rate at baseline was 2.5±1.5, and comorbidities were common: rhinitis (85%), obesity (69%, mean BMI 35.2±5.6 kg/m2), GERD (46%) and nasal polyps (31%). Baseline screening found positive skin prick tests in 54% and positive allergen-specific IgE in 46%.

Progressive clinical and airway improvement

Both anti-IL-5 and anti-IL-5Rα therapies produced substantial, progressive and broadly comparable improvements over 12 months, including a steady upward trend in post-bronchodilator FEV1, improved asthma control and quality of life (rising ACT and RCAT scores, falling SNOT-22 scores), and exacerbation frequency that fell drastically, approaching zero at both 6 and 12 months. Peripheral blood eosinophils fell dramatically by 12 months, with near-zero values particularly pronounced in the anti-IL-5Rα group, consistent with its cytotoxic mechanism of action; serum total IgE, by contrast, showed no consistent trend. In BAL, spectral cytometry revealed near-complete eradication of eosinophils by 12 months, with alveolar macrophages becoming the predominant cell population (over 90%), while lymphocytes and neutrophils remained low and stable. A general trend toward reduced destroyed airway epithelium was noted at 12 months, though this did not reach statistical significance given high patient heterogeneity — the researchers suggest the lack of uniform epithelial repair despite eosinophil depletion may point to IL-5-independent pathways contributing to persistent epithelial injury.

A broad systemic immune reset

Spectral cytometry of peripheral blood mononuclear cells also revealed reductions in inflammatory monocytes,

basophils and plasmacytoid dendritic cells, alongside increased CD4+ regulatory T cells. Both therapies progressively reduced basophils, approaching near-zero levels by 6 and 12 months, especially with anti-IL-5Rα, along with plasmacytoid dendritic cells and CD56dim CD16+ NK cells, and a downward trend was seen across all monocyte subsets. Major CD4+, CD8+ T-cell and B-cell compartments remained stable overall, though CD4+ regulatory T cells showed a modest upward trend selectively in the anti-IL-5Rα group. Serum analysis showed a progressive, marked decline across a broad panel of cytokines and chemokines in both groups, including classic type 2 mediators (IL-13, IL-4, IL-5), pro-inflammatory and T-helper cytokines (IL-12(p70), IL-2, IL-10, IL-17A, IL-23, IL-16, IL-6), the chemoattractant MIP-1α, and the type 1 mediator IFN-γ.

What it means

The authors conclude that anti-IL-5 and anti-IL-5Rα therapies modulate both systemic and airway immune responses, supporting their broader role in reducing inflammation and improving clinical outcomes in severe eosinophilic asthma. These findings demonstrate that the effects of these biologics extend well beyond simple eosinophil depletion to suppress a wide network of systemic inflammatory pathways.

Reference: Vlassi L, Theofani E, Kontogianni C, Zervas E, Anagnostopoulou C, Kaltsakas G, Vontetsianos A, Liapi A, Siora A, Papaioannou AI, Galani IE, Bakakos P, Rovina N. Systemic and airway immunomodulatory effects of anti–IL-5 and anti–IL-5Rα therapy in severe eosinophilic asthma. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Infant-Onset Persistent Wheeze and Atopy Trajectories Are Linked to the Poorest Respiratory Outcomes at Age 25 Years and Are Predictable From Early Life

Children who develop persistent wheeze or asthma together with atopy in infancy go on to have the worst respiratory health of any group by age 25, but a landmark 25-year Australian birth cohort study suggests these high-risk children can be identified far earlier in life, using a handful of readily available early-life risk factors.

Tracking wheeze and atopy together from birth to adulthood

Wheeze/asthma and atopy can co-occur, but no previous study had examined how they jointly evolve from birth to adulthood. Researchers used the Melbourne Atopy Cohort Study (MACS), a high-allergy-risk birth cohort of 620 children recruited between 1990 and 1994 and followed from birth to age 25, to identify wheeze/asthma-atopy trajectories, their early-life determinants, and adult outcomes, and to develop prediction models for the most adverse trajectories. Wheeze/asthma data were collected at 26 follow-ups via questionnaire, and atopy was assessed 11 times using skin-prick tests to food and aeroallergens, with group-based multi-trajectory modelling, multinomial regression and random forest models used for analysis.

