REPORT FROM

ERS Congress 2025

Amsterdam

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Lung health in a global perspective

Chiesi Nordic attended the international ERS congress in Amsterdam 2025. We took notes and are happy to share some of our favourite sessions with you!

The theme of the year was Respiratory health around the world. Every third ERS member is based outside of Europe, making this international congress an excellent platform for global information change.

Some of the main challenges impacting respiratory health are also global challenges requiring global efforts: Fighting climate change and air pollution. Only 1% of the world's population breathe air clean enough to comply with the WHO guideline limits, and the poorest countries suffer from the highest exposures (see www.who.int/health-topics/air-pollution).

Many drivers of air pollution are also sources of greenhouse gas emissions. Improving air quality is therefore a win-win strategy for both climate and health and is particularly important for respiratory health, present and future.

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17551-Dec-2025

Fighting global threats to respiratory health

REFLECTIONS FROM HOT TOPIC SESSIONS

Summary: Many preventable respiratory hazards are both a cause and a consequence of climate change. Fine particulate matter, especially from wildfires and household cooking and heating, poses serious health risks that disproportionally affects vulnerable populations. To protect public health, targeted actions to reduce emissions and improve air quality must be taken – urgently.

Most diseases are modifiable and preventable

Non-communicable diseases are the main cause of death and disability in Europe, yet most are preventable. WHO Europe reports that 60% of these cases can be avoided by minimizing risk factors and improving public health measures. One in four adults in Europe uses tobacco,[1] highlighting the need for better tobacco control and investing in cessation efforts.

Air pollution is deadly and drives disease

According to IHME 2021 data, air pollution is the world's second leading risk factor for death.[2] It increases risk of illness and death in COPD, diabetes, lung cancer, stroke, and heart disease and has been associated with increased risk of Parkinson's, Alzheimer's, ADHD, autism, dementia, and breast cancer.

Why particulate matter matters

Long-term exposure to fine particles (PM2.5) is particularly harmful as it crosses the lung barrier and enter the blood stream to reach multiple organs. A growing body of evidence also points out fine particulate matter (PM2·5) from wildfire smoke as considerably more harmful to human health than "regular" PM.[3] In 2025, wildfires have so far burned more than 1 million hectares in EU countries, the highest value ever recorded since 2006.[4] Fires emit massive amounts of particulate matter and CO_2 that travel across continents. Wildfires are a global concern: they are both a consequence of climate change, and a cause.

Clean Air – A Luxury?

Asia accounts for a third of global air pollution deaths,[2] mostly from coal and biomass use. There is a strong correlation between air pollution and poverty. Burning biomass (for cooking and heating) causes 3.2 million early deaths each year. Women, elderly and children have the highest exposures.[5]

Even in high income countries, indoor air quality is a social determinant of health in that low-income households are more likely to be exposed to poor indoor air quality due to mould (causing asthma[6]), dust mite, cockroaches.

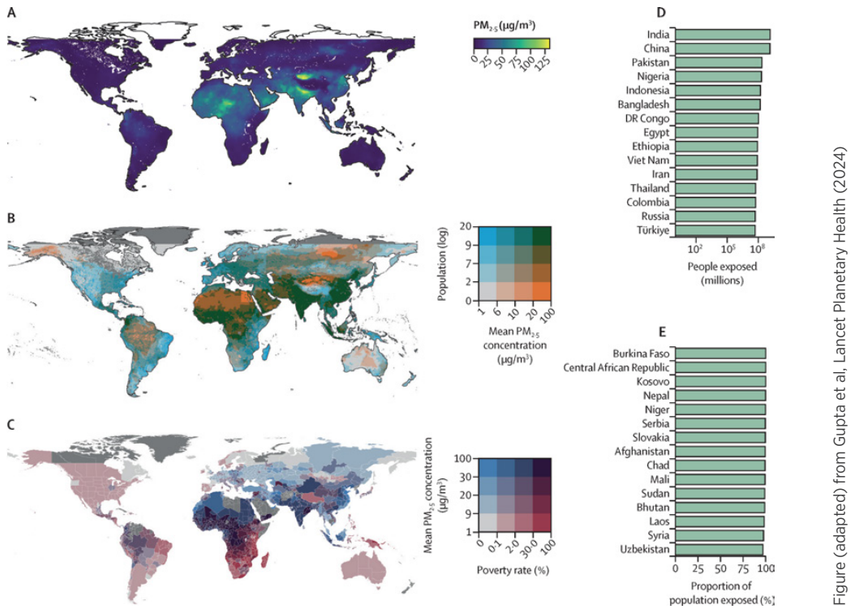

Figure (adapted) from Gupta et al, Lancet Planetary Health (2024)

We can tackle respiratory hazards

Smoking cessation is effective—it prevents lung cancer, improves respiratory health, and reduces societal costs.

