

EDITORIALS



Air Pollution and Health — Good News and Bad

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Early concerns regarding the health-related effects of air pollution originated from severe episodes in Meuse Valley, Belgium, in 1930; Donora, Pennsylvania, in 1948; and London, in 1952. Although the overall effects of these episodes continue to be debated, well-documented, episode-related increases in morbidity and mortality from cardiopulmonary causes provided dramatic evidence that extremely high concentrations of air pollution can have serious adverse effects on health. Early public-policy efforts to improve air quality in the United States, Britain, and elsewhere were largely attempts to avert such “killer” episodes of air pollution. In the United States, a series of national legislative and regulatory efforts to control air pollution were initiated (Fig. 1); National Ambient Air Quality Standards were mandated and established; and dramatic, extremely severe episodes of air pollution were essentially eliminated.

From the 1960s through the 1980s, a few scattered studies continued to suggest that air pollution had adverse effects on health.^{1,2} Then, during the relatively short period of 1989 through 1995, several loosely connected epidemiologic studies reported adverse effects of unexpectedly low levels of particulate-matter air pollution.³⁻⁶ Although highly controversial,⁷ these results prompted serious reconsideration of the particulate-matter standards and health guidelines (Fig. 1). They also prompted extensive efforts to reanalyze key studies⁸ (which were largely confirmatory) and motivated rapid growth in epidemiologic, toxicologic, and other studies of fine particulate matter and other combustion-related air pollutants.

Research has continued to suggest that a level of air pollution that is common in many urban and industrial environments is an important risk factor for various adverse health effects in humans. Al-

though many such studies have focused on respiratory disease, substantial and growing evidence indicates that fine particulate air pollution is also a risk factor for cardiovascular disease.^{9,10} Short-term exposure exacerbates existing pulmonary and cardiovascular disease and increases the risk of symptoms, the need for medical attention, and death.¹ Long-term, repeated exposure increases the cumulative risk of chronic pulmonary and cardiovascular disease and death.⁹⁻¹³

One notable research effort that began in the early 1990s in the midst of the controversies about air quality was the Children’s Health Study. This study prospectively monitored the lung function of schoolchildren from the ages of 10 to 18 years in 12 southern California communities with a relatively wide range of air pollutants. As reported by Gauderman et al. in this issue of the *Journal*,¹⁴ air pollution was significantly associated with deficits in lung development. Within the context of the overall literature on air pollution and human health, this article makes several important and confirmatory contributions.

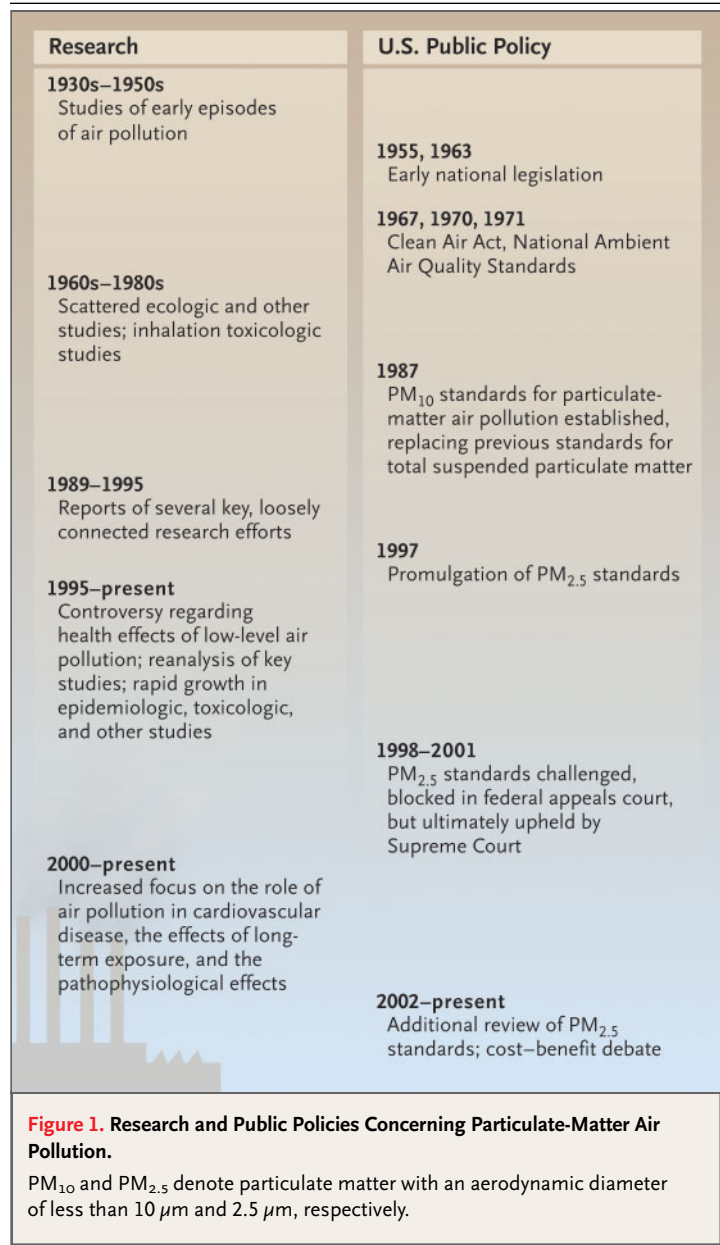
The Children’s Health Study evaluated the cumulative exposure to various pollutants over an eight-year period. Deficits in the growth of lung function over the eight-year period were associated with a correlated set of pollutants that included fine particulate matter with an aerodynamic diameter of less than 2.5 μm , nitrogen dioxide, acid vapor, and elemental carbon. These results are consistent with those of previous epidemiologic studies that have implicated fine particulate matter and associated combustion-related air pollutants as being largely responsible for the observed health effects of air pollution.^{1,2,5,11-13} Various physiological and toxicologic findings suggest that exposure to fine particulate matter may be an important pub-

