Can What You Breathe Trigger a Stroke Within Hours?

Air pollution is a pervasive consequence of modern societies. Beyond being an environmental hazard, several pollutants such as particulate matter (PM) pose significant health threats. Fine PM (<2.5 µm in diameter; PM2.5) is a heterogeneous mixture of compounds (e.g., carbon, sulfates, nitrates, and metals) principally derived from the combustion of fossil fuels. Given the billions of people exposed, the adverse effects of PM represent a major global health epidemic.

Although adverse pulmonary responses may be the most conspicuous effects of PM, in actuality, cardiovascular (CV) diseases account for the largest proportion of the PM-related public health burden. Exposures to ambient PM2.5 over both the short and long term have been associated with a variety of CV events. The epidemiologic evidence and the mechanistic bases for such an association have grown substantively. In this context, the American Heart Association recently published an updated scientific statement concluding that PM2.5 is an important modifiable factor contributing to CV diseases.

Over the past decade, several (but not all) studies have also demonstrated a linkage between air pollutants (including PM) and stroke. This putative relationship has enormous public health ramifications, since stroke is the second leading cause of death worldwide. Moreover, low- to middle-income countries that are subject to severe air pollution problems also bear a disproportionate burden of global stroke mortality. Though the immediate risks posed by air pollution to any single individual may be small, the population-attributable risk for stroke is highly significant, given the pervasive nature of the problem. For example, while PM exposure increases myocardial infarction risk by only 1% to 2% within days, owing to the enormous number of individuals continuously exposed, it can account for the instigation of up to 4% of all events in a population. In addition, air pollutants have been shown to adversely modulate several of the traditional CV risk factors (such as blood pressure), which altogether account for roughly 90% of all strokes worldwide.

In this issue of the Archives, an article by Wellenius et al provides some important information that helps to further elucidate the relationship between air pollutants and stroke. In a study of patients admitted to a stroke center in Boston, the authors found that the risk for ischemic stroke was elevated acutely following short-term exposure to higher levels of ambient PM2.5. Hemorrhagic strokes were not investigated; however, this subtype has been less consistently associated with stroke in the past. Although this single-center study was smaller than some prior studies, there are several unique aspects. Fine particulate matter, black carbon, and nitrogen dioxide were associated with ischemic stroke, but ozone or sulfates were not, suggesting a major contribution of traffic-related particulates. These positive associations were also shown to persist when PM2.5 levels were well below current daily ambient air quality standards (<35 µg/m³), suggesting that more aggressive regulations are required to lower risk among all susceptible people. The risks for ischemic stroke subtypes were also well characterized and shown to be related to large-artery and small-vessel events, but not cardioembolic events. Perhaps most noteworthy among their findings was the importance of very recent PM2.5 exposures over the preceding 6 to 24 hours for triggering an event. Though several studies have found positive relationships with same-day or previous-days pollution levels, none has investigated the degree of temporal resolution provided by this analyses. The extremely rapid increase in stroke risk is an important novel insight; however, it should be noted that the temporal nature of PM-mediated CV events is a complex issue that has been controversial of late.

Why are these findings important? Current US and World Health Organization air quality standards focus only on daily and annual PM2.5 mean concentrations. There is no biological basis that these specific durations of exposure are required to instigate strokes or other CV events. These temporal windows have typically been chosen owing to limitations of available air pollution data and because longer averaging periods were thought to provide more reliable exposure estimates. The findings by Wellenius et al remind us that clinical events could be initiated by even briefer subdaily periods of PM2.5 inhalation. Indeed, controlled-exposure studies and panel studies corroborate this possibility showing that adverse biological responses can occur within hours. With the availability of improving pollutant data, future studies should aim to elucidate the health effects of subdaily time windows of exposure. If similar findings are corroborated and/or found for other diseases, those who create air quality standards might need to consider the difficult task of curbing subdaily...
PM concentrations, as they did to create the existing 8-hour ozone regulations.1

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Evacuee camps from the tsunami and radiation disaster in Fukushima, Japan.

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