Vaccination—influenza vaccines lower overall mortality risk.

Improved indoor climate – including schools and workplaces - risks from mould, mites, and particulate matter should be minimized. For children with sensitivities, measures like allergen-proof covers, HEPA filters, air purifiers, pest control, and mould management help reduce asthma symptoms.

Smoke-free laws—bans on indoor smoking are associated with fewer paediatric and adult asthma hospitalizations.

Traffic reduction—initiatives like Stockholm's congestion pricing and London's low emission zones have cut pollution and respiratory events.

The climate and respiratory health interplay

Many preventable respiratory hazards are both a cause and a consequence of climate change. Mary Rice from Harvard University, USA talked about scientific foundations for respiratory health and reminded us that fighting climate change is an important part of reducing burden of disease from respiratory diseases. Focusing on health equity will bring the greatest wins: prioritize highest-risk patients and communities with the highest burden.

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Exposome and respiratory health

SPEAKERS: Ane Johannessen (Norway), Erol Gaillard (UK), Hsiao-Chi Chuang (Taiwan), Nikoleta Bizymi (Greece)

Summary: Lifelong exposure to environmental factors — especially air pollution — significantly affects the risk and progression of chronic lung diseases. Understanding these interactions is key for early intervention and personalized prevention strategies.

Chronic respiratory diseases like asthma and COPD are widespread non-communicable illnesses. Their risk is shaped by lifelong exposure to various environmental, or exposome, factors, which interact in complex ways. At ERS 2025, experts discussed how the exposome influences the onset and progression of chronic lung diseases.

Understanding exposome: a holistic view of environmental influences

Environmental exposures are, according to the exposome concept, categorized into three main domains[1]:

- General external exposome: climate, air pollution, societal changes and chemicals in our environment impacting populations.
- Specific external exposome influence individuals such as diet, water, physical activity and personal habits.
- Internal exposome reflects what happens inside our body biologically, such as DNA mutations and disruptions in cellular pathways.

Challenges in capturing the full picture

The complexity of measuring the exposome in its entirety, is key challenge mentioned by all speakers. Most studies focus on isolated exposures, which limits our understanding of how multiple factors interact. Some exposures may even impact offspring health before conception, highlighting the need for a life-course and even multigenerational perspective.[2]

Overcoming barriers in exposome research

Exposome research faces multiple challenges, such as variability in measurements across populations, temporal issues, heterogeneity in measurements and the practicality of capturing biomarkers over large populations. EXPLAIN-IT, an international interdisciplinary translational network, is working to address these challenges through interdisciplinary collaboration.

Epigenetics and critical windows of vulnerability

Environmental exposures can alter gene expression via epigenetic modifications, sometimes lasting across generations. Preconception and prenatal periods are especially sensitive, with exposures during these times impacting fetal development and long-term respiratory health.[3]

> *”Air pollution is, as made clear in this session, a modifiable exposure that must urgently be addressed”*

Air pollution- a modifiable and urgent risk factor

Air pollution is a significant modifiable exposome factor, contributing to both climate change and increasing respiratory disease risk. Studies consistently link air pollution to hospital admissions for children.[4] Pre-natal exposure lowers lung function in children mid childhood[5]. It affects respiratory health both directly and indirectly, as rising global temperatures lead to more thunderstorms triggering worsening of asthma.[6] More heavy rain periods may advance mould and fungi in our homes, impacting lung function.[7]

From research to action – a new paradigm in prevention

The exposome framework marks a shift in prevention of chronic lung diseases. Better understanding of exposures helps us identify populations at risk, enabling early intervention.

Air pollution is, as made clear in this session, a modifiable exposure that must urgently be addressed.

Interdisciplinary collaboration is key as it will help translate exposomic insights into practical healthcare strategies that, realized in clinical practice, will advance individualized care and disease prevention.

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Optimized inhaler use can improve asthma and COPD outcomes

SPEAKERS: Omar S. Usmani (UK)

Summary: Effective inhaler therapy hinges on matching the right device to the right patient—and ensuring proper technique through training. As sustainability goals evolve, clinical decisions must remain patient-centred to avoid unintended harm.

Poor inhalation technique still a barrier

Prof. Omar Usmani addressed inhalation technique for the tenth time at the ERS congress — an indicator of its enduring clinical relevance. Despite advancements in inhaler technology, improper technique remains a major barrier to effective treatment in asthma and COPD.