Five distinct trajectories

Among 511 children with data at two or more timepoints, five trajectories emerged: infant-onset persistent wheeze/asthma plus atopy (75 children, 14.7%); infant-onset persistent wheeze/asthma plus adolescent-onset atopy (74, 14.5%); infant-onset transient wheeze/asthma (90, 17.6%); childhood-onset atopy (156, 30.5%); and a minimal wheeze/asthma-plus-atopy reference group (116, 22.7%). The two infant-onset persistent wheeze/asthma groups, combined, are referred to as the 'infant-onset persistent wheeze/asthma with atopy' trajectories.

Worst lung health by age 25

At age 25, children in the infant-onset persistent wheeze/asthma with atopy trajectories had lower pre- and post-bronchodilator lung function (FEV1, FEV1/FVC and FEF25-75%), greater airway reversibility and inflammation (higher FeNO), and higher use of asthma medication compared with other groups.

Early-life risk factors, and a predictive risk score

Several early-life factors were independently associated with the infant-onset persistent wheeze/asthma with atopy trajectories: parental asthma (multivariable odds ratio, mOR, 2.1, 95% CI 1.2-3.8), infantile eczema (mOR 7.8, 95% CI 3.7-16.2), having older siblings (mOR 1.9, 95% CI 1.1-3.3), childcare attendance (mOR 1.7, 95% CI 1.0-3.0), and lower respiratory tract infections in infancy (mOR 3.6, 95% CI 1.6-7.9).

The same risk factors, except parental asthma, were also associated with the infant-onset transient wheeze/asthma trajectory. The researchers derived risk score charts, stratified by sex, from a prediction model incorporating these factors, which achieved an area under the curve of 66.3% (95% CI 63.3-69.2%), sensitivity of 57.7%, specificity of 62.7%, and a notably high negative predictive value of 87.9% (95% CI 86.0-89.9%) — meaning the model was particularly good at ruling out the highest-risk trajectory.

What it means

The authors conclude that infant-onset persistent wheeze/asthma with atopy trajectories had the poorest respiratory health in adulthood, but that these high-risk children may be identifiable much earlier in life. Risk prediction models based on readily available early-life factors, they suggest, could eventually support early risk stratification, creating opportunities for closer monitoring and, ultimately, precision prevention.

Reference: Idrose NS, Lodge CJ, Lowe AJ, Vicendese D, Perret JL, Svanes C, Bennett CM, Walters EH, Wurzel DF, Chang AB, Abramson MJ, De Silva AP, Bui DS, Dharmage SC. Infant-onset persistent wheeze and atopy trajectories are linked to the poorest respiratory outcomes at age 25 years and are predictable from early life. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Plasma Proteomics Identifies Altered Immune Function in Paediatric Wheeze

Large-scale plasma proteomic profiling has uncovered more than 1,200 differentially expressed proteins linked to immune function in children with wheeze, according to Australian researchers who combined two independent paediatric cohorts to characterise the biological heterogeneity underlying this common condition.

Rationale

Wheeze is one of the most common reasons for children's hospital visits and contributes a significant medical burden. It is a heterogeneous condition with potentially multiple underlying biological mechanisms, including those related to inflammation and immune function. Recent advances in plasma proteomics may assist in understanding paediatric wheeze; the study aimed to characterise plasma proteomic signatures in paediatric wheeze, hypothesising that these signatures are altered in children with wheeze.

Two complementary cohorts

The study combined two cohorts: the Mechanisms of Acute Viral Respiratory Illness in Children (MAVRIC) cohort of 30 children with acute wheeze, and the Western Australian Epithelial Research Program (WAERP) cohort of 44 children, split into those with a history of wheeze (n=21) and those without (n=23). Blood and plasma were collected alongside clinical history, and plasma proteomics was performed using the Olink Explore HT platform, followed by principal component analysis, differential expression analysis, weighted gene co-expression network analysis (WGCNA), and pathway analysis. The MAVRIC children with acute wheeze were significantly older (median 7.4 years) than the WAERP children with (4.5 years) or without (3.4 years) a history of wheeze (p=0.0045), and the groups also differed significantly on measures including current wheeze, recurrent wheeze, asthma diagnosis, positive allergy testing, and parental history of asthma or allergies.

