Cardiovascular disease is by far the largest cause of mortality in the U.S. population. While several individual biological characteristics, such as hypertension, diabetes and cholesterol levels, have long been known to represent important risk factors for the development of disease, there has been relatively little scrutiny in the clinical literature of the role of the environment in the etiology of circulatory disease or in the precipitation of acute events. However, in environmental health and epidemiology journals, scores of studies conducted on five continents have documented consistent associations between acute (i.e., 24-hour) exposures to ambient air pollution and daily mortality, especially among older individuals with preexisting cardiac and respiratory diseases.1

These findings are supported by numerous reports linking ambient concentrations of several pollutants, notably particulate matter (PM), carbon monoxide and ozone, to hospitalizations for cardiovascular events.1 These remarkably consistent associations suggest that exposure to ambient air pollution is a risk factor for exacerbation of preexisting cardiac illnesses, though pathophysiological mechanisms are still incompletely understood.

Of particular research interest is the relationship between traffic emissions and occurrence of acute events and possibly chronic illness as well. Traffic emissions consist of a heterogeneous mixture of biologically active gases, such as nitrogen oxides and carbon monoxide and particulate matter (PM), which includes diesel soot, condensed combustion gases, tire fragments and entrained dust and soil. The concentrations of all of these and other traffic emissions vary in both time and space, with the highest concentrations near busy roads. Within the many pollutants associated with traffic, researchers have focused especially on fine and ultrafine PM, both of which can easily penetrate to the deep lung and, in the case of ultrafine PM, translocate into the blood to be transported throughout the body. Fine PM is 2.5 micrometers or less in diameter, while ultrafine PM is even smaller: 0.1 micrometer or less. By comparison, a human hair is typically 50 to 60 micrometers in diameter.

Last year German investigators published the results of a study examining the activities of 691 myocardial infarction (MI) survivors during the four days before they had symptoms.2 They reported a statistically significant near-tripling of the risk of MI within one hour of having been in traffic compared with other times when the subjects were not in traffic. This association was present regardless of whether the subject had been in a car or public transportation, or on a bicycle or motorcycle. Statistical adjustment for potential effects from the degree of exertion on a bicycle or from getting up in the morning decreased the estimate of this association by less than 10 percent. The observation that the effect was present for individuals taking public transportation suggests that the relationship could not be explained solely by the stress associated with driving a car.

Another biologically plausible explanation for at least some of the epidemiological findings linking air pollution and acute cardiovascular events concerns disturbances of the autonomic regulation of the heart, which are often measured as alterations in heart rate variability (HRV). HRV describes changes in consecutive normal sinus beat-to-beat intervals; decreased HRV has been associated with sudden cardiac death and mortality from heart failure. At least half a dozen studies have linked ambient fine PM with transiently decreased HRV. One recent paper found that the black carbon fraction of PM2.5, which is primarily found in vehicular (particularly diesel) exhaust, was more strongly associated with...
HRV decrements than other subcategories. Other work has shown associations between pollutants and electrical discharges of implantable defibrillators, again with the strongest associations linked to vehicular emissions.

In contrast to the plethora of environmental epidemiological studies linking daily mortality or cardiovascular hospital admissions with ambient air pollution, there are but a handful examining the relationship between long-term exposure to air pollution and mortality from cardiopulmonary disease. The largest of these involved an examination of the mortality experience of over 500,000 adults in 151 U.S. cities who participated in the American Cancer Society II (ACS) cohort. After controlling for individual risk factors such as smoking, occupational exposures, body mass index, alcohol consumption, diet, and co-pollutants, a 10 microgram/m3 increase in the annual average concentrations of fine particles in these metropolitan areas was associated with significant increases in relative risks (RR) for total cardiovascular mortality (RR = 1.12, 95 percent CI 1.08 - 1.15), as well as for specific cardiac causes of death, including ischemic heart disease (RR = 1.18, 95 percent CI 1.14 - 1.23) and a combined mortality category of arrhythmia, heart failure or cardiac arrest (RR = 1.13, 95 percent CI 1.05 - 1.21).

Increased risks of mortality were also observed in relation to several other pollutants, including sulfur dioxide and sulfate particles (both arising mainly from fossil fuel combustion) and summertime ozone. Interestingly, however, the increased risks of mortality were limited to the group without formal education beyond high school, suggesting that one or more factors associated with educational attainment modified the effect of air pollution. Such factors could include nutrition or residential location in relation to busy streets. A smaller Dutch cohort study found that an individual’s exposure to air pollution may vary as much within a single city as across different cities. In that study, involving 5,000 adults followed up for eight years, the authors found that exposure to traffic-related air pollutants was more strongly related to mortality than were citywide background air pollution levels. While the investigators measured pollutant exposures in a variety of ways, the metric most strongly associated with cardiopulmonary mortality in this cohort was whether a subject lived near a major road (RR = 1.95, 95 percent CI 1.09 - 3.52).

During the last decade, the state of knowledge about the relationship of air pollution to cardiovascular disease has advanced from extensive ignorance to a point where the American Heart Association (AHA) could publish a thorough review of this area, with an implicit recognition of a causal relationship. The AHA review found that exposure to air pollution could “accelerate the development of coronary atherosclerosis and worsen its sequelae,” stating further that “some of these effects may occur over time, as with acceleration of the progression of atherosclerosis, or rather abruptly, as with the triggering of an arrhythmia or myocardial infarction by acute inflammatory responses, altered platelet adhesiveness, or perhaps vascular endothelial dysfunction.” As noted above, recent work suggests marked increases in risk for traffic-associated acute events, such as myocardial infarction. Given that motor vehicle exhaust and other sources of ambient air pollution are ubiquitous, especially in urban areas, it is reasonable to consider that exposures to PM and other pollutants present a significant risk to public health.

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