Patient factors versus device factors

When selecting inhalers, consider both the patient's ability to use the device (inspiratory effort, technique) and the device features (internal resistance, aerosol velocity). Pressurized Multi Dose Inhalers (pMDIs) are activated by the device, while Dry Powder Inhalers (DPIs) depend on the patient's inspiratory force.[1]

Hidden cost of inhaler errors

A systematic review revealed that only 12% of healthcare professionals could correctly demonstrate inhaler use to patients.[2] These errors are not trivial—critical inhaler errorsare strongly associated with poor disease control, increased exacerbations, and higher healthcare costs.[3]

What prescribers need to know about device resistance and inspiratory flow

Dry powder inhalers differ in resistance: high-resistance types need slow, steady inhalation, while low-resistance ones require quick, forceful breaths. Both resistance and flow affect powder activation. Studies show that using multiple inhaler types with different techniques can cause confusion and worsen outcomes in asthma and COPD patients.[4,5]

> *"A systematic review revealed that **only 12%** of healthcare professionals could correctly demonstrate inhaler use to patients.[2]"*

A 2022 study found that 29% of patients did not achieve adequate peak inspiratory flow (PIF) with their prescribed device, mainly due to insufficient muscle strength or poor inhalation technique/device misuse. The highest rate of suboptimal PIF occurred in users of medium-to-low resistance DPIs.[6]

ACT algorithm: A stepwise approach to optimal inhaler use

ACT – Assess, Choose, Train is an inhaler algorithm introduced recently in the ERS handbook[10] to support clinical decision-making, breaking the decision-making into three steps:

Assess
Evaluate the patient's ability to perform the correct inhalation maneuver (e.g., slow and steady for pMDIs; quick and deep for DPIs).

Choose
Select an inhaler based on the patient's capability, ideally considering environmental impact (e.g., low-carbon pMDIs or DPIs).

Train
Provide education through face-to-face sessions or instructional videos to reinforce proper technique.

(Re-) consider inhaler choice when discharging from hospitalization

Patients discharged after exacerbations often have weakened respiratory muscles. In such cases, pMDIs or soft-mist inhalers—which do not require forceful inhalation—are preferable. This approach may reduce 30-day readmission rates.[7]

Sustainabilty in practice – green goals and patient safety

Balancing greenhouse gas reduction with patient safety is essential. A US study of 260,268 patients found that switching from pMDIs to DPIs led to a 5–24% rise in emergency visits and higher hospitalization rates for respiratory and pneumonia conditions.[8]

Switching strategies must never be at the expense of patient safety.

Inhaled volume – a digital biomarker

Prof. Usmani highlighted that inhaled volume could serve as a digital biomarker for predicting chronic respiratory disease outcomes.[9] Clinicians were also advised to ensure patients fully exhale before inhaling for optimal drug delivery.

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Do patients know enough to take control of their disease?

SPEAKERS: Maria Granados Santiago (Spain)

Summary: Health literacy is the ability to understand health. In a Spanish cross-sectional study, it was found that the more the patient knew about COPD, the lower the burden of disease and higher the quality of life.

Disease knowledge – linked to less disease burden

A Spanish cross-sectional study evaluated whether dyspnea, quality of life, and proper inhaler use correlate with patients' knowledge of COPD. Upon hospital admission, participants were assessed for dyspnea, inhaler skills, disease impact on autonomy, quality of life, and COPD knowledge, then categorized by understanding: superficial or comprehensive (COPD-Q score ≥8).

Invest in patient education

Patients with greater knowledge had significantly fewer symptoms, rated quality of life higher and used their inhalers more effectively. These findings underline that interventions to improve the patient's knowledge about their disease, such as providing patient education, are an important part of optimized disease management.

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Body, mind and spirit: lung conditions are not just somatic

PANEL DISCUSSION WITH CLINCAL EXPERTS: Psychologists Marieke Verklej (Netherlands), Liz Sted (UK), and Pulmonologist Anders Løkke (Denmark) and patient representatives

Summary: Though living with a chronic lung disease puts a mental toll on patients, not much mental support is available. A patient-centred, holistic approach that addresses both mental and physical health is essential for effective management of chronic lung conditions, the international panel concluded.

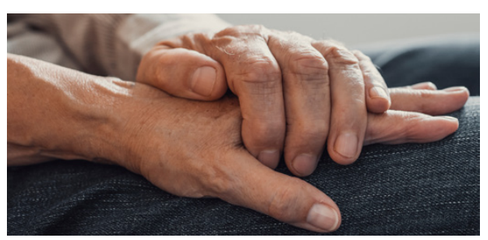
Psychological burden of lung disease

Loss of control, lifestyle changes, isolation and stigma are profound psychogical challenges that often follow a diagnosis. More than 20% experience loneliness, anxiety and depression, yet, fewer than 1% of lung patients receive phsychological support, according to Anders Løkke.

With 408 million people living with lung diseases worldwide, the economic burden is immense – €1.4 trillion, measured in disability-adjusted life years (DALYs). Early psychological support can reduce hospital admissions, maintain patients' ability to work, and reduce disease progression.[1]

Key Recommendations

- Screen all lung disease patients early for mental health issues.
- Include psychological support in standard treatment.
- Train healthcare professionals on mental health.
- Provide support groups and self-help options for patients and families.
- Involve relatives in care.