Cohort membership drives most of the variation

Cohort membership was found to be the major source of variation in the study: children in the MAVRIC cohort differed markedly from children in the WAERP cohort, while children within the WAERP cohort shared more similar protein expression profiles. Overall, 1,251 differentially expressed proteins related to immune function were identified between cohorts.

Shared protein networks and endotypes

To better understand the heterogeneity of paediatric wheeze, the researchers used WGCNA to identify unique protein modules showing significant network overlap between cohorts, and performed consensus network analysis to identify shared networks of protein expression. Top pathways enriched across these modules included neutrophil degranulation, platelet activation, signalling and aggregation, immunoregulatory interactions between lymphoid and non-lymphoid cells, extracellular matrix organisation, metabolism of nucleotides, and regulation of the complement cascade. In module-trait analysis, the 'blue' and 'brown' protein modules correlated with wheezing and allergic outcomes and were enriched for proteins associated with asthma: in the MAVRIC cohort, age, asthma, history of hayfever and eczema correlated with the blue module, and history of hayfever correlated with the brown module, whereas age was the

only significant correlate observed in the WAERP cohort.

What it means

The authors conclude that plasma proteomics showed differences between acute exacerbation and stable disease states, suggesting that health status at the time of sample collection can affect the proteomic profile obtained. Protein expression networks related to immune function were shared between children in the MAVRIC and WAERP cohorts, while participant characteristics such as asthma, a history of hay fever, and eczema correlated with protein modules enriched for asthma-associated proteins. These findings support plasma proteomics as a useful tool for dissecting the biological heterogeneity of paediatric wheeze.

Reference: Murali D, Catchpole E, Kim-Khoo S, Stick S, Noble P, Kicic A, Laing I, Le Souëf P, Hancock D, Iosifidis T. Plasma proteomics identifies altered immune function in paediatric wheeze. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Impact of Staphylococcal Enterotoxin Sensitization on Immunological Responses During Allergen Immunotherapy in Severe Allergic Rhinitis

Sensitisation to staphylococcal enterotoxins, which has long been linked to more severe and persistent allergic rhinitis, does not appear to blunt the immune response to allergen immunotherapy (AIT), according to a Ukrainian retrospective study. The findings suggest that such sensitisation need not exclude patients from receiving immunotherapy.

Background and cohort selection

Staphylococcal enterotoxin (SE) sensitisation is linked to severe allergic rhinitis, persistent symptoms and polysensitisation, but whether it affects allergen immunotherapy efficacy has been unclear. The study aimed to compare allergen-specific IgE dynamics during two years of subcutaneous AIT in patients with (SE+) and without (SE-) SE sensitisation. From a baseline cohort of 109 patients with severe allergic rhinitis, 23 completed two years of subcutaneous AIT for ragweed and/or birch. Of these, ten were SE+ (specific IgE to SEA, SEB, SEC or TSST ≥0.1 kU/L) and 13 were SE-; allergen-specific IgE was measured at baseline, one year and two years using ImmunoCAP. The two groups were closely matched: median age 39 years in both, similar sex distribution, and both groups had severe allergic rhinitis and were treated with ragweed or birch AIT.

Comparable immunological trajectories regardless of SE status

Both groups showed comparable specific-IgE dynamics, following the classical AIT pattern of specific IgE rising in year one before declining in year two — consistent with the development of immune tolerance. SE+ and SE- trajectories were similar, indicating that the core immune mechanisms of immunotherapy were preserved regardless of enterotoxin sensitisation status, with no significant differences found between the groups.

