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## Uncertainties relating to the health effects of particulate air pollution: The US EPA's particle standard

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### Abstract

Although the epidemiologic associations between urban particulate air pollution and human mortality and morbidity have been accumulating for several years, the causal agents (a specific chemical component, a specific particle size range, one or more pollutant combinations, etc.), and the physiological mechanisms behind the associations have yet to be identified. Significant questions regarding confounding effects due to weather, indoor air pollutant exposures and co-pollutants (that accompany particulate matter) stubbornly remain. The events in the United States began with recent epidemiological associations, followed by a lawsuit forcing the US EPA to accelerate the standard-setting process, and finally controversy over the scientific basis of the new standard. In contrast to the potential risks posed by particulate air pollution, many of the sources of such particles are positive contributors to human health; control measures to meet the proposed standard may therefore produce offsetting enhanced mortality and morbidity. In order to establish the information required for well-informed public health policies, a substantial research program is needed because of uncertainties relating to, the affected individuals, the potential causal agents, and the consequences of particle-control activities. Not only are the remaining scientific questions significant, but the particle exposure/health effects associations also call into question some of the current scientific assumptions relating to the nature of effects of population exposures to low concentrations of pollutants. © 1998 Elsevier Science Ireland Ltd. All rights reserved.

*Keywords:* Air pollution; Inhaled particles; EPA air standards; Research needs; Particle epidemiology; Particle toxicology

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### 1. Introduction

The implications of the recently-adopted US EPA particulate air pollution air standard, both for direct improvements in human health and for adverse economic impacts, are substantial. Fur-

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thermore, these standards present important challenges to several groups, including scientists, public officials and environmental advocates. Scientists are challenged to generate and interpret the relevant research data base, to provide criteria for establishing valid cause-and-effect relationships, and even to re-evaluate the common assumption that 'less is better' when air contaminant exposures are considered. Public officials also are challenged to properly use the available science in their deliberations, and to set aside political expediency when public welfare is at stake. Environmental advocates are likewise challenged to forgo short-term gains, when achieving these gains may adversely impact future public health and welfare. The consequences of failure of these three groups to meet their challenges include the basing of new air pollution standards on inadequate scientific knowledge, the possibility of increasing human suffering, and even a deterioration of the role that science will play in future public-health policy decisions. These consequences are not limited to the United States of America; they are world-wide in scope.

The chronology of events leading to the proposed particulate air pollution standard is instructive to review. In the past 10 years, several published epidemiological studies indicated a reproducible association between 'centrally-located' (at a single outdoor location in the community) measures of particulate air pollution and short-term adverse human health effects (Pope, 1989, 1991; Schwartz, 1993, 1994; Dockery and Pope, 1994; Pope and Dockery, 1992; Schwartz et al. 1996). These and other studies are reviewed by Pope et al. (1995), and the major initial findings are summarized in Table 1. It is important to note that the table relates to increments (increases over previous days levels) in particulate air pollution rather than actual air concentrations, and that the magnitude of the associations, which are statistically significant, are weak in relation to the conventionally used criteria to establish causality (Taubes, 1995). Yet, when the small relative risks are multiplied by a potentially-exposed population of millions of US citizens, then tens of thousands of 'excess' deaths are projected. Such an extrapolation was performed, and it led to wide-

spread press coverage in both lay publications as well as widely-read scientific journals. The press coverage was followed by a lawsuit in which the American Lung Association forced the US EPA to accelerate its assessment of the adequacy of the current particulate air standard. In 1996, the staff of the US EPA proposed a stringent new standard that was based on a smaller upper particle size limit ( $2.5 \mu\text{m}$ , as opposed to the older  $10 \mu\text{m}$  standard), and after considerable controversy and debate, the EPA Administrator issued the standard in 1997. This standard, which was subsequently supported by the US President in June 1997, has been challenged by several city mayors, state governors, federal legislators, US industry, and many prominent scientists. Many organized environmental groups, other elected officials, and numerous scientists, have argued in support of the new air standard.

