8th 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

Sputum Autoantibody–Alarmin–ILC2 Crosstalk Underlies Persistent Type 2 Inflammation Across Exacerbation and Stable Disease States in Severe Asthma

A Canadian late-breaking study from Hamilton has mapped a self-reinforcing circuit linking airway autoantibodies, alarmin cytokines and innate lymphoid cells that may explain why some patients with severe asthma remain inflamed regardless of whether they are having an exacerbation or are clinically stable.

Rationale

Airway autoimmunity and dysregulated innate immunity are thought to drive persistent inflammation and treatment refractoriness in severe asthma (SA). Sputum IgG autoantibody (aAb) levels in SA have previously been shown to correlate positively with IL-5$^+$ILC2 counts and airway eosinophilia, and a novel alarmin, TL1A, which activates ILC2 and promotes their steroid insensitivity, is significantly increased in the sputum of SA patients with high aAb levels.

Objective and methods

The study set out to examine how autoantibodies, TL1A and ILC2 subsets relate to one another during both exacerbation and stability in severe asthma. Induced sputum was collected from 24 well-characterised SA patients at the time of an exacerbation and again more than eight weeks later once stable. Samples were examined for antinuclear autoantibodies (ANAs) and alarmin cytokines, and ILC2 subsets were enumerated by flow cytometry; patients reactive to two or more ANAs were classified as aAb$^+$.

Findings at exacerbation and at stability

At exacerbation, the aAb$^+$ subgroup (9 of 24 patients) showed significantly higher IL-13$^+$ILC2, IL-33 and IL-5 levels than aAb$^-$ patients. Anti-nucleosome antibody levels correlated with IL-13$^+$ILC2 counts ($p<0.01$) and with eosinophilia ($p<0.05$), and these latter two measures also correlated with each other ($p<0.05$). Once stable, a slightly different aAb$^+$ subgroup (8 of 24) showed higher IL-5$^+$ILC2 and TL1A levels, both of which correlated with anti-nucleosome antibodies ($p<0.05$); IL-5, IL-13 and IL-33 remained elevated in this group as well.

A dynamic, longitudinal link

Tracking patients over time, changes in anti-nucleosome antibody levels correlated with changes in IL-13$^+$ILC2 ($p<0.05$) and IL-5 ($p=0.05$), with a trend toward correlation with TL1A ($p=0.09$) — indicating that the autoantibody-ILC2 relationship shifts dynamically alongside disease state rather than remaining fixed.

What it means

The authors conclude that sputum autoantibodies are associated with dynamic ILC2 activation, linking IL-33-driven IL-13$^+$ILC2 responses during exacerbation to TL1A-associated IL-5$^+$ persistence during stability — supporting the concept of an autoimmune-driven ILC2 axis that sustains type 2 inflammation across both disease states in severe asthma.

Reference: Merjo E, Emami Fard N, Ju X, Chen L, Wiltshire L, Huang C, Xie Y, Satia I, O'Byrne P, Ho T, Nair P, Mukherjee M, Sehmi R. Sputum autoantibody-alarmin-ILC2 crosstalk underlies persistent type 2 inflammation across exacerbation and stable disease states in severe asthma. Late Breaking Abstract presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Reprogramming of Pulmonary Neuroendocrine Cells as a Novel Mechanism of Eosinophilic COPD

An international team spanning Manchester, Houston and Spain has identified reprogrammed pulmonary neuroendocrine cells (PNECs) as a previously unrecognised driver of eosinophilic COPD, uncovering a hypoxia-linked, remodeling-supportive epithelial programme that could point to new therapeutic targets.

Background

A subset of COPD patients have elevated blood eosinophil counts (BEC) and a distinct phenotype that responds differently to corticosteroids and type-2 biologics. While BEC is used to guide care, the epithelial programmes that sustain airway eosinophilia have been unclear. The researchers hypothesised that specific epithelial niches drive eosinophilic COPD (EOS-COPD).

Three cohorts, spatial and single-cell approaches

Three cohorts were analysed using spatial transcriptomics and single-cell RNA sequencing. Cohort 1 examined peripheral lung tissue from EOS-COPD (BEC ≥300/μL) versus non-eosinophilic COPD (NEOS-COPD; <300/μL; N=64). Patients with BEC 150–300/μL were reclassified as NEOS-COPD based on scRNA-seq findings from Cohort 2 (N=61), which showed lung eosinophil levels similar to those with BEC <150/μL. Integrated analyses defined epithelial states, eosinophil subsets and ligand–receptor signalling, with findings validated in bronchial biopsies from a third cohort (N=41).