A promising signal from adjunctive bacterial lysates

In an exploratory sub-analysis, four SE+ patients with high Staphylococcus aureus colonisation received adjunctive bacterial lysate therapy. Paired SE-specific IgE measurements (for SEA, SEB, SEC and TSST, remeasuring only the seven enterotoxin markers that were elevated at baseline) showed a median reduction of 41% after bacterial lysate treatment, with reductions observed across all seven markers in the four patients. The authors note this analysis was exploratory and lacked a control group, but the consistent direction of the signal was described as promising.

What it means

The authors conclude that SE sensitisation did not impair allergen-specific immune responses during AIT, and that SE sensitisation may not be a contraindication to AIT in severe allergic rhinitis. They suggest that the reduction in SE-specific IgE seen with bacterial lysates warrants further study as a potential adjunctive therapy, and call for prospective controlled studies in larger cohorts with clinical endpoints to determine whether adjunctive immunomodulation with bacterial lysates can improve AIT outcomes in SE-sensitised patients.

Reference: Zaikov S, Kalikina I, Bogomolov A. Impact of staphylococcal enterotoxin sensitization on immunological responses during allergen immunotherapy in severe allergic rhinitis. Poster presented at: European Respiratory Society (ERS) Congress 2026; Barcelona, Spain.

Bronchial Epithelial Cell Pyroptosis Promotes Airway Inflammation and Remodeling in Allergic Asthma via the NLRP3/Caspase-1 Signaling Pathway

Chinese researchers have identified a specific form of inflammatory cell death, known as pyroptosis, in bronchial epithelial cells as a key driver of airway inflammation and remodeling in allergic asthma. They also showed that blocking the underlying NLRP3/caspase-1 pathway with a small-molecule inhibitor reversed multiple features of asthma pathology in mice.

Rationale

Allergic (eosinophilic) asthma is the most common asthma phenotype, and bronchial epithelial injury is a key pathological event in the disease. Pyroptosis is a pro-inflammatory form of cell death that releases IL-1β and IL-18, but its role in allergic asthma has remained unclear. The study aimed to investigate the specific role of bronchial epithelial cell (BEC) pyroptosis in the pathogenesis of allergic asthma and explore the underlying signalling pathway, hypothesising that NLRP3/caspase-1-mediated BEC pyroptosis drives airway inflammation and remodeling.

Methods

The researchers combined bioinformatic analysis with an ovalbumin (OVA)-induced allergic asthma mouse model to assess the role of pyroptosis and confirm pathway specificity using the NLRP3 inhibitor MCC950. Gene set variation analysis (GSVA) was used to assess pyroptosis enrichment in the public dataset GSE137268, while single-sample gene set enrichment analysis (ssGSEA) evaluated immune infiltration. A STRING protein-protein interaction network, together with the MCC algorithm, was used to identify hub genes. Functional and pathological assessments included lung function measurement using Penh, histological analysis with H&E for inflammation, Masson staining and α-SMA for airway remodeling, and PAS and MUC5AC for mucus production. Immunofluorescence was used to assess GSDMD-N localisation, Western blotting evaluated pyroptosis-related markers, and ELISA measured IL-4, IL-5, IL-1β, and IL-18 levels.

Pyroptosis is activated in allergic asthma

GSVA revealed significantly higher pyroptosis enrichment scores in asthma samples than in controls ($p<0.05$). In OVA-challenged mice, GSDMD-N expression, a marker of active pyroptosis, was elevated in bronchial epithelial cells on both immunofluorescence and Western blot analysis. This was accompanied by increased bronchoalveolar lavage fluid (BALF) levels of IL-1β and IL-18 ($p<0.001$).

NLRP3 identified as the central hub gene

Protein-protein interaction network analysis and the MCC algorithm identified NLRP3 as the top hub gene in asthma-associated pyroptosis regulation, with Western blot confirming its significant upregulation in asthmatic lungs ($p<0.001$). Treatment with MCC950 significantly reduced cleaved caspase-1 and GSDMD-N expression, confirming that the enhanced pyroptosis in asthma was specifically mediated by the NLRP3/caspase-1 pathway.

