The Annapolis Center
For Science-Based Public Policy

The "Particle Wars" and a Path to Peace

KEY FINDINGS:

- Airborne particles differ substantially in terms of sources and their physical and health-associated properties. No one knows whether controlling any of them will actually yield net benefits to public health. Further regulation of PM is thus premature.

- The statistical methods used to study short-term (days to weeks) health effects have recently been found to entail substantial uncertainties. Since the findings of the few available long-term (cohort survival) studies are quite disparate, there is no overall consensus on either the "responsible" air pollutants or on their importance to public health.

- To benefit public health, environmental research should be separated from environmental regulation, and the research resources committed must be commensurate with the purported risks to public health.

"Our greatest responsibility is to be good ancestors."
…Jonas Salk

The "Particle Wars" and a Path to Peace

By Frederick W. Lipfert, Ph.D

The medical, environmental, and political news of late has been full of discussions about airborne particles, particulate matter, and aerosols, all of which may be lumped together under the general rubric "PM". For example, in discussing the new Information Quality Act in *The Lancet,*[1] Ashraf said: "Fine particles have been linked to heart and lung disease but industry experts have challenged the EPA [U.S. Environmental Protection Agency] and said that the rule[2] is too broad and that the agency should assess which type and size of particles are linked to poor health. Public health experts and supporters of the EPA say the additional studies would take years while people are dying from the effects of pollution right now." Some background is required to understand the nuances involved here and how these "particle wars" are being fought in the technical literature, at scientific conferences, and in the courts. Key questions include: (1) Who is at risk? (2) Which pollutants are implicated for specific types of health effects? (3) Should all types of particles be given the same emphasis? (4) What assurance do we have that further controls on air pollution (specifically PM) will actually improve public health?

The following sections present brief summaries of the way air quality is regulated in the United States, the evidence implicating PM with various types of health effects, the sources of conflict with respect to the new regulation for fine particles, and possible ways to reduce or eliminate such conflicts, i.e., a "path to peace".

Background

What is PM, anyway, and how is it regulated? The airborne particles in question come from man-made and natural sources, are often submicroscopic, and may include such diverse types as smoke from wood fires and diesel engines, other combustion soot, pollen, hair spray, or dust from traffic, demolition, or agriculture. The Clean Air Act (CAA), under which air pollution is regulated in the United States, is based on operational definitions according to the method of measurement. Thus, for example, for regulatory purposes, sulfur dioxide is not defined by its chemical formula (SO_2) but by its EPA-approved method of measurement. As a practical (and legal) matter then, an air pollutant is what EPA says it is. PM has been measured according to particle size as $PM_{2.5}$ (very small or "fine" particles), PM_{10} (including somewhat larger or "inhalable" particles), and as total suspended particles (TSP, virtually all airborne PM). PM is also determined according to chemical properties, such as elemental carbon (BS, British or black smoke), sulfur content (SO_4, sulfate), or various metals and other compounds.

PM measurement methods have evolved greatly over time and differ in various parts of the world, although EPA-approved methods (Federal Reference Methods) are often adopted by other countries. The earliest methods passed a sample of ambient air through filter paper and estimated the concentrations of PM according to the blackness of the resulting stain. This method was effective in the mid-20th century when coal smoke from factories, railroads, and home heating was dominant, but is much less effective with today's cleaner atmospheres. This "smoke" measurement method has essentially been abandoned in the U.S., where gravimetric methods have long been the standard, beginning with a network of "total suspended particulate"

(TSP) samplers that were deployed nationwide in the 1950s. Part of the motivation then was to check for radioactive fallout. These samplers were essentially inverted vacuum cleaners that collected all sorts of particles on a glass-fiber filter without regard to their size or inhalability, usually on a 6- or 12-day sampling schedule of 24-h samples. Chemical analysis of the collected mass was done routinely to assess average composition for each sample. In 1987, the PM collection system was modified to restrict the catch to those smaller particles most likely to be inhaled, denoted PM_{10} since about 50% of them have an aerodynamic diameter less than 10 micrometers (millionths of a meter [μm]). Unfortunately, routine analysis of chemical composition was substantially curtailed then also, and a 6-day sampling schedule became the usual practice except where ambient standards were not being met.