A distinct PNEC signature in EOS-COPD

EOS-COPD showed enrichment of PNEC-linked pathways, while NEOS-COPD displayed myeloid-dominant programmes instead. Single-cell sequencing revealed increased eosinophils, B cells and innate lymphoid cells in EOS-COPD, with M2-like macrophages predominating in NEOS-COPD. Although the overall number of PNECs was similar between groups, those in EOS-COPD were substantially reprogrammed: neuroendocrine markers (ASCL1, GRP) were reduced, hypoxia and stress pathways (HIF1A, NR4A1) were induced, and eosinophil-supporting factors (POSTN, IGF1) and remodeling factors (TIMP2, ADAM17) were increased. Distinct eosinophil subsets expressing ITGAM and IGF1R were also identified, and bronchial biopsies confirmed submucosal PNEC enrichment in EOS-COPD, consistent with the cells relocating to a different tissue niche.

What it means

The authors conclude that EOS-COPD is characterised by hypoxia-linked PNEC reprogramming and a remodeling, eosinophil-supportive secretome, which may represent a novel therapeutic strategy for this COPD phenotype.

Reference: Higham A, Ito A, Rubio D, Sanz J, Kang S, Zhang Y, Rojas-Quintero J, Malo J, Kheradmand F, Cabrera-Lopez C, Marin J, Lee H, Singh D, Polverino F. Reprogramming of pulmonary neuroendocrine cells as a novel mechanism of eosinophilic COPD. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Effects of Targeting Higher Versus Lower Oxygen Saturation Limits in Preterm Infants With Bronchopulmonary Dysplasia: A Multicentre Randomized Controlled Trial

A Dutch late-breaking randomized trial has found that targeting a higher blood oxygen saturation limit in preterm infants with established bronchopulmonary dysplasia does not improve weight gain, and instead prolongs the need for respiratory support.

Background and aim

Supplemental oxygen remains the primary treatment for preterm infants with bronchopulmonary dysplasia (BPD), but evidence on lower oxygen saturation (SpO2) targets beyond 36 weeks postmenstrual age (PMA) has been lacking. The Supplemental

Oxygen Strategies in children with BronchoPulmonary Dysplasia (SOS BPD) study aimed to assess the effect of an SpO2 lower limit of 95% compared with 90% on weight standard deviation score (SDS) — used as a proxy for lung growth — at 6 months corrected age (CA).

Methods

In this multicentre, non-blinded randomized controlled trial conducted across the Netherlands, preterm infants with moderate or severe BPD were randomized between 36–38 weeks PMA to an SpO2 lower limit of either 90% (n=102) or 95% (n=99), continued until respiratory support was successfully stopped. The primary outcome was weight SDS at 6 months CA, alongside 24 prespecified secondary outcomes analysed up to the same timepoint.

Results

The cohort had a mean gestational age of 27 weeks, and 46.7% were male. Mean weight SDS at 6 months CA was -0.9 in the 95% group and -0.8 in the 90% group, a difference that was not statistically significant (mean difference -0.16, 95% CI -0.46 to 0.13). However, infants targeted to the 95% limit needed respiratory support for longer (median 41.0 vs 31.5 days; hazard ratio 0.60, 95% CI 0.44 to 0.81) and reached a higher postmenstrual age before support was discontinued (median 42.9 vs 41.1 weeks; median difference 2.6 weeks, 95% CI 1.4 to 4.0). No other secondary outcomes differed between groups.

What it means

The authors conclude that targeting an SpO2 lower limit of 95% rather than 90% did not improve weight SDS at 6 months CA in infants with established BPD, and produced no other observed clinical benefit — while prolonging respiratory support. They call for follow-up studies to assess the impact of oxygen targeting on longer-term neurodevelopment and lung function.

Reference: de Ridder R, Balink S, Onland W, Dijk P, Hulsmann A, van Kaam A, Kroon A, Merkus M, Reiss I, Sprij A, Vrijlandt E, Pijnenburg M. Effects of targeting higher versus lower oxygen saturation limits in preterm infants with bronchopulmonary dysplasia: a multicentre randomized controlled trial. Late Breaking Abstract presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Develop0ment of a Novel Cardiovascular Risk Assessment Tool Specific to Patients With COPD That Includes Spirometry, Exacerbations and Inflammatory Biomarkers

A Canadian team from Montreal has built and validated a COPD-specific cardiovascular risk score in nearly 50,000 UK Biobank participants, showing it outperforms the general-population PREVENT score at predicting cardiovascular disease in people with COPD.

