



Towards Personalised and Equitable Care for People with COPD

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EARLIER diagnosis, health equity, and precision medicine were among the key themes at this year's European Respiratory Society (ERS) Congress, with speakers highlighting opportunities to rethink how COPD is identified, assessed, and treated. Hilary Pinnock, University of Edinburgh, UK; Guy Brusselle, Ghent University Hospital, Belgium; Sanjay Ramakrishnan, The University of Western Australia, Nedlands, Australia; and Mireille C. Melles, Erasmus University Rotterdam, the Netherlands, explored how a more personalised approach could address persistent gaps across the COPD care pathway.

THE EQUITY GAP IN COPD

Pinnock began by defining health equity as everyone having a fair and just opportunity to attain the highest level of health within their reach. However, avoidable differences in health and its determinants continue to affect people living with COPD, with socioeconomic circumstances and environmental exposures contributing to an uneven disease burden.

In 2019, 212.3 million cases of COPD were reported globally, with the disease accounting for 3.3 million deaths.¹ The burden is unevenly distributed: age-standardised prevalence per 100,000 population was highest in Denmark

(4,299.5), Myanmar (3,963.7), and Belgium (3,927.7), while age-standardised death rates per 100,000 were highest in Nepal (182.5) and lowest in Japan (7.4).¹ The largest contributors to COPD-related disability-adjusted life years were smoking (46.0%), ambient particulate matter pollution (20.7%), and occupational exposure to particulate matter, gases, and fumes (15.6%).¹

Furthermore, socioeconomic deprivation can compound this burden. For example, a 2012 analysis of Scottish primary care data from 2007 found that people with COPD, coronary heart disease, diabetes, or cancer generally had more comorbidities if they lived in the most deprived areas than in the most affluent ones.² Environmental exposures can also contribute to geographical inequalities. In 2025, the WHO Regional Office for Europe (WHO/Europe) and ERS published the first WHO European report on chronic respiratory diseases and health equity, highlighting the impact of ambient and household air pollution on COPD mortality, a marked disparity across Europe attributed to differences in pollution.³

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These disparities could become more pronounced as the global burden of COPD increases. Predictive modelling based on historical data estimates that the number of people aged 25 years or older living with COPD could increase by 23% between 2020–2050, approaching 600 million.⁴ Moreover, the disparity in COPD burden between low- and middle-income and high-income countries is also projected to widen.⁴ These points highlight that improving equity is not simply about providing the same care to everyone, it also requires identifying where gaps in diagnosis and treatment arise.

include unequal access to healthcare, inadequate investigation and referral of respiratory symptoms, limited specialist training, and restricted access to diagnostic testing such as spirometry, particularly in low- and middle-income settings.⁵

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EARLY DETECTION

Brusselle spotlighted how earlier diagnosis is a key part of addressing these gaps. Up to 70% of adults with COPD remain undiagnosed, with the problem particularly pronounced in low- and middle-income countries.⁵ These patients experience more exacerbations, a greater symptom burden, poorer quality of life, and a higher risk of death, making undiagnosed COPD a substantial clinical and societal burden.⁵ Bruselle explained that reasons for underdiagnosis span the patient, healthcare provider, and health-system levels. These

Although screening might appear to offer a solution, Brusselle noted that the evidence does not currently support mass or routine screening of asymptomatic adults. There is no direct trial evidence that mass or routine screening of asymptomatic adults improves health-related quality of life, morbidity, or overall mortality.⁶ This makes it an expensive approach with a relatively low yield.⁶ Instead, he advocated for active, opportunistic case-finding, which involves proactively searching for people at risk of COPD, such as those with recurrent chest infections, tobacco smoke exposure, or unexplained respiratory symptoms. This approach could provide

a more targeted route to earlier diagnosis while avoiding the resource demands of population-wide screening.

Earlier identification also creates an opportunity to move away from treating COPD as a single clinical entity. Brusselle argued for personalised, precision medicine that considers patients' aetiologies, phenotypes, and endotypes, particularly when differences in access to care and the needs of marginalised populations

are taken into account. "We should base treatment not on clinical labels, but on the biological mechanism."

One example is the identification of Type 2-high versus Type 2-low inflammation using biomarkers such as blood eosinophils and fractional exhaled nitric oxide. Where evidence of Type 2 inflammation is present, patients may be more likely to benefit from an inhaled corticosteroid (ICS) alongside long-acting β 2-agonist (LABA) plus long-acting muscarinic antagonist therapy.⁷

PRECISION THERAPY IN COPD

The potential to use biomarkers to guide treatment was developed further by Ramakrishnan, who opened with the assertion that "precision therapy is equitable therapy." In stable COPD, treatment has two broad goals: reducing symptoms and reducing future risk by preventing exacerbations and disease progression. Blood eosinophil count (BEC) is one biomarker that can help inform the latter. Higher eosinophil counts have been associated with a greater exacerbation risk and, in some studies, faster lung-function decline.^{8,9} Importantly, BEC can also help identify patients more likely to benefit from ICS, providing a means of bringing greater precision to corticosteroid prescribing.

Ramakrishnan also highlighted that BEC is dynamic biomarker rather than a fixed patient characteristic, and tracking how it changes may help to explain the varied response to ICS in patients with COPD. In a 2024 post-hoc analysis of the FLAME trial, higher BEC off ICS, higher BEC on ICS, and significant BEC suppression during ICS treatment each corresponded to a greater response to LABA plus ICS than LABA plus long-acting muscarinic antagonist in terms of exacerbation rate and time to first exacerbation.¹⁰

The emergence of biologic therapies has further expanded the potential for biology-led treatment. Ramakrishnan discussed two biologics approved in the EU for eosinophilic COPD: dupilumab, an antibody targeting the IL-4 receptor α , and mepolizumab, an



antibody targeting IL-5. However, precision therapy does not simply mean ordering more tests. Rather, it requires knowing which clinical question a test is intended to answer and using the result to guide an appropriate treatment decision.

DEFINING SEVERE COPD

A further challenge is determining which patients should be considered for advanced treatment in the first place. Melles highlighted that, although international guidelines assess several dimensions of COPD severity, there is currently no formal definition of severe COPD. A standardised definition could help to identify patients who need further evaluation and support more consistent selection for advanced treatments. It could also help prevent undertreatment, facilitate timely advance care planning, and inform policy and reimbursement decisions.

To address this long-standing gap, Melles described an ongoing modified Delphi study aiming to develop a consensus definition and standardised assessment for severe COPD. She emphasised that that implementation cannot simply involve applying a

single framework uniformly across all healthcare systems. Global implementation would require local adaptation, including of diagnosis and assessment in low- and middle-income countries, and that future updates may be needed as new biomarkers and diagnostic tests emerge.

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IN SUMMARY

Taken together, the presentations outlined a COPD care pathway that begins with identifying people earlier and more equitably, then moves towards treatment informed by underlying biology and clearer definitions of disease severity. The challenge will be translating these advances into routine practice across very different healthcare settings. Expanding access to diagnostic testing, biomarkers, and newer therapies could be an important part of ensuring that increasingly personalised COPD care does

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