As PM_{10} data were collected and concentrations were compared to the ambient standard, it became clear that most of the violations of standards were in arid western locations and that they could be attributed to fugitive dust, which is the major source of PM_{10} emissions. Some Western cities complained that their street-cleaning costs would skyrocket in order to meet this standard. Further, it had long been realized that only much smaller particles were likely to penetrate deep into the lung, where they might lead to more serious health effects. This, in combination with EPA’s long-standing desire to reduce PM-forming emissions from combustion sources such as power plants, led to the promulgation of a new ambient standard for $PM_{2.5}$ (otherwise known as “fine” particles) in 1997. The difference between PM_{10} and $PM_{2.5}$, which is now to be determined by subtraction instead of by direct measurement, is denoted here as “coarse particles” (CP).

Establishing a new health-based air quality standard requires EPA to publish a comprehensive state-of-science report called a “Criteria Document” (CD) that presents what is known about the properties, sources, and effects of that pollutant (See Reference 3, for example). This is done for each pollutant, one at a time, even when the main sources to be regulated emit several pollutants simultaneously. Each draft CD is subject to public comment and to approval by an EPA-appointed review board of independent experts, in this case, the Clean Air Scientific Advisory Committee (CASAC). The new ambient standard is to be set at a level that protects the public from adverse health effects with an adequate margin of safety, based on the information in the CD. CDs for regulated air pollutants are supposed to be updated every five years, but in practice, it is usually longer.

Under the CAA, promulgation of a new ambient standard triggers the following steps:

1. Ambient air quality data are collected to determine the localities where the standard is met and those where it is not.
2. Where it is not met, plans must be formulated to reduce specific source emissions sufficiently to achieve such compliance.
3. If these plans are unsuccessful or unduly delayed, sanctions may be imposed on the area that could impede economic development, and EPA can implement its own compliance plan.

For the purposes of this discussion, it is important to note that improvements in public health, which are the primary motivation for new standards, are not an explicit part of the subsequent regulatory process. The regulatory goal is to achieve compliance with standards, not to assess their actual benefits.

What Is Known About the Health Effects of PM?

The toxicological evidence for health effects from the types of particles most commonly found in ambient air at present concentrations is quite limited. For example, exposure to high concentrations of quartz and silica (so-called crustal materials) can cause respiratory disease, asbestos can cause lung cancer, pollen can cause allergic responses, and metals can have various adverse effects. However, long-term experiments with dogs exposed to sulfate particles failed to show serious health effects. The situation is complicated because the air we breathe daily may contain any or all of these particles in varying amounts, but at much lower levels than those required to demonstrate toxicological responses.

The recent draft of EPA's PM CD[3] cites numerous toxicological studies where animals were exposed to concentrated ambient particles. The most notable feature of these studies is the lack of a severe response to any of these exposures. However, the epidemiological literature indicates that exposure to current ambient concentrations of air pollution (including PM) can result in a variety of adverse health effects, ranging from minor changes in symptoms to premature death, primarily in susceptible individuals. Their interpretation is complicated by the lack of uniqueness: air pollution is never the only factor contributing to a given disease or death. Typically, the associations defining air pollution health effects are not unique to specific diseases or pollutants, in contrast to occupational effects like asbestosis, beryllium disease, or vinyl chloride (liver cancer).

In contrast with PM, the mechanisms of health effects attributed to most other regulated pollutants are reasonably well known as a result of toxicology studies under controlled conditions. Carbon monoxide (CO) interferes with the oxygen-carrying capacity of blood. Ozone (O_3) and other photochemical oxidants can cause inflammation. Sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) are primarily respiratory irritants. Lead (Pb) is a neurotoxicant that can cause brain damage. However, because PM can comprise many different substances and can also act as a vehicle to transport surface-adsorbed gases into the respiratory tract, no such simple descriptions of its physiological response may be possible. An exception may be the triggering of the cough reflex in the upper respiratory tract by inhaled particles. A further important distinction is the lack of a reliable biomarker for exposure to PM. For example, exposure to CO creates a change in blood chemistry (carboxyhemoglobin content) that is a reliable indicator of cardiovascular response, and the Pb content of blood or bone has been linked to neurological responses. No such specific exposure indicators are currently available for PM.

The epidemiology of air pollution health effects is conveniently classified according to the time scale of action (acute vs. chronic), the diseases involved, and the ages of those most affected. Thus, the case for causality should be considered across all of these strata in relation to the specific attributes of each pollutant, exposure patterns, and likely physiological responses. Note that for epidemiology studies in community settings, PM is never the only air pollutant involved, so that determining the PM share of responses to the mixture is ambiguous at best.