> *"Psychological support is a cost-effective investment in patients' overall health — cheaper than many advanced medical treatments."*
>
> Professor Anders Løkke

A more holistic approach to health is needed, where care is patient-centred, supporting both physical and psychological aspects of living with a chronic disease. Cross-disciplinary collaboration is key. Efforts must be done to break down stigma.

Conclusions

Integrating psychological with physical care improves quality of life and supports sustainable healthcare. The panel stressed that mental and physical health are inseparable, and investing in mental health for lung disease patients is both necessary and cost-effective.

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Finding disease early
– a chance to precede and limit injury

SPEAKERS: Sonia Stajonevic (USA), Shawn Aaron (USA), Yunus Çolak (DK)

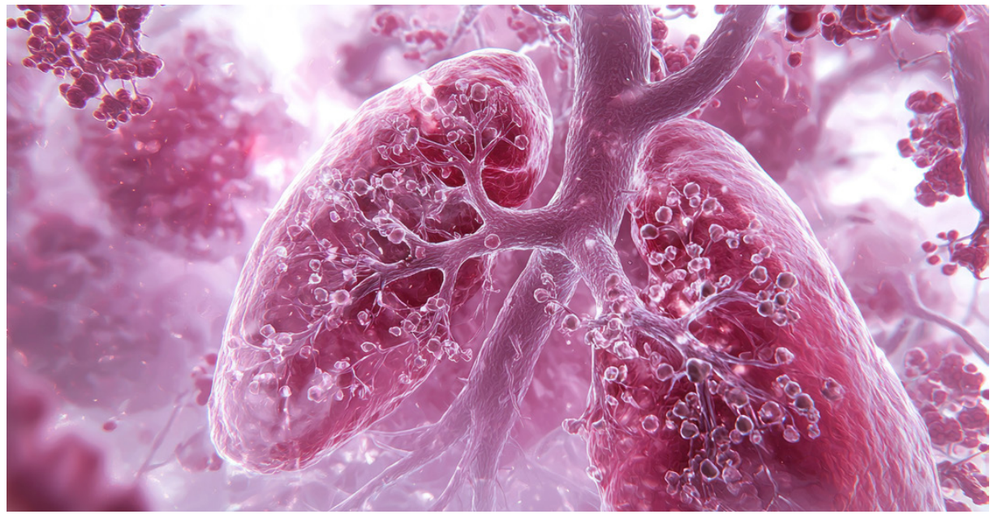
Summary: Some get a bad start in terms of lung development; others are left a long time in the dark about their own lung health. Even a small amount of tobacco links to disease and reduced life expectancy - there is no safe level of exposure.

Preterm birth - lifelong vulnerability

Preterm birth and disruption of lung development are risk factors for early-onset COPD. As preterm lungs are less complex, less vascularized, and differ molecularly and immunologically from normal lungs, prematurely born may also be more susceptible to other diseases.[1]

Nordic registry data shows that greater prematurity increases disease risk, but the impact of early-life treatments remains uncertain. Patients are typically grouped by bronchopulmonary dysplasia (BPD) status, though it is a proxy, influenced by indication bias. Data interpretation is complicated by inconsistent definitions of BPD across regions and time.

Prof. Stajonevic noted that outcome studies often exclude non-survivors of neonatal critical care, which can skew our understanding of disease burden. Ultimately, individuals born preterm have more vulnerable lungs and require attentive care throughout life.



Finding the undiagnosed: Screening versus case finding

It is believed that up to 70% of people eligible for a COPD diagnosis are undiagnosed. Professor Aaron outlined two methods to identify these individuals: broad screening or targeted case finding in high-risk groups, exhibiting symptoms, or having known exposure. Online questionnaires can assess the likelihood of COPD or asthma and encourage users to seek evaluation.

Two thirds already progressed

Of the cases detected through these methods, 2/3 had sadly already progressed to moderate or severe disease stages. Once identified, guideline-based treatment led to better CAT and St. George´s Respiratory Questionnaire outcomes, compared to standard care.[2]

Tobacco Exposure: No safe level

Yunus Çolak, using data from the Copenhagen City Heart Study, found that over 20% of people with less than 10 pack-years of tobacco exposure developed COPD.[4] Even low tobacco exposure lowers life expectancy by about five years, and greater exposure raises the risk. There is no safe number of ”pack-years” for low COPD risk or good outcomes.

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Role of biological sex in asthma and COPD

SPEAKERS: Dawn Newcomb (USA) and Martin Van Den Berge (Netherlands)

Summary: Women are more affected by asthma in adulthood and increasingly by COPD. Sex hormones influence immune responses through distinct receptor pathways. Still, clinical decisions should be guided by clinical phenotyping and identifying treatable and not sex alone.

