

Asthma exacerbations, air pollution, and allergens

Peter Burney and André Amaral¹ refer to the episodes of severe asthma epidemics in Barcelona, Spain, to suggest that aeroallergens could be more threatening for people with asthma than are urban air pollutants. The principal cause of the asthma epidemics in Barcelona was a protein from the hull of the soybean (which was dispersed from the harbour silos to the city air when the cargo was unloaded) and not urban air pollutants, such as black carbon particles or nitrogen dioxide. However, the effect of exposure to soybeans on asthma exacerbations was lower than was the effect of exposure to air pollutants in the whole population.^{2,3} This difference in effect is because only a proportion of people with asthma had an allergic sensitivity to soybeans⁴ and the soybean cargo was unloaded only on a small proportion of days, dispersing the allergen into the air infrequently.⁵ By contrast, pollution from combustion sources is present every day and its irritant and inflammatory effects put all people with asthma at risk for exacerbations. Besides, the presence of the soybean allergen in the air did not confound the role of pollutants on the number of asthma-associated admissions to the emergency rooms in Barcelona,⁶ unlike how pollen did in London.⁷ In conclusion, although we agree with Burney and Amaral that further research should improve the characterisation and our understanding of the role of air particles, including allergens, on the exacerbations of asthma, we do not agree that the effect of urban air pollution on asthma exacerbations should be downplayed. In particular, we propose that research should assess whether air pollution could augment the effects of allergens as temperatures increase and the climate changes worldwide.

We declare no competing interests.

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Authors' reply

We thank Jordi Sunyer and Josep Antó for their comments regarding our Viewpoint.¹ Their ground-breaking studies in the 1980s showed that what had been reported as an association of an outbreak of asthma with an increase in ambient levels of oxides of nitrogen,² was in fact caused by exposure to an allergen. Without identifying the specific allergen, there would have been no way of knowing that the initial report was misleading. After the epidemics had been controlled, sporadic cases of asthma exacerbations that were associated with soybean allergy continued to occur. These cases would also have been missed if the allergen had not been known and monitored in air, and if people with sporadic exacerbations were not tested for this specific sensitisation.³

Sunyer and Antó state confidently that “By contrast, pollution from combustion sources is present every day and its irritant and inflammatory

effects put all people with asthma at risk for exacerbations”, but how do they know? Even more importantly, how can they be sure about the size of the effect? Allergens are specific to each person who is allergic and there is no generic test for allergens in the air. Pollen counts are a poor marker of free allergen in the air⁴ and pollen itself is a poor trigger of asthma compared with the much finer allergen particles that are released from pollen, for instance during thunderstorms. Under controlled conditions, people with asthma react strongly to even small doses of allergens to which they are sensitised, but react only slightly to usual levels of inorganic particles.

We did not suggest that generic air pollution could not exacerbate asthma. However, we did emphasise that one of the largest pollution episodes in the past century had so little effect on asthmatic patients that the absence of an effect was thought to be worth reporting in *The Lancet* at the time.⁵ We also pointed out that the severe smog in Europe in 1985 showed a reduction in asthma-associated admissions to hospital in the areas affected in Germany, despite increases in admissions for chronic obstructive pulmonary disease and cardiovascular events.⁶ These findings are incompatible with the generally held view, echoed by Sunyer and Antó, that particulate air pollution per se causes asthma exacerbations.

Although we agree that more research is needed, we would emphasise the need to prioritise the specific issue of how to distinguish the effects of small doses of multiple allergens that are dispersed as fine particles in air, from short-term changes in general levels of pollution, which are largely driven by climatic conditions. Without this distinction, more studies associating general air pollution with asthma exacerbations will remain uninterpretable and unhelpful.

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