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Indoor Air Pollution and Lung Function Decline
"Should I Stay or Should I Go?"

We spend most of our time indoors, and we will likely do so more often as outdoor extreme weather events increase, driven by global climate change. The evidence that outdoor air pollution adversely affects respiratory health is compelling (1), but is indoor air safer? The World Health Organization reports that, each year, 3.2 million deaths are attributable to indoor air pollution (IAP) caused by the incomplete combustion of solid fuels and kerosene used for cooking. This issue affects primarily people in low- and middle-income countries and is likely driven by socioeconomic status (2). However, knowledge gaps remain, especially in high-income countries (HICs), regarding the broad range of indoor pollutants that can harm our lungs in the short and long term and how we can effectively prevent the associated health effects. This is particularly relevant for more susceptible individuals such as children, pregnant women, the elderly, and patients with chronic lung disease.

In this issue of the *Journal*, Hansel and colleagues (pp. 1042–1051) contributed to the evidence on the topic by studying the association between indoor particulate matter ⩽2.5 μm in aerodynamic diameter ($PM_{2.5}$) and nitrogen dioxide (NO_2) concentrations and annual lung function decline among current and former smokers with or without chronic obstructive pulmonary disease (COPD) followed longitudinally for 3 years (3). The authors used the SPIROMICS (Subpopulations and Intermediate Outcomes in COPD Study) Air Pollution Study, an ancillary study of SPIROMICS (4), a U.S.-based multicenter cohort study of ever-smokers (⩾20 pack-years) aged 40–80 years with or without COPD, defined as post-bronchodilator $FEV_1/FVC < 70\%$. Indoor home $PM_{2.5}$ and NO_2

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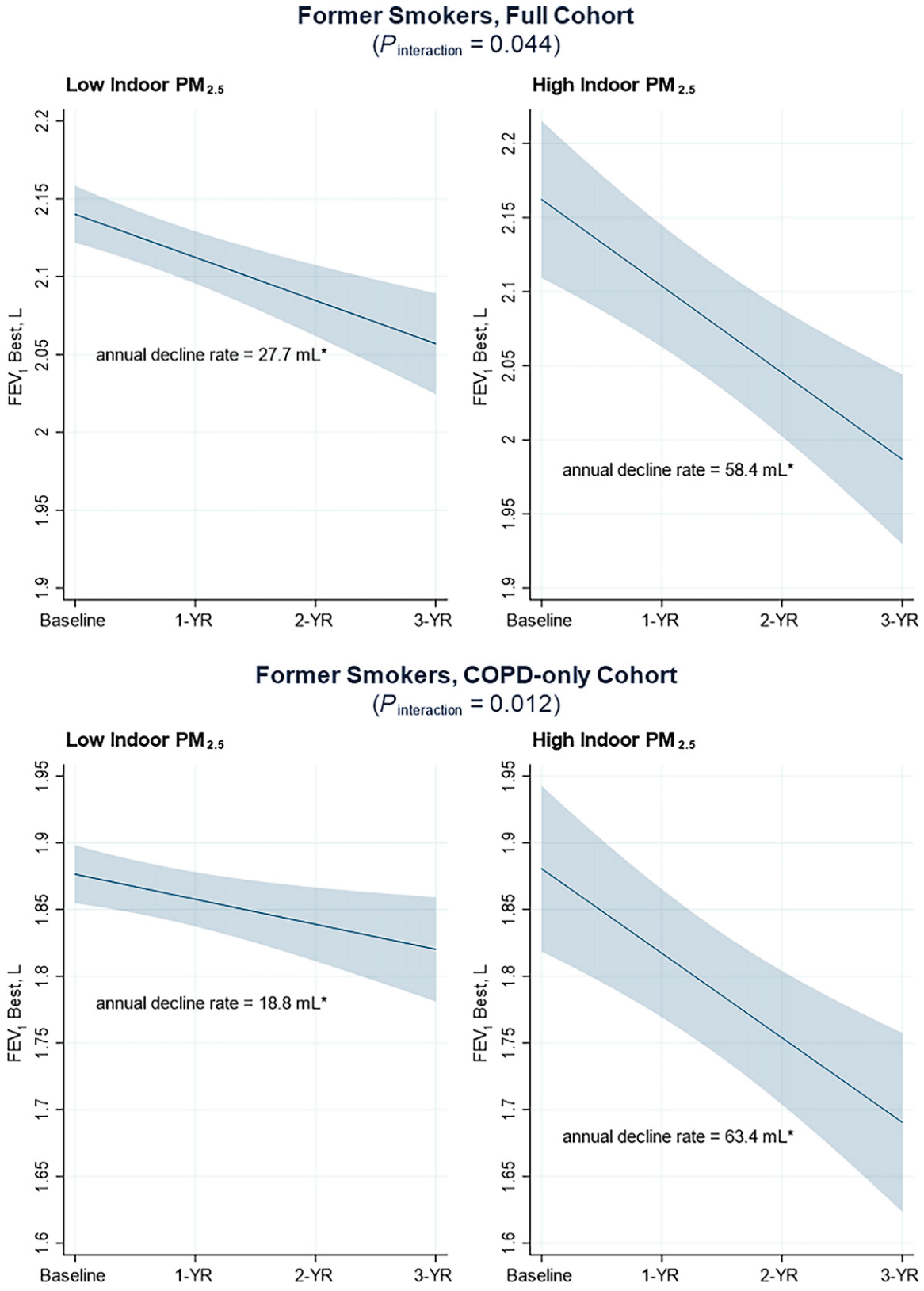