Introduction

Cardiovascular disease (CVD) risk scores are known to underestimate risk in patients with COPD, and the researchers proposed that this could be improved by incorporating CVD risk factors specific to COPD.

Aims and methods

The study aimed to determine whether adding spirometry, exacerbations, inflammatory biomarkers and inhaled corticosteroid (ICS) use to traditional cardiovascular risk factors would improve CVD risk prediction in COPD, hypothesising that a COPD-specific model would outperform validated scores such as PREVENT. Participants with COPD (defined by FEV1/FVC <0.7 or z-score <-1.645, or a recorded ICD code for COPD) and no prior history of CVD were drawn from the UK Biobank and followed for 10 years for incident myocardial infarction, stroke or heart failure. Competing-risk Cox models were derived separately in men and women, adding FEV1, exacerbations requiring hospitalisation, ICS use, neutrophil/lymphocyte ratio and C-reactive protein to the standard PREVENT risk factors (age, smoking, BMI, total cholesterol, HDL, systolic blood pressure, diabetes, statins, antihypertensives and eGFR). Discrimination and calibration were compared against PREVENT.

Results

Among 27,073 men and 23,460 women with COPD, 11.5% and 7.2% respectively

went on to develop CVD. The new COPD-specific risk score achieved a C-statistic of 0.697 (95% CI 0.688–0.706) in men and 0.733 (95% CI 0.721–0.744) in women — significantly better discrimination than PREVENT, which scored 0.669 (95% CI 0.661–0.678) in men and 0.708 (95% CI 0.697–0.720) in women. Calibration of the new model was similar to that of PREVENT in both sexes.

What it means

The authors conclude that patients with COPD benefit from a dedicated cardiovascular risk score that combines spirometry, exacerbations, inflammatory biomarkers and ICS use with traditional risk factors, offering better discrimination than the existing PREVENT tool.

Reference: Krishnan S, Li P, Bourbeau J. Development of a novel cardiovascular risk assessment tool specific to patients with COPD that includes spirometry, exacerbations and inflammatory biomarkers. Late Breaking Abstract presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Relevance of Bronchial Mucosa "Inflammometry" in Severe OCS-Dependent Refractory Asthma

An Italian study using bronchial biopsies has characterised a distinct inflammatory signature in severe oral-corticosteroid-dependent asthma, identifying cellular thresholds that could help predict which patients have this steroid-resistant phenotype.

Background and aim

Steroid-resistant asthma includes severe oral corticosteroid-dependent refractory asthma (SOCDRA), a phenotype whose underlying molecular mechanisms remain unclear; current guidance recommends investigational bronchoscopy to characterise airway inflammation in severe refractory asthma. This study aimed to evaluate the inflammatory profile of the bronchial mucosa in SOCDRA and its relationship with clinical and functional features.

Methods

In this cross-sectional study, bronchial biopsies from 40 asthmatics were analysed across three groups: 10 with severe OCS-dependent asthma (OCS use ≥3 months/year; OCS^+), 15 with severe non-OCS-dependent asthma (OCS^-), and 15 with mild asthma (CTRL-mild). Clinical and functional data were compared between groups, and immunohistochemistry and immunofluorescence were used to assess inflammatory cells, T1/T2/T3 cytokines, inflammasome components, and eosinophil glucocorticoid receptor-β (GRβ) expression in the bronchial mucosa.

Results

Compared with the OCS^- and CTRL-mild groups, OCS^+ patients showed a higher exacerbation rate and prevalence of osteoporosis, increased residual volume and functional residual capacity, reduced pre- and post-bronchodilator FEV1 and F_{ENO}, and longer disease duration. Both severe groups showed increased bronchial neutrophils and $IL\text{-}13^+$ and $eotaxin\text{-}3^+$ cells compared with CTRL-mild, while OCS^+ patients additionally showed greater bronchial eosinophilia and higher $CD4^+$, $eotaxin\text{-}3^+$ and $IL\text{-}1\beta^+$ cells than the other two groups. Confocal microscopy revealed higher eosinophil-GRβ expression in the OCS^+ group. $Eotaxin\text{-}3^+$ and $IL\text{-}1\beta^+$ cell counts correlated with exacerbation frequency, while IL-1β correlated with prednisone dose, and ROC analysis identified cut-offs of eotaxin-3 >14.04, IL-13 >5.67 and IL-1β >4.65 cells/mm² as predictive of SOCDRA.