Acute responses. Responses to transient perturbations in ambient air quality have been reported in association with all criteria air pollutants, as well as to hydrocarbons, aeroallergens, and weather changes, including thunderstorms. Such responses include mortality, hospitalization and emergency room usage, respiratory symptoms, and lung function. However, the magnitudes of such transient lung function excursions are generally well below those that might be considered clinically significant; mortality and hospitalization are clearly the most serious types of responses, by virtue of their irreversibility.

Most of the acute studies show that the ambient air quality situations that are typically implicated are unexceptional and do not involve unusually high levels. This leads to an important question that is seldom discussed:

1. ***Why should a given individual die or seek hospitalization on a given day in response to an air pollution exposure that he/she has experienced many times before?***

The immediate answer to this conundrum relates to the fact that none of these putative responses to daily air quality perturbations have been observed directly. They all result from complex statistical modeling that is sometimes referred to as “black-box epidemiology,” since few practitioners have a complete grasp of all of the computational details. The seriousness of this situation was recently highlighted by the discovery of errors in the software that has been widely used in analyzing acute responses.[4] Note that the mortality effects that were reported in a large U.S. multi-city study[5] are now much less than 1%, which is smaller than the mortality perturbations seen in conjunction with sports contests[6] or birthdays.[7] At the most recent review of EPA’s latest Criteria Document for PM, these computational errors were discussed and the mathematical modeling of such acute health effects was described as “more of an art than a science”, especially with regard to questions involving adjustments for seasonal cycles in both health and air quality. As a result, several different types of models are now being used and the uncertainties in these acute responses have widened. EPA and CASAC have identified about 40 key epidemiology studies that are being reanalyzed using more appropriate models and modeling criteria. Until these results are received, peer-reviewed, and fully discussed, conclusions about acute health effects are premature. An initial report[8] on this subject was recently released.

However, with respect to acute effects on mortality, an alternative scenario is associated with the underlying health status of the individuals involved, for which no direct observations are available. Recent epidemiological studies of specific groups of individuals illustrate this premise quite clearly, notwithstanding the numerical uncertainties discussed above. A study of sudden death in previously healthy individuals showed nearly significantly *fewer* deaths associated with fine PM[9], while a similar study of previously hospitalized COPD patients showed very strong positive associations (more deaths) with air quality.[10] Unfortunately, individual exposures were not estimated in either study.

The inability of most of the analyses of acute responses to address this question of individual susceptibility results from their ecological design, in that only group (population) exposures and responses are involved, even though the deaths (and probably the hospitalizations) involve different individuals each day. Further, most of the existing studies of personal exposures have focused on the general public rather than on the most susceptible individuals, whose specific exposures remain unknown. Toxicology and controlled exposure studies have shown that healthy individuals are not at risk from such ordinary daily perturbations in ambient air quality, which is an important public health context that has not been emphasized.

However, recent studies in the literature address this question and provide a possible plausible scenario for acute mortality responses. Frank and Tankersley[11] present experimental animal data showing the increasing inability of aging animals to control homeostasis as death approaches. As such, this theory represents a physiological explanation of the "population at risk" concept that was quantified by Murray and Nelson[12] and by Smith et al.[13]. Both of those studies of mortality displacement or "harvesting" showed that this at-risk sub-population of the frail elderly comprises a tiny fraction (~0.2%) of the total population aged 65 and over. A measure of the life expectancy of this subgroup is obtained by dividing the frail sub-population count by the appropriate daily mortality rate. Both the Smith et al. and Murray and Nelson analyses derived life expectancies for this frail sub-population of about 1-2 weeks, and the estimated effects of air pollution on these life expectancies were small, representing reductions of the order of a few days. The Montreal study of Goldberg et al.[14] that linked prior illnesses in individuals with subsequent mortality showed that certain diseases were important precursors of pollutant-associated mortality.

This scenario of loss of homeostasis followed by an acute response to essentially ordinary levels of air quality is consistent with all of the key features of the extant time-series health-effects studies:

1. Responses within a few days of exposure, often on the same day.
2. Similar responses to most pollutants, including both fine and coarse PM and various gases.
3. Similar responses to other types of atmospheric disturbances, including hot days and changes in atmospheric pressure.

There are thus several overarching conclusions about acute responses to air pollution:

1. The magnitudes and significance of such responses have been overstated in the past.
2. The most appropriate statistical models have not yet been defined.
3. The public health significance of the putative effects on mortality is likely to be minimal.