lic health concern. Such matter, which can be breathed deeply into the lungs, includes sulfates, nitrates, acids, metals, and carbon particles with various chemicals adsorbed onto their surfaces. Furthermore, fine particulate matter is ubiquitous because it is largely derived from common combustion processes (such as engines in motor vehicles, manufacturing, power generation, and burning of biomass) and because it is transported over long distances and readily penetrates indoors.

Understanding the shape of the exposure–response relationship and determining whether there are safe thresholds are important for the formulation of public health policies for pollution control. Mortality studies suggest that the exposure–response relationships for particulate-matter pollution in the case of both short-term¹⁵ and long-term¹¹ exposures are nearly linear, with no discernible safe thresholds within relevant ranges of exposure. Likewise, in the Children’s Health Study, the exposure–response relationships appear to be nearly linear, without discernible safe thresholds.

An issue with clinical implications concerns the identification of groups that are most at risk or that are most susceptible to the effects of pollution. One evaluation of the literature¹ suggests that the proportion of a given population that is at risk for death, hospitalization, or life-threatening conditions owing to short-term exposure to air pollution is very small and limited to the elderly, infants, and persons with chronic cardiopulmonary disease, influenza, or asthma. There appears to be a much broader susceptibility to small, transient changes in lung function, low-grade pulmonary inflammation, or other subclinical physiological changes in response to short-term exposure.

With regard to the cumulative effects of long-term, repeated exposure, there is little evidence of a unique, well-defined, susceptible subgroup. The Children’s Health Study reports pollution-related deficits in the development of lung function in boys and girls, children with asthma and those without asthma, and smokers and nonsmokers — results “suggesting that most children are susceptible to the chronic respiratory effects of breathing polluted air.” The authors of the current study also note that reduced lung function is a risk factor for complications and death during adulthood and suggest that the effect of these pollution-related deficits in lung function may occur later in life. In fact, studies have shown that long-term, repeated exposure to air pollution is associated with an increased risk



of death from cardiopulmonary causes in broad-based cohorts or samples of adults.^{5,6,9,11,13}

Much additional research is required to understand the biologic mechanisms that link exposure to fine particulate matter with increases in morbidity and mortality from cardiopulmonary causes. However, several recent studies suggest that general mechanistic pathways probably include pulmonary and systemic oxidative stress and inflammation, enhanced initiation and progression of atherosclerosis, and altered cardiac autonomic function.^{9,10}

Secondhand cigarette smoke has also been shown to promote inflammation and atherosclerosis and to be a risk factor for illness and death from cardiopulmonary causes — suggesting that exposure to fine particles from common outdoor sources of combustion and from tobacco smoke may invoke similar pathophysiological processes.^{9,10} The Children's Health Study does not provide direct evidence regarding the mechanisms of air-pollution effects, but the authors suggest a role of airway inflammation, such as that observed in smokers and persons who have lived in polluted environments. Although there has been much interest recently in the importance of pulmonary inflammation, atherosclerosis, and cardiovascular disease, the Children's Health Study reminds us not to forget or ignore potentially important effects of pollution on pulmonary function.

From at least one perspective, the overall results of research involving air pollution are good news — the control of air pollution represents an important opportunity to prevent disease. Air pollution is just one of many risk factors for pulmonary and cardiovascular disease, but it is one that can be modified. In the United States and elsewhere, commendable progress has been made on improving air quality and, with regard to fine particulate pollution, new standards have been implemented (Fig. 1). Extremely high concentrations of air pollution remain in many areas of the world, and decreasing these concentrations offers substantial opportunities for disease prevention. As efforts to reduce air pollution progress, debates over the relative benefits and costs associated with additional marginal improvements are inevitable. Nevertheless, continued efforts to improve our air quality are likely to provide additional health benefits.

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Allergen Avoidance to Reduce Asthma-Related Morbidity

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Central to the reduction of the severity of allergic disease is a decrease in — and preferably the removal of — the offending environmental allergen. Such allergen avoidance is particularly relevant to the successful treatment of allergic asthma. However, statistically significant reductions in such asthma-inducing allergen concentrations have been difficult to accomplish. Until recently, strategies to reduce exposure to environmental allergens have

not decreased asthma-related morbidity. In fact, a meta-analysis failed to demonstrate the efficacy of any environmental-control measures in reducing the severity of asthma.¹ Such interventions, however, have usually focused on a single maneuver² — for example, the use of semipermeable bedcovers to exclude dust mites, floor polishing, or the use of high-efficiency particulate air filters — but have not been accompanied by detailed educational pro-