What it means

The authors conclude that SOCDRA is characterised by increased T2 cytokines and IL-1β expression in the bronchial mucosa, which promote eosinophil-related GRβ overexpression, recurrent exacerbations and OCS-associated comorbidities.

Reference: Ricciardolo F, Arrigo E, Carriero V, Levra S, Profita M, Guida G, Bertolini F. Relevance of bronchial mucosa "inflammometry" in severe OCS-dependent refractory asthma. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

ECG Abnormality Increased Risk of COPD, Cardiovascular Events, and Death in Individuals With Pre-COPD: A Prospective Cohort Study

A South Korean cohort study has found that the combination of pre-COPD and an abnormal ECG carries a synergistically higher risk of cardiovascular events, COPD progression and death — with either factor alone posing far less risk.

Background

The prognostic significance of electrocardiogram (ECG) abnormalities in individuals with pre-COPD has remained unknown.

Methods

Using data from the Ansan-Ansung cohort, the researchers conducted a prospective analysis in which participants were classified into four groups based on the presence or absence of pre-COPD and ECG abnormalities suggestive of ischaemic cardiac pathology. Multivariable Cox proportional hazards regression models evaluated associations between these exposure groups and incident COPD, major adverse cardiovascular events (MACE), and all-cause death.

Results

Among 6,487 participants included in the MACE/COPD analysis and 5,972 in the mortality analysis, individuals with both pre-COPD and an ECG abnormality had significantly higher risks of incident MACE (adjusted hazard ratio [aHR] 2.14, 95% CI 1.52–3.03), incident COPD (aHR 1.59, 95% CI 1.08–2.34), and all-cause mortality (aHR 1.46, 95% CI 1.07–1.99) compared with those with neither condition. By contrast, isolated pre-COPD or an isolated ECG abnormality showed only modest, non-significant associations with these outcomes.

What it means

The authors conclude that the coexistence of pre-COPD and ECG abnormalities synergistically increases the risk of cardiovascular events, COPD progression and death, and suggest that ECG assessment may help identify high-risk individuals among those with pre-COPD who warrant closer surveillance and early intervention.

Reference: Kim S, Rhee C, Choi J, Min K. ECG abnormality increased risk of COPD, cardiovascular events, and death in individuals with pre-COPD: a prospective cohort study. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Clinical Determinants of Blood Eosinophil Count in Obstructive Lung Disease: A Nationwide Cohort Study

A Danish nationwide study of more than seven million blood eosinophil measurements has shown that everyday clinical factors, including recent steroid use, hospital admission, and antibiotic use, can meaningfully shift blood eosinophil counts, with important implications for how this biomarker is interpreted and used to guide treatment.

Background

Blood eosinophil count (BEC) is widely used to guide treatment decisions in obstructive lung disease. However, individual BEC measurements can be influenced by several clinical factors that may lead to misclassification during clinical decision-making. The study aimed to investigate how BEC measurements are influenced by common clinical factors in individuals with asthma and COPD.

Methods

The researchers assembled a Danish nationwide cohort of 678,096 individuals aged 35 years or older who had at least five BEC measurements taken between 2015 and 2025, with each measurement obtained at least one month apart. Within each period, the highest value was selected, resulting in a total of 7,196,276 BEC samples for analysis.

Clinical factors examined included age, sex, oral corticosteroid (OCS) use, antibiotic use, blood neutrophil count, and whether the blood sample was obtained during a hospital admission. The associations between BEC and these

clinical factors were evaluated using linear mixed-effects models stratified according to disease category.

Results

All investigated clinical factors were significantly associated with BEC. OCS use during the seven days before sampling was consistently associated with lower BEC across all disease groups, with reductions ranging from 38 to 85 cells/μL.

In contrast, both hospital admission and antibiotic use were associated with higher BEC, with increases ranging from 15 to 67 cells/μL. Blood neutrophil count showed only a small inverse association with BEC, suggesting a comparatively limited influence on eosinophil levels.

