Indoor and outdoor air pollution rank as the fourth and ninth leading causes of global morbidity and mortality in the most recent Global Burden of Disease (GBD) report and collectively outrank other major risk factors in terms of importance (1). The fine particulate matter \(<2.5 \, \mu \text{m} (\text{PM}_{2.5})\) component of inhaled particulate matter, more than other larger fractions, dominates risk through its impact on cardiovascular events (2). A substantial body of evidence supports a stereotypic activation of adverse mechanisms, including heightened inflammation, vascular dysfunction, autonomic imbalance, and enhanced thrombosis in response to air pollution exposure (2). Interestingly, these same mechanisms also potentiate other chronic diseases (e.g., atherosclerosis, hypertension, and diabetes), and thus, may contribute to a much larger attributable risk from pollution than previously appreciated.

Although indoor air pollution from combustion of biomass fuels including wood, charcoal, crop residues, and dried animal dung continues to remain a stubborn consequence of economics in many parts of the world, in the large industrial megacities of China, outdoor sources of air pollution, including coal power generation, automobile exhaust, and industrial entities dominate (3,4). Daily outdoor air pollution levels in many major cities in Asia, such as Beijing, Shanghai, and Wuhan, often exceed 100 to 500 \(\mu \text{g/m}^3\). Recent data derived from satellite based measurements of air quality in East Asia are sobering and point to an increase in the proportion of patients in East Asia living above the World Health Organization (WHO) interim Target-1 of 35 \(\mu \text{g/m}^3\) (increased from 51% in 1998 to 2000 to 70% in 2010 to 2012) (5). In stark contrast in North America, the vast majority of the population lives below the WHO Air Quality standard of 10 \(\mu \text{g/m}^3\), with 20% exposed to \(\text{PM}_{2.5}\) above this level (5). However, individuals living within regions meeting air quality standards can still face health risks posed by “hot spots” of exposure (e.g., near roadways, point sources of emissions).

Studies of air pollution have traditionally focused on exclusive outdoor and indoor domains for reasons relating to the nature of pollutants, sources, and populations exposed. This dichotomization is irrelevant in cities in China and India, where outdoor air pollution has an outsized effect on indoor levels. Given the fact that individuals in most societies spend a substantive amount of times indoors, considerable exposure to air pollution may occur in this setting. Improving outdoor air quality has been a Sisyphean task in Asia, with short-term improvements being overshadowed by an inexhaustible appetite for cheap power and automobile use. The indoor environment in contrast to outdoor air pollution can be controlled. The evidence to date on the impact of improving indoor air quality has been mostly from the West using air purifiers to improve pollutants attributable to specific sources at relatively lower levels (5 to 50 \(\mu \text{g/m}^3\)) (6–9). An obvious question is whether reducing much higher levels of indoor air pollution (50 to 150 \(\mu \text{g/m}^3\)) also will translate into similar effects on cardiovascular events and if so, at what time course?

In this issue of the Journal, Chen et al. (10) publish a timely investigation of the effectiveness of a

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double-blind, randomized, cross-over study of an air filter intervention >48 h, among 35 healthy young university students in Shanghai, China. The chosen location is a strength of the study, being conducted in a very polluted part of the world, with average levels of 103 μg/m³. Active air filtration proved capable of reducing indoor PM$_{2.5}$ concentration by just more than one-half, from 96.2 to 41.3 μg/m³. After 48 h of “cleaner air” exposure, systolic and diastolic blood pressure fell, whereas a few of the measured metrics of inflammation (MCP-1, interleukin-1β, myeloperoxidase) and platelet activation (sCD40L) were significantly reduced. Several other biomarkers also trended towards improvement, but were not significantly changed. Fractional exhaled nitric oxide, an established marker of respiratory inflammation, was reduced, but lung function measured by spirometry did not change, possibly owing to the short-term nature of the intervention. Added to the results from a few previous studies, these new findings bolster the evidence that improving indoor air filtration may be a practical “personalized” method to reduce overall PM$_{2.5}$ exposure and mitigate adverse health effects. The observed improvement in outcomes, despite particulate levels remaining high during air filtration (41.3 μg/m³) supports the prevailing understanding of a log-linear dose-response relationship between exposure and health effects, whereby any lowering of pollution can translate into benefits with larger absolute benefits, the higher the level of air pollution (2). The population was homogeneous, taking no potentially confounding medications, and residing within 2 nearby buildings, thus making for a clean study design. Assuming the participants complied with the protocol, potential confounding effects of lifestyle changes (e.g., diet, activity), unmeasured environmental factors (e.g., traffic, noise, temperature), and PM$_{2.5}$ exposure misclassification(s) were all likely minimized. Most of the improved health endpoints also were significantly linearly associated with the directly measured indoor PM$_{2.5}$ levels across both exposure scenarios, lending further credence to a probable causal exposure–health response relationship. Though changes in gaseous pollutants did not likely confound the main results, as they were thought not to be altered by filtration, their levels were not evaluated. It thus remains unclear whether a concomitant reduction in these copollutants could provide further or differing health benefits. The specific sources (e.g., traffic, coal-fired power plants), particle sizes (fine vs. ultrasize ranges), and components (organic/elemental carbon, sulfates, metals) most strongly linked to the health effects also were not reported.

Although improvement in outdoor air quality ultimately will be needed to change indoor air quality in the cities of China and India, when rapid changes in outdoor air quality occur, as it did during the Beijing Olympics (PM$_{2.5}$ levels 101 and 84 μg/m³ before and after, averaging 69 μg/m³ during the games), several biomarkers of inflammation thrombosis, as well as blood pressure, improved (11). The good news is that air quality regulations, when enforced, can substantially reduce prevailing outdoor air pollution concentrations and improve societal life expectancy rates (12). In the meantime, personalized behavioral and small-scale interventions to lower exposures (e.g., filters in homes and cars) may be needed in order to optimally protect citizens in these areas (13).

**REFERENCES**


indoor air filtration and acute changes in cardiorespiratory health in a First Nations community. Indoor Air 2013;23:175-84.


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