Blocking NLRP3 reverses asthma pathology

MCC950 treatment attenuated airway hyperresponsiveness as measured by Penh values ($p<0.01$), reduced inflammatory cell infiltration ($p<0.001$), decreased mucus hypersecretion as measured by MUC5AC expression ($p<0.01$), and reduced airway remodeling as measured by collagen deposition and α-SMA expression ($p<0.01$).

What it means

The authors conclude that bronchial epithelial cell pyroptosis contributes to the progression of allergic asthma through the canonical NLRP3/caspase-1 pathway, suggesting NLRP3 as a potential therapeutic target for allergic asthma.

Reference: Li M, Luo Z, Zhu W. Bronchial epithelial cell pyroptosis promotes airway inflammation and remodeling in allergic asthma via the NLRP3/caspase-1 signaling pathway. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Metabolically Armoured CAR-T Cells Enable Durable Targeting of Eosinophils in Chronic Respiratory Diseases

A newly engineered form of CAR-T cell therapy designed to target and eliminate disease-driving eosinophils, without the toxic preconditioning normally required for CAR-T treatment, has shown safety and efficacy in a mouse model of eosinophilic leukaemia. Chinese researchers suggest that the approach could eventually be applied across a range of eosinophil-driven respiratory diseases.

An unmet need across eosinophilic disease

Eosinophil-driven diseases span a broad clinical spectrum, from respiratory disorders such as severe asthma, chronic eosinophilic pneumonia and allergic bronchopulmonary aspergillosis, to rare haematological malignancies including hypereosinophilic syndrome and chronic eosinophilic leukaemia. These conditions are characterised by persistent eosinophilic inflammation, frequent relapse and limited long-term treatment options, resulting in substantial unmet clinical need, particularly in haematological disease. To address this, the researchers developed an IL-5 CAR-T cell therapy designed to selectively eliminate pathogenic eosinophils by targeting the restricted expression of the IL-5 receptor on eosinophils and their progenitors.

Overcoming the lymphodepletion problem

Conventional CAR-T approaches rely on lymphodepleting preconditioning, which imposes significant treatment burden and toxicity, limiting their suitability for non-malignant chronic inflammatory diseases. To overcome this limitation, the team engineered metabolically armoured CAR-T cells that constitutively secrete interleukin-10 (IL-10), providing metabolic and functional support within chronically inflamed tissues. This strategy is designed to reduce dependence on lymphodepletion while enhancing CAR-T cell persistence and therapeutic stability, enabling effective and sustained eosinophil depletion. The design is described as first-in-class: a cytokine-anchored IL-5 CAR-T cell expressing an endogenous IL-5 CAR, offering scFv-independent, low-immunogenicity engagement with the IL-5 receptor, intended to provide long-lasting eosinophil depletion with enhanced persistence in vivo.

From cell engineering to in vivo testing

The researchers conducted in vitro functional assessment including activation and cytotoxicity assays and tumorigenicity testing, followed by in vivo functional assessment using tumour xenograft models, sampling peripheral blood, bone marrow, lung, kidney, brain, heart and spleen to track the proportion of hIL-5 CAR-T cells, inflammatory factor release, tumorigenicity and tumour clearance in a leukaemia model, alongside dose-escalation pharmacokinetic evaluation.

What it means

The authors conclude that their findings show hIL-5 CAR-T cell therapy is safe and effective in a mouse model of eosinophilic leukaemia, highlighting its potential as a targeted immunotherapy for eosinophilic leukaemia and laying the groundwork for future clinical applications, including the broader treatment of eosinophil-driven chronic respiratory diseases.

Reference: Zhang R, Wu Y, Sun B, Xu F, Chen G, Chen X, Ying S. Metabolically armoured CAR-T cells enable durable targeting of eosinophils in chronic respiratory diseases. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

BreathDetective: Manual and Automated Exercise Breathing Analysis for Robust Research

A newly developed, freely available software toolbox aims to make cardiopulmonary exercise testing (CPET) data far more useful for studying disordered breathing, offering researchers and clinicians an open, evidence-based pipeline for analysing raw breath-by-breath data that current commercial tools largely leave untapped.