Long-term (chronic) responses. Studies of long-term responses to air pollution consider time scales of years or more, but only a few such toxicology and epidemiology studies are available. However, it is important to understand that human exposures to ambient air pollution differ markedly from those used in typical long-term toxicological studies, for which precisely controlled exposures are typically used.

Although long-term exposures are often characterized in terms of concentrations averaged over the duration of the appropriate period, people are not actually exposed in this way. First, most people spend up to 95% of their time indoors, especially the elderly, and indoor air quality may differ substantially from that recorded by centrally located routine (outdoor) ambient monitors.

In general, long-term exposures comprise a series of irregularly spaced peaks, due to the combination of meteorological variability and changing activity patterns. To the extent that human respiratory defenses are effective, only the peak exposure levels may be important. Thus, studies of long-term effects should not be confined to considering only long-term averages in air quality, which presupposes a linear response. Further considerations are the fates and cumulative effects of inhaled pollutants having different chemical and physical properties.

For both intermediate and long-term responses, the major question is:

What are the mechanisms for responses to repeated acute responses over time scales of months to years?

Its corollary question is:

Are the indicated long-term responses bona fide chronic effects or the time-integrals of acute effects?

Acute and long-term responses may differ physiologically. Further, all pollutants have been implicated in the (acute) time-series studies, but EPA claims that only $PM_{2.5}$, sulfate aerosol (SO_4), and SO_2 are reliably implicated by long-term cohort studies, although other long-term studies have found effects for gases. While time-series studies usually find the largest effect estimates for the elderly and for respiratory disease deaths, cohort studies do not. Thus, coherence between acute and chronic studies depends on the studies that are given the most credence.

According to the exposure considerations outlined above, long-term responses must largely involve repeated (acute) rather than steady (chronic) exposures. Thus, the body of epidemiological findings must be considered jointly with pollutant dosimetry and toxicology findings in order to create a plausible scenario of pollutant build-up (increased body burden) and subsequent responses to soluble and insoluble PM (and other pollutants).

Long-term studies are of two general types: cohort studies and ecological studies. They vary greatly in their treatments of exposure, both with respect to spatial and temporal scales. EPA emphasizes the American Cancer Society (ACS) studies, by Pope et al.,[15] as reanalyzed,[16] and a recent update.[17] No retrospective exposures were considered in these studies, which is inconsistent with the latency periods required for chronic disease development. This alone makes it inappropriate to describe the ACS or Six City[18] cohort study findings as implicating new cases of chronic disease. On the other hand, historical exposures were specifically considered in the Veterans' Study[19] and in an ecological study[20], with little evidence of cumulative or historical effects.

The one consistent trend in three of the long-term mortality studies is a decrease in effect estimates over time. Decreasing trends from 1960 to 1990 are seen in responses to TSP and SO_4 and from 1980 to 1997 for PM_{10} and $PM_{2.5}$ in the ecological study. While the updated ACS study did not specifically discuss time trends, the responses for the 1982-98 mortality follow-up period were substantially lower than for 1982-89. The important conclusion that follows from considering all of the long-term mortality studies is that results for specific cohorts and/or time periods should not be applied to other populations or periods. In this sense, the heterogeneity in long-term air pollution effects follows that seen in the time-series studies.

Long-term health effects must be deduced from differences in discrete populations, averaged over time. Such studies are called "cross-sectional" and must account for all other potentially confounding factors that may differ among populations, including long-term trends.

Socioeconomic effects are among the most difficult to quantify. For example, people living near a major pollution source are likely to differ from those living in more distant affluent areas in many ways, including education, income, and access to medical care. "Smokestack" communities with declining industries are likely to have lost population, while Sunbelt and communities with cleaner industries may have gained population. Studies have shown that such situations may involve selective migration, in which the poor and the sick are often left behind in the distressed communities.

A final question on long-term health effects is:

How coherent are the various studies and how do overall effect estimates compare by pollutant?

These questions have not yet been really considered by EPA, because they do not give all studies equal consideration and have not considered gaseous pollutants. The following table presents averages over all of the available long-term mortality studies, including those of the California Adventist Health Study.[21-23] Note that the Veterans' study[19] pertains only to males; studies that did not distinguish by gender were applied to both males and females. The table entries are the percentages of all-cause mortality associated with the specified concentration increments for each pollutant.

