Dietary Fat Intake, Particulate Matter Exposure, and Asthma Severity

It seems intuitive that exposure to combustion-related particulate matter would worsen asthma symptoms, given the known proinflammatory effects of combustion-related byproducts and deposition in the lungs. A great deal of research during the past several decades has been performed to establish this link. On the whole, the findings from these studies have generally supported the connection between combustion-related air pollution and asthma severity (1). However, collectively, the associations shown to date have been relatively modest. For example, in the randomized crossover study of exposure to high–vehicle traffic areas in London, United Kingdom, although adult patients with asthma had modestly lower lung function (~6% lower) after walking in an area of London with high truck traffic than after walking in Hyde Park, there was no effect on asthma symptoms (2). Also, the timing of the large population increase in asthma prevalence in places such as urban communities of the United States, starting in the latter half of the 20th century, does not coincide with dramatic increases in combustion-related air pollutants. A study of sediments from a lake in Central Park in New York City lake found that polycyclic aromatic hydrocarbons from air pollution sources peaked in the 1910s–1920s and then decreased during the remaining century as the city transitioned from coal to petroleum fuel and then to cleaner-burning petroleum under tighter environmental regulations in the 1970s (3). This suggests that if air pollution is important in asthma development and exacerbation on a population level, that factors or exposures influencing susceptibility to combustion byproducts must have changed in urban US communities in the latter half of the 20th century.

In this issue of the *Journal*, the article by Brigham and colleagues (pp. 1478–1486) could offer some insight into one of those possible effect modifiers (4). In a panel study, they followed 135 predominantly African-American children from low-income households. The children were followed for three 1-week periods during a year, during which domestic particulate matter was measured and dietary intake of omega-3 and omega-6 fatty acids and asthma symptoms were assessed. Notably, the researchers found that associations between particulate matter exposure and lung function and symptoms were modified by reported intake of omega-3 and omega-6 fatty acids. Higher omega-6 fatty acid intake was associated with an increased association between particulate matter $\leqslant$2.5 μm in aerodynamic diameter and asthma symptoms, whereas higher omega-3 fatty acid intake was associated with a reduced association between particulate matter $\leqslant$2.5 μm in aerodynamic and asthma symptoms.

In the United States, low-income minority communities have suffered disproportionately from the asthma epidemic. Again, considering the timing of the asthma epidemic, the latter half of the 20th century in the United States saw dramatic changes in diet. This included a notable increase in dietary fat from soy bean–derived oils, which were commonly used in processed food because of their lower costs (5). Since then, soy bean–derived oils have been a major source of omega-6 fatty acids in the diet in the United States, although the most recent decades have seen a decrease in these fatty acids among higher–socioeconomic status communities, which has not been observed in lower-income communities (6). Therefore, it is compelling to think that dietary changes could underlie some of the increased prevalence and morbidity observed with the asthma epidemic and the disparities observed in lower-income, urban communities.

The majority of research on dietary fatty acids and asthma has focused on the potential protective effects of omega-3 intake in pregnancy and asthma development. Randomized control trials in which mothers received omega-3 supplementation during pregnancy have produced mixed results (7–10). A recent systematic review and meta-analysis of observational and trial data concluded that although the data have been inconsistent, they are suggestive of the benefits of increased maternal consumption of omega-3s in pregnancy and outcomes of childhood allergic disease (10). Data from prospective birth cohorts have shown associations between higher omega-6 intake in pregnancy and higher risk for eczema (11) and allergic rhinitis in childhood (12). However, higher omega-6 has not been consistently shown to increase risk for allergic disease (13, 14). One recent review focused on the potential of dietary supplementation in mitigating the effects of several environmental pollutants, including dietary fatty acids, and concluded that although the Mediterranean diet (high in omega-3s) has been shown to be beneficial in respiratory disease, evidence on the protective effects of dietary changes on the detrimental effects of pollutants has been limited to tobacco smoke exposure (15).

Therefore, the study by Brigham and colleagues is novel in its demonstration of risk for asthma morbidity with particulate matter exposure that was modified both by omega-6 (detrimental) and omega-3 (beneficial) intake, and that these associations were independent of each. Although the study had a relatively small sample size, the inclusion of only patients with asthma and the repeated sampling during different times of the year contributed to the robustness.

The study has its limitations. The findings are essentially cross-sectional, and thus subject to reverse causality. Also, the study did not directly measure omega-3 and omega-6 consumption, but the researchers did assess it, using an instrument relevant to their study population. Therefore, an important next step would be a prospective intervention trial on dietary modification to confirm whether changes in diet truly modify the association between indoor pollution and asthma symptoms. It was also of interest that

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the detrimental modifications observed with omega-6 consumption appeared to be more robust across outcomes than the beneficial effects of omega-3 consumption. This might have implications for the type of intervention that could be successful. In addition, although the authors discuss two modifiable risk factors, diet and indoor particulate matter, they did not present any information regarding the potential sources of particulate matter, which would be necessary to design an intervention in this community.

In short, the study by Brigham and colleagues potentially provides novel insight into the intersection of environmental and dietary factors affecting asthma exacerbation that may be relevant to the as-of-yet unmitigated disproportionate burden of asthma morbidity in low-income urban communities in the United States. The important next steps include validation in other populations and demonstrating the efficacy of dietary modifications and/or particulate matter reduction to reduce the burden of asthma in these communities. ■

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Small but Mighty: Prenatal Ultrafine Particle Exposure Linked to Childhood Asthma Incidence

Research demonstrating a role for air pollution in the prenatal programming of asthma has largely considered exposures to criteria pollutants (pollutants routinely monitored to assess air quality), especially particulate matter with an aerodynamic diameter of 2.5–10 μm or fine particles ≤2.5 μm ($PM_{2.5}$) and ambient nitrates (NO_3) (1). Although air quality regulations currently do not address ultrafine particles (UFPs) ≤0.1 μm, submicron-sized particles may exert greater toxic effects compared with larger molecules because of their larger surface area/mass ratio, chemical composition, deeper lung penetration, and enhanced oxidative capacity and ability to translocate to the systemic circulation (2–4). A recent workshop identified the lack of studies differentiating the effects associated with UFP exposures from effects related to other particle size fractions and gaseous

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