Prevalence patterns differ

Both asthma and COPD are complex diseases influenced by environmental, genetic and epigenetic factors. Though they may present differently in men and women, current guidelines do not addres this.[1] Before puberty, prevalence of asthma is higher in boys than girls , but in adulthood, more women suffer from asthma, including severe asthma. COPD prevalence is rising in women, who are also more susceptible to harm from smoking.[2]

Biological mechanisms behind sex differences in asthma

Dr Dawn Newcomb highlighted the impact of sex hormones on immune response in asthma. Females more often develop autoimmune diseases and respond better to vaccines, while males are at higher risk for Hepatitis B, C, and non-reproductive cancers. Notably, women show higher CD4+ T-cell activation and proliferation, which drives inflammation in asthma, although the direct effect of sex hormones on these cells remains unclear.

Sex hormones as modulators of immune response

Dr Newcomb presented research conducted with her colleagues, showing that both oestrogen and androgen receptors are involved. Oestrogen signaling through ER-a increases eosinophil and neutrophil infiltration, while signaling through ER-b decreases eosinophil infiltration. Androgens on the other hand, such as testosterone, signaling through AR, decrease eosinophil and infiltration.[4]

In addition, in a series of elaborate in vivo and in vitro experiments, it was shown that females have a higher metabolic protein expression in Th17 cells, and that androgens, signaling via AR, decreased Th17 cell differentiation and restricted airway inflammation.[5]

Clinical phenotyping and potential sex differences for treatment of asthma and COPD

The ATLANTIS study, a large multinational observational study on asthma, attempted to also answer questions around the importance of sex differences and clinical phenotyping.[6] Dr. Maarten van den Berge presented findings from this study, showing that women experienced more exacerbations, and reported more symptoms than men with similar lung function. In addition, a higher airway resistance, as measured by impulse oscillometry, was seen in women, possibly explained by the airway size, i.e. because the resistance is higher in smaller lungs. Therefore, his suggestion was to account and adjust for sex when performing oscillometry. Male patients, on the other hand, were found to have a more severe airflow obstruction, and a higher prevalence of PAL.

Inflammatory biomarkers

For the majority of the inflammatory markers, i.e. FeNO, and blood and sputum eosenophils, there were no significant sex differences. However, women with asthma had significantly higher blood neutrophils counts. Treatment patterns were similar for men and women.

Clinical phenotyping and treatable traits

In summary, sex differences are present in respiratory diseases, particularly in asthma, and should be considered in the clinical setting. Nevertheless, the picture is complicated, with many factors involved (environmental, genetic, epigenetic) and decision making should be guided by clinical phenotyping and identifying treatable traits and not by sex alone.

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Redefining remission in severe asthma

SPEAKER: Celeste Michala Porsbjerg (Denmark)

Summary: A new framework published in a new Lancet Respiratory Medicine series paper redefines remission in severe asthma by focusing on disease activity, not damage. Early, targeted treatment during the "at-risk asthma" phase may prevent lasting harm.

Introducing "At-Risk Asthma"

The authors introduce the concept of "at-risk asthma" – a phase marked by high disease activity and early remodelling, where timely treatment can still alter the disease trajectory. By intervening before irreversible damage occurs, clinicians may improve long-term outcomes and prevent progression to fixed airway obstruction and comorbidity-driven symptoms.

> *"Timely, mechanism-based intervention in early disease stages offers the best chance for remission"*

Key insight from the paper:

Four central domains underpin asthma pathology:

In addition to airway hyper-responsiveness and structural remodelling, two new terms are introduced: Airway immune hyper-responsiveness and immune remodelling.

A domain-based assessment strategy can help clinicians distinguish symptoms from active disease versus permanent damage or comorbidities, supporting more personalised care.

Timely, mechanism-based intervention in early disease stages offers the best chance for remission, while later structural changes are often resistant to therapy.

Ontime treatment shape long term outcomes

This biologically grounded framework reframes remission, not as the absence of symptoms, but as the suppression of active disease biology. It also underscores the central role of on-time treatment decisions in shaping long-term disease outcomes.

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COPD and comorbidities: 8 new leads

CLINICAL SESSION: New data from recent studies

Take-aways: These studies underscore the multifaceted nature of COPD, emphasizing the importance of metabolic markers, socioeconomic context, and comorbidity management. As COPD patients live longer, understanding and addressing these interconnected risks will be critical to improving outcomes and tailoring therapies.

Global leading researchers —strongly represented by the Nordics—presented compelling findings on chronic obstructive pulmonary disease (COPD) and its complex interplay with cardiovascular, metabolic, genetic, and socioeconomic factors.

