

Air pollution and asthma

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Outdoor air pollution is a major risk factor for both acute and chronic respiratory illness and mortality. Short-term exposure (from a few hours to several days) to elevated levels of air pollution has been associated with a range of adverse health effects on lung function. There is increasing epidemiological evidence of the association between air pollution and asthma exacerbations in children,¹ with some studies also suggesting a contribution to new onset of asthma. Air pollution, however, is a complex mixture of particles and gases emitted from a wide range of sources or generated in the atmosphere through reactions of chemical species. It is difficult to disentangle the effect of specific pollutants on health using common epidemiological methods, particularly in cases where these pollutants, for example, combustion-related particles and nitrogen dioxide, correlate strongly.

From a mechanistic point of view, fine particles <2.5 µm in diameter (PM_{2.5}) can penetrate deep into the airways and induce alveolar inflammation. In addition to PM_{2.5}, gaseous air pollutants such as nitrogen dioxide (NO₂) and ozone (O₃) can cause oxidative injury to the airways, leading to inflammation and remodelling.² Young children with asthma are particularly susceptible to air pollution because of their developing lungs, immature metabolic pathways, high breathing rates per bodyweight and amount of time spent exercising outdoors.¹

In a large longitudinal study presented in this journal, Bouazza *et al*³ examined paediatric visits for asthma exacerbation to emergency departments (EDs) in Paris, France, in relation to air pollution and other factors such as daily weather (including temperature and humidity), viral infections and pollen data. The authors analysed over 47 000 cases of paediatric visits in 20 hospitals over 5 years using a non-linear modelling approach to examine the exposure-response relationship between single or multiple pollutants

and asthma exacerbations. Their exposure-response model, which was approximated with a sigmoid function, indicated that daily asthma ED visits increased gradually with PM_{2.5} up to a plateau, with the annual European Union (EU) limit value of 25 µg/m³ corresponding to 65% of the maximum estimated effect. In other words, most of the short-term effects of PM_{2.5} on asthma exacerbations in children occurred at concentrations below the EU limit value. Even the more conservative WHO guideline of 10 µg/m³ as an annual mean for PM_{2.5} corresponded to 42% of the estimated maximum effect.

This is an important finding indicating that the current EU limit value for PM_{2.5} (25 µg/m³ as an annual mean) is unlikely to provide sufficient protection from short-term exposure to particulate air pollution to children with asthma. A significantly lower annual limit value for PM_{2.5}, closer to the WHO guideline of 10 µg/m³, potentially combined with an appropriate daily limit value, would be needed to protect children from asthma exacerbations based on the evidence presented in this study. These asthma ED visits need to be considered as 'the tip of the iceberg', that is, cases that were serious enough for the children's parents to bring them into hospital, and do not include potentially many milder exacerbations. Separate evidence suggests that complying with the WHO guideline of 10 µg/m³ for PM_{2.5} as annual mean in Europe would bring substantial public health benefits in terms of avoidable premature mortality in the adult population.⁴ Fine particulate pollution has health impacts even at very low concentrations as no threshold or safe level of exposure has been identified below which no damage to health is observed.

Bouazza *et al*³ did not detect additional effects of O₃ or NO₂ on asthma ED visits after accounting for PM_{2.5}, which suggests

that fine particles could be the trigger of asthma exacerbations on days of relatively high air pollution. However, PM_{2.5} and NO₂ correlated strongly and could be surrogates of traffic-related pollution in urban areas. Lagged effects were not tested in this study, although other authors have detected strong effects on the day after high-level pollutant exposure.¹

Associations between exposure to air pollution, pollen and vegetation with asthma hospitalisations are complex.^{5, 6} Bouazza *et al*³ examined pollen as a single entity, although it consists of many species which have different levels of allergenicity and size, altering its role in asthma exacerbations. Additional work examining pollen at the species level may provide more refined results.

Bouazza *et al*³ used pollution data from the air quality monitoring network in the region of Paris. The study included a limited number of monitoring stations, particularly for PM_{2.5}, for which data from only four locations (one very near and three outside Boulevard Périphérique, the ring road of Paris) were obtained. This may have resulted in underestimation of variability in population exposure to air pollution. This common limitation of epidemiological studies highlights the need for better spatial coverage and more refined exposure assessment techniques, potentially taking into account time spent in different microenvironments (home, school, playground, commuting). Refined exposure assessment is particularly important in studies focusing on the potential role of air pollution in causing asthma in children living close to busy roads.

AIR POLLUTION CONTROL AND CHILDHOOD ASTHMA EXACERBATIONS

EU air quality legislation is more stringent for NO₂, in the sense that the EU limit value matches the WHO guideline for NO₂ as an annual mean (table 1). In recent years, attention has shifted from particulate air pollution to NO₂, the pollutant which is causing most regulatory

Table 1 European Union (EU) air quality regulations and WHO guideline values for key pollutants

Pollutant (µg/m ³)	EU limit values				WHO guidelines			
	Annual	24 hours	8 hours	1 hour	Annual	24 hours	8 hours	1 hour
PM _{2.5}	25*				10	25		
NO ₂	40			200†	40			200
O ₃			120‡				100	

*With additional objectives targeting the exposure of the population.

†Not to be exceeded >18 times a year.

‡Not to be exceeded by >25 times a year averaged over 3 years.

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compliance issues in Europe (ie, many urban areas are exceeding the annual NO₂ limit value of 40 µg/m³). However, we need to remember that the evidence associating exposure to air pollution with adverse health outcomes, including asthma exacerbations, is generally stronger for fine particulate matter than for other pollutants. In many cases, policies aiming to reduce NO₂ will also have a positive impact on ambient PM_{2.5} levels. For example, a shift from motorised transport to active travel (ie, more walking and cycling) in cities will reduce traffic-related NO₂ and PM_{2.5}, as well as carbon emissions and noise, providing multiple opportunities ('co-benefits') for improved physical and mental health.

There is growing consensus among environmental and respiratory scientists that levels of outdoor air pollution need to be lowered, particularly in densely populated areas, to protect children's health. Considering the importance of air pollution as a trigger for asthma exacerbations in children, and its potential role in asthma initiation, improved mechanistic understanding of these associations is needed, so that prevention strategies can be better targeted.¹ In addition to improved outdoor air quality, reduced exposure to secondhand smoke, allergens and unflued

heaters is likely to reduce the risks, while improved medication compliance will have a protective effect.⁷ Additional large population studies are required to assess the effectiveness of policy interventions in reducing asthma symptoms, exacerbations and mortality. As progress in reducing hospital admissions and mortality in people with asthma has stalled in the past 10 years,⁸ decisive action is now needed to improve air quality and respiratory health in children.

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