## 2. Challenges to the epidemiological findings

As presented at two international colloquia on the health effects of particulate air pollution (Phalen and Bates, 1995; Lee and Phalen, 1996), challenges to the scientific validity of the epidemiological findings have been substantial. First, since the air pollution samplers are located outdoors, and people spend the majority of their time in buildings, how could the incremental changes in outdoor pollution be a major cause of deaths

Table 1  
The early epidemiological associations for  $10 \mu\text{g}/\text{m}^3$  increments in particulate air pollution

Mortality increase	
Total: 1%	Respiratory: 3%
Hospital admissions and visits	
Respiratory: 1%	Asthmatics: 3%
Other associations	
Asthmatic attacks: +3%	URT symptoms: +0.7%
Cough: +2.5%	Lung function: -0.1%
LRT symptoms: +3%	

LRT, lower respiratory tract and URT, upper respiratory tract.

and illnesses? Second, there is an absence of clinical plausibility in that no known clinical syndrome could produce these effects at such low levels of pollutants. Third, seasonal and weather-related factors are large confounders, for which corrections are difficult to establish. Fourth, the epidemiological models used can overestimate the effects of any given pollutant if a combination is producing the effects. Fifth, the same data, models and methods can be employed to arrive at negative results if the analyses are performed with different choices in lag times, seasons and types of effects. Sixth, errors in exposure assessments for individuals can lead to the appearance of a no-threshold for effects. Seventh, the toxicology data base does not support the epidemiology for effects at such low levels of soot or other particles. Eighth, the effects are invariant in relation to the actual levels of exposure (as opposed to increments in past days exposure), so a true dose-response relationship is not evident.

At this time, these and other issues are being vigorously debated, the US public and congress are being heavily lobbied, and nations all over the world are watching the events unfold.

### 3. The proposed particle air standard in perspective

The US EPA, under the Clean Air Act, must at five-year intervals review the scientific literature on selected 'criteria' air pollutants, and set National Ambient Air Quality Standards (NAAQS) that will protect 'particularly sensitive citizens' while allowing an 'adequate margin of safety'. Particulate matter is a criteria pollutant that has had PM<sub>10</sub> standards of 150  $\mu\text{g}/\text{m}^3$  averaged over 24 h and 50  $\mu\text{g}/\text{m}^3$  annually averaged. PM<sub>10</sub> is that mass per  $\text{m}^3$  of air sampled that passes through a pre-collector with a 50% efficiency at a 10  $\mu\text{m}$  aerodynamic diameter (AD). Thus, PM<sub>10</sub> includes particles up to about 10  $\mu\text{m}$  AD, and PM<sub>2.5</sub> includes particles up to about 2.5  $\mu\text{m}$  AD. The newly-adopted PM<sub>2.5</sub> levels are 65  $\mu\text{g}/\text{m}^3$  over 24 h and 15  $\mu\text{g}/\text{m}^3$  annually. The old annual PM<sub>10</sub> standard is to remain.

These ambient air standards can be compared to the occupational exposure standards established by the US Occupational Safety and Health Administration (OSHA), which are 10000  $\mu\text{g}/\text{m}^3$  for total particles not otherwise classified (PNOC) and 3000  $\mu\text{g}/\text{m}^3$  for respirable PNOC. Worker's exposure is 40 h per week for 50 weeks per year, for several decades at least. In terms of the ratio of parts of pollutant by mass per unit of mass of air, the adopted annual PM<sub>2.5</sub> standard is 0.000000012. If the adopted PM<sub>2.5</sub> level of particulate matter (15  $\mu\text{g}/\text{m}^3$ ) is breathed for 70 years, one would expect a total deposition in the deep lung of about 1 g. Assuming that this is spread over about 70  $\text{m}^2$  of lung surface and over 90% will be removed by normal clearance mechanisms, one concludes that the doses received must be extraordinarily small. Smokers who inhale can have daily particulate mass depositions on the order of 1 g. Comparisons such as the foregoing support the notions that the epidemiological associations between particulate matter and mortality and morbidity may indeed be difficult to explain with the existing data base in toxicology.