Comorbid conditions—such as cardiovascular disease, metabolic syndrome, osteoporosis, depression, anxiety, and lung cancer—can significantly impact the clinical course, prognosis, and quality of life of individuals with COPD. Recognizing how COPD and its comorbidities interact is key to creating effective, holistic treatment plans.

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01. Genetics and Thrombosis: α1 antitrypsin deficiency under the microscope

(Sine Woss Winther, Denmark)

α1-Antitrypsin deficiency is a genetic disorder that causes emphysema as well as liver cirrhosis. However, α1-antitrypsin deficiency also affects the coagulation cascade and may increase the risk of blood clot formation. A Danish cohort study revealed that individuals with α1-antitrypsin deficiency have higher risks of pulmonary embolism, deep vein thrombosis, and venous thromboembolism, even after adjusting for known confounders such as sex, COPD, and age.[3] Mortality from pulmonary embolism was also elevated in individuals with α1-antitrypsin deficiency. While causality cannot be confirmed, the deficiency may disrupt thrombin regulation, raising risk of clot formation.

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02. Metabolic Markers and Lung Function: The TyG Index

(Mohammad Azizzadeh, Austria)

The triglyceride-glucose (TyG) index is a marker of metabolic dysfunction. Data from the LEAD study highlighted the TyG index as a potential marker for restrictive lung impairment. At baseline, TyG was positively associated with age and was consistently higher in males than in females. Additionally, TyG levels were elevated in individuals with impaired lung function, as assessed by spirometry. Cross-sectional analysis showed that TyG was associated with a higher risk of impaired lung function. However, longitudinal data showed only borderline significance, prompting calls for further research. The TyG index may serve as an indicative marker of impaired lung function, similar to other indexes of metabolic dysfunction.

Ref: Khan SH, Sobia F, Niazi NK, Manzoor SM, Fazal N, Ahmad F. Metabolic clustering of risk factors: evaluation of Triglyceride-glucose index (TyG index) for evaluation of insulin resistance. Diabetol Metab Syndr. 2018 Dec;10(1):74.

03. Silent cardiovascular risk in Small Airways Obstruction

(Sam Bartlett-Pestell, UK)

Ther is no gold standard test for isolated small airway obstruction (SAO). Typically, Forced Expiratory Flow between 25% and 75% of forced vital capacity (FEF_{25-75}), the ratio of Forced Expiratory Flow in 3 seconds to forced vital capacity (FEV_3/FVC), or the ratio of FEV_3 to FEV_6 (FEV_3/FEV_6), combined with a normal FEV_1/FVC, is used to identify isolated SAO.[1]

Individuals with isolated SAO report higher scores on the St. George's Respiratory Questionnaire, experience more breathlessness, and have more gas trapping compared to individuals without isolated SAO. Isolated SAO is associated with increased mortality,[2] including cardiovascular mortality.[3]

However, the longitudinal association between SAO and cardiovascular disease is unknown, and this was the focus of Dr. Sam Bartlett-Pestell's study. In the study, isolated SAO was defined as FEV_3/FEV_6 below the lower limit of normal, combined with an FEV_1/FEV_6 above the lower limit of normal. Bartlett-Pestell emphasized that FEV_6 was used instead of FVC because FVC is likely underestimated in this population.

Using UK Biobank data, isolated SAO were linked to a 5% increased risk of cardiovascular disease, particularly in women and ever-smokers.[4] These findings suggest that SAO may serve as an early warning sign for cardiovascular issues.

Ref: **1.** Hogg JC, Paré PD, Hackett TL. The Contribution of Small Airway Obstruction to the Pathogenesis of Chronic Obstructive Pulmonary Disease. Physiological Reviews. 2017 Apr;97(2):529–52. **2.** Quintero Santofimio V, Knox-Brown B, Potts J, er al. Small Airways Obstruction and Mortality. CHEST. 2024 Oct;166(4):712–20. **3.** Knox-Brown B, Patel J, Potts J, et al. The association of spirometric small airways obstruction with respiratory symptoms, cardiometabolic diseases, and quality of life: results from the Burden of Obstructive Lung Disease (BOLD) study. Respir Res. 2023 May 23;24(1):137. **4.** Quintero Santofimio, V. et al. "Small Airways Obstruction and Mortality: Findings From the UK Biobank." Chest vol. 166,4 (2024): 712-720.

04. Cholesterol Paradox in COPD

(Yu Liu, China)

A NHANES-based study revealed a dual role for the non-HDL/HDL cholesterol ratio (NHHR): while a higher NHHR increased the risk of developing COPD, it was paradoxically associated with lower all-cause mortality among COPD patients. This finding underscores the complexity of lipid metabolism in chronic lung disease.