An underused source of data

Exertional breathlessness and disordered breathing are complex, understudied, and may contribute to

burdensome respiratory pathologies or performance limitations. Current screening approaches rely on expensive diagnostics and physiotherapist assessment, while cardiopulmonary exercise testing is common in clinical and research settings but underutilised for this purpose. Raw flow data may enrich CPET assessment, but few tools are available to make use of it, even though breath-by-breath data could help clinicians and researchers better understand breathing pattern disorders when benchmarked against evidence-based thresholds. The project's objective was to develop an open, evidence-based pipeline for semi-automated processing of CPET data to enable accurate, scalable analysis of exercise breathing; the resulting toolbox is freely available on the MATLAB file exchange under the name 'breathDetective'.The pipeline takes raw flowmeter data through automatic breath detection — including adaptive filtering, flow phase detection, CO2 validation and breath-by-breath output — followed by breath shape analysis and optional manual point-click editing, generating breath-by-breath data that feeds into automated dysfunctional breathing assessment, threshold detection and vital capacity calculation.

Matching manufacturer accuracy, with added insight

In a validation comparison against manufacturer software, breathDetective detected a very similar number of total breaths (10,021 vs 10,046 for the manufacturer software), and identified more instances of high respiratory frequency (over 80 breaths per minute: 261 vs 231) and a similar number of entrainment events during running (532 vs 580) — indicating that raw flow data analysis can improve CPET with novel, clinically relevant insights and context-specific accuracy, such as detecting entrainment between breathing and locomotion.

Practical applications

The developer suggests that breath shape analysis may support better characterisation of airway pathologies, such as exercise-induced laryngeal obstruction (EILO), or a better understanding of breathing techniques such as pursed-lip breathing. Dysfunctional breathing assessment leverages existing CPET data to complement existing screening and treatment protocols, and the tool is available as a standalone app compatible with most CPET exports; evidence-based thresholds built into the software help identify dysfunctional breathing phenotypes, such as erratic breathing versus hyperventilation.

What it means

The author concludes that CPET data could be used for a better understanding of disordered breathing in both clinical and scientific applications, and that the breathDetective toolbox enables rigorous, transparent analysis across various contexts, with opportunities for continuous development as an open resource for the field.

Reference: Harbour E. BreathDetective: Manual and automated exercise breathing analysis for robust research. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

CD84 (SLAMF5): A New Kid on the Block in Human ILC2 Biology

Researchers in Cambridge have identified CD84 (SLAMF5) as a previously unrecognised marker and functional regulator of human group 2 innate lymphoid cells (ILC2s), a discovery that may open new avenues for dampening type 2 inflammation in asthma and related conditions.

Filling a gap in ILC2 biology

The importance of ILC2s in asthma and other type 2 immune responses is well documented, but the mechanisms that modulate ILC2 antiviral and alarmin-driven responses have remained unclear. Mouse studies have implicated the Signalling Lymphocytic Activation Molecule (SLAM) receptor family in innate lymphoid cell regulation, prompting the researchers to investigate whether SLAMF5 (CD84) plays a similar role in human ILC2 biology. The study aimed to define the SLAM receptor landscape on human ILC2s, determine whether CD84 marks the ILC2 lineage and persists upon activation, and test whether CD84 functionally regulates type 2 cytokines in alarmin and viral contexts.

Methods

The researchers used multicolour flow cytometry to re-examine innate lymphoid cell subsets, reanalysed public single-cell RNA-sequencing data from chronic rhinosinusitis patients with and without nasal polyps to map SLAM family expression, and used an anti-CD84 antibody to assess CD84's role in secreted IL-5 and IL-13. A human air-liquid interface airway epithelium infected with rhinovirus (HRV-A16) was co-cultured with ILC2s, with or without anti-CD84, to assess cytokine output under viral challenge.