Figure 1. Among former smokers, the decline in FEV_1 is steeper for those residing in homes with higher indoor particulate matter ⩽2.5 μm in aerodynamic diameter ($PM_{2.5}$) concentration, allowing nonlinearity in FEV_1 decline. The chart compares the nonlinear FEV_1 progression over time for those residing in homes with an indoor $PM_{2.5}$ concentration at the 5th percentile (1.7 $\mu g/m^3$) versus the 95th percentile (31.3 $\mu g/m^3$). Reproduced by permission from Reference 3. * = annual decline rate was statistically significant; COPD = chronic obstructive pulmonary disease.

concentrations were estimated using a validated, individual-based prediction model (5). Among the 1,208 participants with complete exposure and spirometry data, the authors found that among former smokers, every 10 $\mu g/m^3$ increase in estimated indoor $PM_{2.5}$ was associated with an additional 10 ml/yr decline in FEV_1 ($P = 0.044$). The decline was steeper among individuals with greater exposure and also among patients with COPD only (Figure 1). Of note, among current smokers, FEV_1 decline did not differ by indoor $PM_{2.5}$. The results of indoor NO_2 concentration suggested similar but weaker trends.

The study findings are consistent with the literature supporting a significant effect of IAP on accelerated lung decline also among patients with COPD (6). The causal association is biologically plausible, given that IAP has been demonstrated not only to trigger an inflammatory response and oxidative stress but also to impair macrophage phagocytosis and surface adherence, to reduce bacterial and mucociliary clearance, and to disrupt the alveolar-capillary barrier in the lungs (7). Surprisingly, no effect of socioeconomic status was found: IAP in HICs might be driven by other factors, or the selected sample, mostly White and educated, may have prevented disentangling the effect of this important determinant (8).

There are several study strengths, including the longitudinal study design, the spirometry-based COPD definition, and the adjustment for relevant individual fixed and time-varying confounders, including outdoor $PM_{2.5}$ and NO_2 exposure. Of note, the authors considered coexposure (even if only self-reported) to occupational hazards, which are often (regrettably) forgotten in environmental epidemiological studies, despite the important associated respiratory health burden (9). Indeed, the steeper lung function decline among male former smokers than among women could be attributed to residual confounding by occupational exposures. Also, the authors tested the robustness of their findings in several sensitivity analyses (by sex, smoking, prebronchodilator spirometry, and COPD status).

Notwithstanding these considerations, there are limits that hamper the causal interpretation of this study's findings. The absence of never-smokers is a major weakness, given they would have been the ideal population to study the effect of IAP, excluding a residual confounding effect of tobacco smoking. The authors restricted the analyses for in-home secondhand smoke exposure, measured in former smokers as indoor nicotine concentration based on self-reported secondhand smoke questionnaires, but residual confounding cannot be ruled out. Indeed, the steeper decline among former versus current smokers that the authors interpret as lack of sufficiently high indoor $PM_{2.5}$ exposure to make the effect detectable among active smokers could instead be attributable to misclassification bias for smoking. Indoor $PM_{2.5}$ and NO_2 exposure was not measured, but only estimated. Also, the broad variety of sources of IAP are missing, such as woody biomass; organic dusts; pesticides; volatile organic compounds; carbon monoxide; and allergens from indoor pets, pests, and molds. Cooking and cleaning practices, known to be associated with respiratory health effects (6, 10), were not evaluated. Also, no information on house ventilation was reported.

To address the above shortcomings, future research studies should ideally perform large longitudinal analyses in smoke-free houses (including e-cigarettes) in both rural and urban settings with long-term personal exposure monitoring of a broad variety of indoor pollutants, taking into account house, behavioral, and occupational factors. Also, identification of specific underlying biological pathways would strengthen the hypothesis of direct adverse health effects from indoor air pollutants. Sadly, this could be unfeasible to achieve in large epidemiological studies.