What It Means

The authors conclude that individual BEC measurements are meaningfully influenced by the clinical context in which they are obtained. Recent OCS exposure, hospital admission, and antibiotic use should therefore be considered when interpreting BEC and using it as a treatment-guiding biomarker in patients with asthma and COPD. Awareness of these factors may help clinicians interpret individual measurements more accurately and reduce the risk of treatment decisions being based on potentially misleading BEC values.

Reference: Ascanius Jacobsen P, Colak Y, Afzal S, Håkansson KEJ, Dam Lauridsen M, Torp-Pedersen C, Kragholm K, Møller Weinreich U. Clinical determinants of blood eosinophil count in obstructive lung disease: a nationwide cohort study. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Exacerbation Reduction Over Three Years With Beclometasone/Formoterol/Glycopyrronium in Asthma: Real-World Evidence From the TriMaximize Study

Real-world data from 162 sites across eight European countries show that single-inhaler triple therapy with beclometasone/formoterol/glycopyrronium produces a large and sustained reduction in asthma exacerbations over three years of routine clinical use.

Background

Frequent exacerbations remain a significant challenge in asthma management, contributing to increased morbidity and healthcare burden. Single-inhaler triple therapy with an extrafine formulation of beclometasone dipropionate/formoterol fumarate/glycopyrronium (BDP/FF/G) has confirmed clinical benefits in controlled trials, but real-world evidence on its impact on exacerbation rates in patients with uncontrolled moderate-to-severe asthma has been limited. The TriMaximize study was designed to evaluate exacerbation outcomes with BDP/FF/G in routine clinical practice.

Methods

Asthma patients prescribed BDP/FF/G were observed at 162 sites across eight European countries (Germany, UK, Austria, Denmark, France, Spain, Poland and Italy). Moderate exacerbations were defined as two or more consecutive days of increased rescue bronchodilator use (≥4 puffs/day), and severe exacerbations as those requiring systemic corticosteroids for three or more days, or resulting in emergency care or hospitalisation.

Results

A total of 1,445 patients were included in the analysis (62.8% female; mean age 57.6 years; 17.9% current smokers). Before initiating BDP/FF/G, 85.0% of patients had experienced at least one moderate exacerbation and 27.1% at least one severe exacerbation. After one year, these proportions fell to 9.6% and 9.3% respectively, with further reductions in the following two years. The mean number of moderate and severe exacerbations declined from 1.8 at baseline to 0.2 after the first year and 0.1 after the third year — a reduction from baseline of 1.5 exacerbations in year one ($p<0.0001$), 1.6 in year two ($p<0.0001$), and 1.5 in year three ($p\leq0.0021$).

What it means

The authors conclude that a significant and sustained reduction in exacerbations was observed over a three-year period in patients with moderate-to-severe asthma treated with BDP/FF/G in real-world clinical practice.

Reference: Trinkmann F, Bogoevska V, Russell R, Ulrik C, Pohl W, Plaza V, Bourdin A, Kupczyk M, Braido F, Hahn A, Fritz C, Grickschat V, Gessner C. Exacerbation reduction over three years with beclometasone/formoterol/glycopyrronium in asthma: real-world evidence from the TriMaximize study. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Lifetime Smoking Duration, Intensity and Cigarette Constituents Differentially Affect Fractional Exhaled Nitric Oxide

Swedish analysis of more than 6,000 NHANES participants has shown that cumulative smoking exposure and specific cigarette characteristics, but not nicotine or tar content, are associated with lower fractional exhaled nitric oxide in current smokers. The findings have implications for interpreting this biomarker in people with a smoking history.

Background

Fractional exhaled nitric oxide (FeNO) is widely used as a non-invasive biomarker of type 2 airway inflammation. However, active cigarette smoking can markedly suppress FeNO levels, potentially complicating clinical interpretation. The smoking exposures and cigarette characteristics responsible for this effect remain incompletely understood.

Methods

Researchers analysed data from 6,229 adults in NHANES 2007–2012 with FeNO measurements and documented smoking history, including 3,124 current smokers and 3,105 former smokers. Linear regression models estimated FeNO reduction across quartiles of smoking exposure, using the lowest quartile as reference. Analyses were adjusted for age, sex, race or ethnicity, body mass index, blood eosinophil count, and physician-diagnosed asthma and COPD. Smoking exposures included pack-years, years smoked, and cigarettes per day. In current smokers, cigarette carbon monoxide, nicotine and tar content, and cigarette length were also assessed.

