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⊗ Air Pollution and Suppression of Lung Function Growth: A Triumph for Epidemiology

The link between exposure to air pollutants such as particulate matter (PM) and nitrogen dioxide (NO₂) and suppression of growth of lung function in children and young people is now used by policy-makers to justify potentially unpopular exposure-reduction initiatives. For example, when Sadiq Kahn, the mayor of London, introduced the Ultra Low Emission Zone (ULEZ) for central London, where penalty charges are £12.50 per day for the most polluting cars and £100 per day for polluting heavier vehicles, he emphasized that “every child

in London breathes toxic air daily, damaging their lung growth” (1). The current ULEZ was recognized by a C40 Cities Bloomberg Philanthropies Award in 2019, and it is proposed that, by October 2021, it will be extended to cover the area within London’s North and South Circular Roads—an enlargement that will bring over 640,000 vehicles into the zone, with approximately 135,000 vehicles currently liable for the charge. A major contributor to the evidence base for lung growth suppression and air pollution is the Southern California Children’s Health Study (CHS), a series of longitudinal assessments of lung function in children and young people. The seminal outputs of this study included a description of the association between background concentrations of air pollution in different communities and suppression of lung function growth (2), the independent effect of locally generated air pollution on lung function growth within communities (3), and the finding that improvement in air

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quality over time is associated with improvements in lung function growth (4). Thus, the CHS is truly an exemplar of the vital role of epidemiology in guiding public health policy. A potential way to extend data from studies such as the CHS is to ask the “what if” question. For example, if the ULEZ had been introduced 5 years ago and all diesel cars and vans below the current “Euro 6” standard had been banished from Greater London, what would have been the improvement in children’s lung function, and how many cases of pediatric asthma would have been avoided? The first part of this calculation, the effect of changes in vehicle mix on emissions, is relatively easy to calculate. For example, modeling done by researchers at King’s College London estimates that policies that would bring the proportion of diesel cars down to 5% in inner London, in combination with a move toward cleaner alternatives across other vehicle types, would bring 99.96% of London into compliance with the current European Union legal levels for NO₂ (5). Until now, a “what if” approach has not been widely available for health outcomes reported in epidemiological studies focused on air pollution. However, in this issue of the *Journal*, Urman and colleagues (pp. 438–444) use casual inference methodology to address the question of what would have happened to lung function growth in children in the CHS if they had grown up in communities that conformed to international air pollution standards (6). Their finding that a 30% reduction in NO₂ would have increased FEV₁ growth by 4.4% adds to our armamentarium of data that can be used to advocate for the right of all children to breathe clean air.

Clearly, the analytic approach used by Urman and colleagues is ideally suited for assessing the effect of exposure reduction on other adverse health effects and would be a very powerful tool in combination with modeling the types of interventions needed to achieve clinically meaningful outcomes within conurbations, such as banning fossil fuel-powered vehicles. Using the same methodology, these researchers recently reported that compliance with a hypothetical 20 ppb NO₂ standard in southern California would result in a 20% (95% confidence interval, –27% to –11%) lower incidence of childhood asthma (7). Given that we now have the tools to advocate for cleaner air for children, do we need any more epidemiological studies of the health effects of air pollution? The answer must be yes. First, the independent effects of NO₂ and PM are still unclear. This may not be an issue where exposure-reduction policies reduce both PM and NO₂, but it would be important when the switch to electric vehicles eliminates NO₂ emissions but not PM emissions from tire and brake wear. The difficulty of identifying the independent effects of NO₂ in current studies is illustrated by a recent report published by the UK government’s Committee on the Medical Effects of Air Pollutants (8). In assessing the association between long-term exposure to NO₂ and mortality, a

dissenting group of the Committee’s members considered that the uncertainty in the estimation of the hazard ratios in two-pollutant models precludes their use in quantification exercises (8). An additional area of uncertainty is whether PM from sources other than fossil fuels, such as biomass burning, has similar adverse health effects on children, and without this knowledge we cannot readily apply the important findings of Urman and colleagues to pediatric lung health on a global scale. ■

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