Another perspective on the adopted standard derives from considering the types of activities that produce PM<sub>10</sub> and PM<sub>2.5</sub> (Table 2). All of these activities are important for maintaining human health, so their curtailment can be expected to produce excess mortality and morbidity as well due to loss of jobs, cost of food, shelter and electricity, etc. Whether or not the adopted standard represents a favorable trade-off is clearly debatable.

### 4. The big questions and needed research

Among the substantial uncertainties and questions regarding the linkage between PM<sub>10</sub> or PM<sub>2.5</sub> and human health are the following.

1. Who (what sub-population(s)) is actually harmed?
2. What property of PM<sub>2.5</sub> is harming them, (mass, metals, acids, reactive species, a gas co-pollutant, etc.)?
3. How are people harmed by these minute concentrations, if indeed they are?

Table 2  
Particle sources and their benefits

Source	Associated benefits
Electric power plants	Affordable electricity for lighting, air-conditioning, heating, food refrigeration, and production of goods and services.
Diesel engines	Trucks, trains, ships, and equipment for farming, construction and mining are important for economic viability.
Farming, construction, manufacturing and mining	All provide needed food, shelter, goods and jobs.
Waste combustion	Necessary for environmental preservation and quality of life.
Spraying operations	Paints, pesticides, disinfectants and similar products protect structures and food supplies, and prevent disease epidemics.
Natural phenomena	Natural biological, geological and hydrological processes are necessary for a habitable planet.

4. Will additional control of particle sources decrease or increase human mortality and morbidity?
5. Can particle levels ever be made low enough so that even the most sensitive people will not be harmed?

At the two previously-mentioned colloquia, an attempt was made to identify the research projects needed to understand the epidemiological associations (Phalen and McClellan, 1995). A sample of the projects suggested by the attendees, after hearing over 120 papers, is summarized in Table 3. It is clear that such research is needed before final judgements can be securely drawn (Wolff, 1996).

It is useful to engage in speculation concerning some of the key assumptions that are made regarding the toxicity of environmental contaminant exposures of human populations. First, it is commonly assumed that any exposure to a contaminant will produce adverse effects. Yet, it is also possible that variations in environmental

quality are essential for maintaining effective defenses against unavoidable exposures to infectious agents, allergens and other disease-producing aerosols. If this is true, it is yet another example of the principle of biological parsimony. That is, if a physiological system is not exercised, it will diminish over time. Another common assumption that might be questioned is that multiplying a small risk by a large at-risk population will produce a number that can be interpreted as a census of victims; that is, a body-count. When the relative risk may not represent a true cause-and-effect relationship, the body-count can be misleading, or even totally fictitious. Also, this 'low-risk, large population' problem implies that a further reduction in the risk will result in a net saving of lives. Clearly, if trade-offs are considered, this may not be the case (Arrow et al., 1996). It is apparent that the particulate air pollution issue has ramifications that cover the worlds of public health, politics, and science, and that these are all intertwined.

Table 3  
Selected needed research on particulate air pollution

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|-------------------|--|
| (A) Epidemiology  | Longitudinal, panels involving sick and well with personal exposures, not just 'central' samplers<br>Effects of long-term exposure with assessment of personal exposures<br>Effects of exposure assessment errors on epidemiological findings<br>Cost-benefit analyses |
| (B) Toxicology    | Mechanism of action including cardiac and pulmonary effects<br>Roles of specific chemicals including metals, ions and reactive species<br>Roles of particle mass, surface area and number<br>New, compromised animal models and studies                                |
| (C) Sampling etc. | Improved equipment and procedures for size, composition, etc.<br>Smaller, cheaper equipment for personal exposure assessments<br>Additional data on PM size and chemistry<br>Indoor/outdoor and personnel exposure characterizations                                   |

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