Ref: Zhong Y, Zhou K, Li S, et al.. Association Between the Non-High-Density Lipoprotein Cholesterol-to-High-Density Lipoprotein Cholesterol Ratio (NHHR) and Mortality in Patients with COPD: Evidence From the NHANES 1999-2018. Int J Chron Obstruct Pulmon Dis. 2025 Mar 28;20:857-868

05. Socioeconomic disparities persist – even with universal healthcare

(Sigrid Anna Aalberg Vikjord, Norway)

Socioeconomic status (SES) is associated with COPD risk and outcomes.[1] However, its impact on pre-COPD states with respiratory symptoms remains unclear. Norwegian researchers used data from the HUNT study to investigate lung function and mortality in relation to SES among COPD patients, individuals with pre-COPD, and the general population.

The data showed that lower SES correlates with reduced lung function and higher mortality across all groups, including those with pre-COPD symptoms. Additionally, people with low SES had approximately 3.4 years of lung function loss at age 45 and about 7 additional years at age 75 compared to individuals with higher education.

Interestingly, there were no differences in the rate of decline over time. Furthermore, a socioeconomic gradient was observed in mortality rates, which was more pronounced in the COPD population.

In conclusion, the study demonstrated a clear socioeconomic gradient in lung function levels but no difference in the speed of decline over time. Moreover, the impact of SES on mortality was more significant in individuals with COPD than in those with pre-COPD or in the general population. These findings call for targeted public health interventions and improved health literacy, even in high-income countries.

Ref: Lowe KE, Make BJ, Crapo JD, et al. Association of low income with pulmonary disease progression in smokers with and without chronic obstructive pulmonary disease. ERJ Open Res. 2018 Oct;4(4):00069–2018.

06. Lung Function Trajectories and Mortality Risk

(Helena Backman, Sweden)

Using longitudinal spirometry data, Helene Backman presented findings from a study investigating the trajectories of FEV_1 and FEV_1/VC among adults with airway obstruction and their association with all-cause mortality.

The data, derived from a clinical sub-cohort of the Obstructive Lung Diseases in Northern Sweden (OLIN) studies, revealed that rapidly declining FEV_1 or starting with low FEV_1 trajectories are associated with increased mortality. Smoking and obesity emerged as key risk factors, reinforcing the importance of early intervention and lifestyle modification.

Ref: Backman H, Blomberg A, Lundquist A, et al. Lung Function Trajectories and Associated Mortality among Adults with and without Airway Obstruction. Am J Respir Crit Care Med. 2023;208(10):1063-1074. doi:10.1164/rccm.202211-2166OC

07. Multimorbidity: COPD and Rheumatoid Arthritis – A Deadly Duo

(Olivia Murrin, UK)

COPD often co-occurs with rheumatoid arthritis (RA) and other inflammatory diseases.

Data from the UK highlighted that patients with both COPD and RA experience the worst outcomes in terms of mortality and hospitalizations. Genetic links and shared risk factors suggest a need for integrated care strategies and early identification of at-risk individuals.

Ref: Murrin O, Mounier N, Voller B, et al. A systematic analysis of the contribution of genetics to multimorbidity and comparisons with primary care data. eBioMedicine. 2025 Mar;113:105584.

08. Troponin Levels in COPD: A Cardiovascular Signal?

(Jonas Eriksson Ström, Sweden)

There is substantial evidence of elevated troponin levels in stable COPD patients without known cardiovascular disease (CVD). The study, presented by Jonas Eriksson Ström, aimed to investigate the association between troponin I and COPD, and to further explore the influence of coronary artery disease (CAD).

Contrary to previous studies, Swedish SCAPIS data found no association between troponin I levels and COPD in patients without known CVD. Researchers suggest that elevated troponin may reflect undiagnosed heart conditions rather than COPD itself, particularly in mild to moderate cases.

Ref: Nilsson U, Van Fleteren LEGW. Troponin as a biomarker for mortality in stable COPD. Eur Respir J. 2020 Feb;55(2):1902447.

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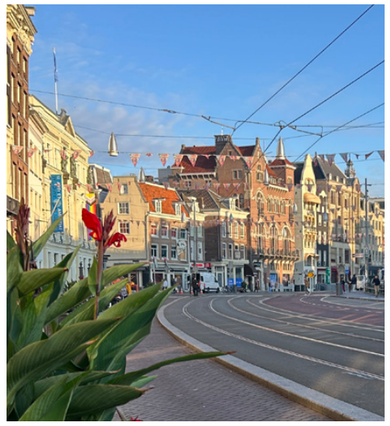
Chiesi Nordic



On our website for healthcare professionals, you can find more scientific summaries and updates on lung health. Scan this QRcode to visit chiesipro.no

EKSTRAFINE PARTIKLER[1]

Chiesi

Hele dosen avgitt – bekreftes med inhalasjonsklikket[1,2]