CD84 uniquely marks the ILC2 compartment

SLAMF1, SLAMF2, SLAMF5 and

SLAMF6 were all found to be enriched on ILC2s, with CD84 uniquely defining the ILC2 compartment. CD84 expression increased upon stimulation with the alarmins IL-33 and TSLP, whereas expression of the canonical ILC2 marker CRTH2 diminished — revealing a distinct CD84+CRTH2− ILC2 population. Reanalysis of single-cell RNA-sequencing data identified a corresponding CD84+/GATA3+ cluster.

A functional brake on type 2 cytokines

Functionally, engaging CD84 with an antibody reduced both intracellular IL-5 and IL-13 levels and secreted IL-5 from ILC2s. In rhinovirus-infected air-liquid interface co-cultures, anti-CD84 similarly suppressed type 2 cytokine output, suggesting that CD84 engagement could dampen ILC2-driven inflammation in both alarmin-driven and viral infection settings.

What it means

The authors conclude that CD84 emerges as a pivotal regulator of ILC2 biology, warranting further investigation across type 2 inflammatory diseases and positioning it as a potential new therapeutic target for dampening ILC2-driven inflammation in asthma and related conditions.

Reference: Haughey E, Chirichella M, Wyatt O, O'Callaghan C, Strickson S, Cohen S, Romero Ros X. CD84 (SLAMF5): A new kid on the block in human ILC2 biology. Presented at: ERS International Congress 2026; Barcelona, Spain.

Assessment of Small Airway Dysfunction in Patients Diagnosed With Rhinitis and Sinusitis According to Delphi Consensus

Small airway dysfunction (SAD) was detectable by oscillometry in the large majority of patients presenting with rhinitis and sinusitis, according to an Ecuadorian study applying a newly published expert consensus. The findings suggest that upper airway disease may be a stronger marker of lower small-airway involvement than previously appreciated.

Applying a new consensus for interpretation

Rhinitis and sinusitis are common chronic upper airway diseases and recognised risk factors for small airway disease. In 2025, an expert consensus was issued to guide oscillometry interpretation for SAD in adults with asthma or COPD. This study aimed to determine the prevalence of SAD in patients with rhinitis and sinusitis in accordance with that consensus.

Methods

A cross-sectional study was conducted at a tertiary centre on patients with recent-onset respiratory symptoms and rhinitis and/or sinusitis, recruited between May 2024 and August 2025. Oscillometry was performed using PulmoScan following ERS standards. SAD was defined by a z-score of R5 and AX greater than 1.64 and X5 less than -1.64, and bronchodilator response (BDR) was defined as a percentage change of more than 40% (R5), 50% (X5) or 80% (AX).

SAD found in the large majority of patients

Seventy-four patients were included (mean age 32 years, IQR 15.3-51.3; 67.6% female). The main comorbidities were rhinitis (63.5%), allergic rhinitis (14.9%), sinusitis (1.4%) and rhinosinusitis (20.3%). Median oscillometry values were R5 0.38 (IQR 0.30-0.52), R5-20 0.08 (IQR 0.04-0.25), X5 -1.64 (IQR -2.39 to -0.90), Ax 1.42 (IQR 0.74-5.08) and Fres 16.36 (IQR 10.07-20.76). Abnormal R5 was found in 68.9% of patients, with 16.2% classified as mild, 32.4% moderate and 20.3% severe impairment ($p<0.05$). X5 was impaired in 79.7% of patients, and AX was altered in 50%. Bronchodilator response was positive in 2.7% for R5, 55.4% for X5, and 9.5% for AX. Severity as assessed by R5 differed across diagnostic groups, while age influenced Z-score results for X5 and AX.

What it means

The authors conclude that oscillometry revealed small airway dysfunction in most patients with rhinitis and sinusitis, underscoring its value as a diagnostic tool in this population and its potential to help guide therapeutic decisions.

Reference: Cherrez-Ojeda I, Cardenas-Ponce F, Robles-Velasco K, Sagñay-Pinilla J, Crespo-Shijin C, Felix M, Hoheisel A, Calderon J. Assessment of Small Airway Dysfunction in Patients Diagnosed with Rhinitis and Sinusitis according to Delphi consensus. Presented at: ERS International Congress 2026; Barcelona, Spain.

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