Also, more randomized controlled trials (RCTs) aimed at understanding which intervention to lower IAP is effective and efficient, especially in the long term, are warranted, given that currently, the results are inconsistent. In an HIC, a recent RCT found that personal high-efficiency particulate indoor air cleaners among former smokers with COPD improved respiratory symptoms but not quality of life (11). In low- and middle-income countries, RCTs suggested that individual household-level interventions for IAP exposure reduction have limited benefits for respiratory health (12). The results of such studies would also inform evidence-based guidelines for healthcare professionals to guide personal mitigation strategies among their patients, especially those housebound (13).

So, is indoor air safer? The simple, unsatisfactory answer is, it depends. For symptomatic patients with chronic lung conditions on days with high outdoor pollution and extreme temperatures, to stay indoors might be the safest option. However, if their home is close to high-traffic roads with high IAP exposure and scarce ventilation, it could be safer to walk in a park. Pending sound evidence on the most cost-effective interventions to prevent IAP-related health effects, more than putting the responsibility on the individual (who often cannot decide where to live or afford expensive air purifiers), governments should implement policies at the community and national levels to reduce air pollution, both outdoor and indoor, such as smoking bans and smarter house and city planning (14). Given that no safe level of outdoor air pollution is known, and therefore that exposures should be as low as possible (15), the same precautionary principle should be followed for IAP, especially to protect the most susceptible individuals, who are often housebound because of their health conditions. ■

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Role of Community Health Workers in Chronic Obstructive Pulmonary Disease Care in Low- and Middle-Income Countries

Around 80% of the 8 billion global population live in low- and middle-income countries (LMICs). Adeloye and colleagues (1) estimated that out of the 391.9 million chronic obstructive pulmonary disease (COPD) cases worldwide, 315.5 million reside in LMICs. More than 86% of the 3.23 million annual COPD deaths and 86% of the 74.4 million annual COPD disability-adjusted life-years occur in LMICs (2). COPD is the third leading cause of death and the seventh leading cause of disability-adjusted life-years worldwide, mostly because of the COPD burden in LMICs (2). In high-income countries, tobacco smoking contributes to 70% of the COPD cases, but in LMICs nonsmoking risk factors, such as exposure to biomass smoke, ambient air pollution, occupational exposures, recurrent respiratory tract infections during childhood, past history of pulmonary tuberculosis, poverty, poor nutrition, and poorly treated asthma account for 60–70% of the COPD burden (3). More than 90% of the patients with COPD in LMICs remain undiagnosed, untreated, or wrongly treated (4). Limited resources, lack of availability of proper diagnostic tools, poor access to affordable drugs, and overstretched healthcare systems contribute to poor quality of care in the LMICs. Furthermore, poor doctor-to-patient ratios make healthcare delivery a significant challenge (5). Many LMICs are trying to address their healthcare delivery issues with the help of community health workers (CHWs).

CHWs are individuals who reside within the community and carry out functions related to healthcare delivery for the community without receiving any formal professional training. They provide culturally appropriate health education and information, help people get the care they need, give informal counseling on health behaviors, and advocate for individual and community health needs. They have the potential to offer limited healthcare services after receiving appropriate training. Because of their geographic and cultural proximity to the population they serve, CHWs are often described as vital bridges between health services and communities (6). Can CHWs play a potentially important role in COPD care in LMICs? In this issue of the *Journal*, Pollard and colleagues (pp. 1052–1062) have addressed this question through a pilot study conducted in resource-limited settings from three different continents (7).

GECo (Global Excellence in COPD Outcomes) is a multinational study that was aimed at investigating the diagnostic accuracy of case finding for COPD using a questionnaire with or without peak flow meter versus gold standard spirometry (GECo-1) (8) and the effectiveness of a multifaceted intervention comprising a self-directed COPD action plan delivered and supported by CHWs for the management of COPD exacerbations (GECo-2) (7). The study was conducted in Peru, Uganda, and Nepal and reported a COPD prevalence of 2.7%, 7.4%, and 18.2%, respectively. Among the 467 subjects with COPD whom they identified, 95.3% were unaware that they had COPD, 49.5% were never-smokers, 43% were exposed to biomass smoke daily, 10% had previous pulmonary tuberculosis, and 50% had moderate-to-severe COPD (old Global Initiative for Chronic Obstructive Lung Disease B to D).

From the GECo-1 study, the investigators recruited 239 subjects with moderate-to-severe COPD and randomized 119 to the control

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