Trimbow® NEXThaler®

beklometasondipropionat/
formoterolfumaratdihydrat/
glykopyrronium

INHALASJONSKLIKKET

Hele dosen avgitt – uten risting[3,4]

Trimbow®

beklometasondipropionat/
formoterolfumaratdihydrat/
glykopyrronium

Trimbow (beklometasondipropionat, formoterolfumaratdihydrat, glykopyrronium)
Inhalasjonsaerosol 87 µg/5 µg/9 µg og 172 µg/5 µg/9 µg
Inhalasjonspulver 88 µg/5 µg/9 µg

Indikasjoner: ***Kronisk obstruktiv lungesykdom (kols):*** *Inhalasjonsaersol 87 µg/5 µg/9 µg og inhalasjonspulver 88 µg/5 µg/9 µg:* Vedlikeholdsbehandling hos voksne med moderat til alvorlig kols, som ikke er adekvat behandlet med en kombinasjon av et inhalert kortikosteroid og en langtidsvirkende ß$_2$-agonist eller en kombinasjon av en langtidsvirkende ß$_2$-agonist og en langtidsvirkende muskarinantagonist (for effekt på symptomkontroll og forebygging av eksaserbasjoner, se SPC pkt. 5.1.). ***Astma:*** *Inhalasjonsaersol 87 µg/5 µg/9 µg:* Vedlikeholdsbehandling hos voksne som ikke er adekvat kontrollert med en vedlikeholdskombinasjon av en langtidsvirkende ß$_2$-agonist og middels dose inhalert kortikosteroid, og som opplevde 1 eller flere astmaeksaserbasjoner i foregående år. *Inhalasjonsaersol 172 µg/5 µg/9 µg:* Vedlikeholdsbehandling hos voksne som ikke er adekvat kontrollert med en vedlikeholdskombinasjon av en langtidsvirkende ß$_2$-agonist og høy dose inhalert kortikosteroid, og som opplevde 1 eller flere astmaeksaserbasjoner i foregående år.

Pakninger og pris (AUP): *Inhalasjonsaerosol: 87 µg/5 µg/9 µg (inhalator):* 120 doser: kr 768.60, 3×120 doser: kr 2173.40. *172 µg/5 µg/9 µg (inhalator):* 120 doser: kr 768.60, 3x120 doser: kr 2233.30. *Inhalasjonspulver 88 µg/5 µg/9 µg (Nexthaler inhalator):* 1×120 doser: kr 767.80, 3×120 doser: 2162.90. **Refusjonsberettiget bruk:** *Inhalasjonsaerosol 87 µg/5 µg/9 µg:* Vedlikeholdsbehandling ved bronkialobstruksjon, iht. preparatomtale. *ICPC/ICD:* R95/J44: Kronisk obstruktiv lungesykdom/Annen kronisk obstruktiv lungesykdom. *ICPC/ICD:* R96/J45: Astma. *Vilkår:* Ingen spesifisert. *Inhalasjonsaerosol 172 µg/5 µg/9 µg:* Vedlikeholdsbehandling ved bronkialastma, iht. preparatomtale. *ICPC/ICD:* R96/J45: Astma. *Vilkår:* Ingen spesifisert. *Inhalasjonspulver 88 µg/5 µg/9 µg:* Vedlikeholdsbehandling ved kols, iht. preparatomtale. *ICPC/ICD:* R95/J44: Kronisk obstruktiv lungesykdom/Annen kronisk obstruktiv lungesykdom. *Vilkår:* Ingen spesifisert. **Reseptgruppe:** C.

Utvalgt sikkerhetsinformasjon

- Ikke indisert til behandling av akutt bronkospasme eller akutt sykdomseksaserbasjon.
- *Bivirkninger:* Hyppigst sett er dysfoni, oral candidose, muskelspasmer og munntørrhet.

For utfyllende informasjon om dosering, kontraindikasjoner, advarsler og forsiktighetsregler, interaksjoner og bivirkninger, se Trimbow SPC godkjent 04.11.2024.

Referanser: 1. SPC Trimbow inhalasjonspulver, https://www.legemiddelsok.no. **2.** Corradi M, Chrystyn H, Cosio BG et al. NEXThaler, an innovative dry powder inhaler delivering an extrafi ne fi xed combination of beclometasone and formoterol to treat large and small airways in asthma. Expert Opin Drug Deliv. 2014;11:1497–1506. **3.** SPC Trimbow Inhalasjons aerosol, https://www.legemiddelsok.no. **4.** Chierici V, Cavalieri L, Piraino A et al. Consequences of not shaking and shake-fi re delays on the emitted dose of some commercial solution and suspension pressurized metered dose inhalers. Expert Opin Drug Deliv 2020 Jul;17(7):1025-1039.

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