Results

Among current smokers, longer smoking duration, more cigarettes per day, higher pack-years, longer cigarette length, and higher carbon monoxide content were associated with greater FeNO reduction. Nicotine and tar content were not associated with FeNO suppression.

In former smokers, associations between smoking duration, intensity, and FeNO suppression were less consistent.

What It Means

The authors conclude that FeNO suppression in current smokers is mainly associated with cumulative smoking exposure, smoking intensity, and selected cigarette characteristics. A detailed smoking history should therefore be considered when interpreting FeNO and using it to support clinical decision-making.

Reference: Abohalaka R, Nwaru B, Kankaanranta H, Backman H, Radinger M, Vanfleteren L. Lifetime smoking duration, intensity and cigarette constituents differentially affect fractional exhaled nitric oxide. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

Biomarkers of Immunothrombosis and Sleep-Related Hypoxaemia Are Independently Associated With Persistent Perfusion Defects 3 Months After Pulmonary Embolism

A Spanish multicentre study has linked circulating markers of immunothrombosis and nocturnal hypoxaemia with persistent lung perfusion defects three months after acute pulmonary embolism. The findings highlight potential biomarkers that may help identify patients at risk of chronic thromboembolic disease.

Background

Persistent perfusion defects following acute pulmonary embolism (PE) are associated with chronic thromboembolic disease and chronic thromboembolic pulmonary hypertension. The researchers investigated whether biomarkers of endothelial dysfunction, inflammation and NETosis, together with sleep-related hypoxaemia, were

associated with residual perfusion defects at three months.

Methods

Patients from the NIX cohort who had completed standard anticoagulation underwent ventilation/perfusion (V/Q) assessment at three months. Clinical variables and sleep parameters were compared between patients with and without persistent perfusion defects.

Sleep-related hypoxaemia was assessed using CT90, defined as the percentage of time spent with oxygen saturation below 90%. Blood biomarkers were measured using ELISA, colorimetric, and fluorometric assays. Group differences were assessed using two-sided Mann-Whitney tests, followed by multivariable logistic regression using predefined cut-offs for vascular endothelial growth factor (VEGF), superoxide dismutase (SOD), cell-free double-stranded DNA (dsDNA), and CT90.

Results

Among 136 patients, 63 had persistent perfusion defects at three months. Statin use was more frequent among patients with defects than among those without defects (37.7% vs 20.5%; p=0.049). CT90 was also significantly higher in the defects group (28.13% vs 16.58%; p=0.02).

Several circulating biomarkers were elevated in patients with persistent defects, including dsDNA (p=0.019), VEGF (p=0.028), nucleosomes (p=0.028), and SOD (p=0.010).

In multivariable analysis, independent predictors of persistent perfusion defects were VEGF ≥236 pg/mL (OR 3.74, 95% CI 1.58–8.88; p=0.003), SOD ≥4.57 U/mL (OR 3.03, 95% CI 1.27–7.23; p=0.013), dsDNA ≥67 ng/mL (OR 2.90, 95% CI 1.18–7.11; p=0.020), and CT90 (OR 1.019, 95% CI 1.002–1.035; p=0.026).

What It Means

The authors conclude that persistent perfusion defects after pulmonary embolism are independently associated with sleep-related hypoxaemia and circulating markers of endothelial activation, oxidative stress, and cell-free DNA. These findings support immunothrombosis and hypoxaemia pathways as potential targets for precision risk stratification after PE.

Reference: Gonzalo-Martin E, Carrera Cueva C, Sola Idigora N, Marín Hinojosa C, Sánchez-Roncero A, Cervantes Borreguero L, Núñez Ollero M, García Sánchez A, Cano Pumarega I, Oscullo Yépez G, Osorio Trujillo J, Urrutia Gajate A, Cabriada Nuño V, Rivas Guerrero A, García Ortega A, Abad Fernández A, Oto Martínez J, Medina Badenes P, Andrade Ruiz H, Asensio Cruz M, Elías Hernández T, Sánchez-Armengol M, Otero Candelera R, Caballero Eraso C. Biomarkers of immunothrombosis and sleep-related hypoxaemia are independently associated with persistent perfusion defects 3 months after pulmonary embolism. Poster presented at: European Respiratory Society (ERS) International Congress 2026; Barcelona, Spain.

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