Table 1 Synthesis of long-term pollution effects for all causes of death

pollutant	concentration increment	males effect(%)	males SE (%)	females effect(%)	females SE (%)
$PM_{2.5}$ (μg/m^3)	10	1.88	1.40	***3.51***	0.61
CP (μg/m^3)	10	-1.35	2.86	4.70	3.84
PM_{10} (μg/m^3)	20	0.60	2.16	***3.94***	1.49
TSP (μg/m^3)	35	1.21	0.97	2.01	1.05
peak O_3 (ppb)	100	***8.06***	2.40	***4.50***	2.10
avg NO_2 (ppb)	25	***5.08***	1.80	***4.53***	2.01

bold italic denotes statistical significance among studies

These cross-study averages and their standard errors show that NO_2 and peak O_3 may be more important than PM, at least for males. Note that multi-pollutant models have not been used extensively in these long-term mortality analyses and that such modeling will be required to help define the separate contributions of each pollutant.

Current Issues of Conflict, i.e., the “Battlegrounds”

While most of the protagonists may agree broadly that air pollution can adversely affect health at sufficiently high exposures, they diverge on the important details, as stated above: (1) Who is at risk? (2) Which pollutants are implicated for specific types of health effects? (3) Should all types of particles be given the same emphasis? (4) What assurance do we have that further controls on air pollution (specifically PM) will actually improve public health?

Susceptible subpopulations. There seems to be broad agreement that healthy adults are not susceptible to the acute effects of air pollution, but that the frail elderly may be. This is consistent with many years’ experience with the effects of severe heat waves, for example. Susceptible subpopulations for long-term effects of air pollution are less well established; although reanalysis of two cohort studies[16] has indicated that most of the excess mortality is found in persons with less than a high-school education, this finding raises questions about the degree of socioeconomic confounder control in those studies.

In addition, EPA has made children's health a priority issue in recent years. A relevant question is thus:

Are U.S. children a generally susceptible subgroup under current conditions?

EPA has consistently overstated the case for PM as a causal factor in children's health. For example, an in-house EPA study[24] has been cited as establishing PM effects on U.S. infant mortality, along with foreign studies under much more polluted conditions. However, a recent reanalysis[25] of the EPA study, while replicating its results as a point of departure for the reanalysis, found that the EPA results were not robust and that the most important air pollution effect on infant mortality was a strong *protective* effect of sulfate aerosol. As a result, there is no reliable evidence linking current levels of U.S. air quality with increased infant mortality.

Ritz et al. have investigated effects on birth defects[26] and low birth weight[27] in Southern California. These studies did not consider maternal smoking, which could be an important confounder in these cross-sectional studies. The birth defect study implicated CO but not PM. In the birth-weight study, results were only given for CO; PM was not considered.

Lung function growth is another endpoint that might have implications for children's health. Gauderman et al.[28] considered longitudinal changes in lung function for 4th, 7th, and 10th grade children in Southern California. Significant pollution-related deficits were seen in 4th graders only, and the most important pollutants were coarse particles (CP), PM_{10} and NO_2 (and not $PM_{2.5}$). This study design did not consider other community-specific attributes as potential confounders of the indicated effects of air pollution. In a follow-up study of a second 4th grade cohort,[29] different pollutants were implicated. Avol et al.[30] considered the effects on lung function of residential relocation to areas of differing air quality, for 110 Southern California children. This is a more reliable study design than the cross-sectional comparisons by community, since each child serves as his own control. However, the maximum change in annual average PM_{10} in this study was 90 μg/m^3, while national annual average data on PM_{10} for 1999 suggest that 30 μg/m^3 is about the lowest change that might now be expected. As a result, the corresponding lung function decrements would thus be 0.15% in vital capacity and 2.1% in peak flow. Such small decrements are of doubtful clinical significance.

Studies of childhood asthma have generally shown that some air pollutants are among the many agents that can trigger an asthma attack, but that there is no reliable evidence establishing air pollution as an underlying cause of the worldwide increase in asthma prevalence.[31]

In summary, the evidence for serious children's health effects at current U.S. air quality levels must be considered problematic.

Identifying which of several correlated agents may be responsible. The existing literature is ambivalent on the question:

Which pollutant(s) or constituents of PM are primarily responsible for the health effects indicated by epidemiological studies?

Much of the literature uses relative statistical significance (p-value) as an indicator of the most important pollutants(s). This reflects a naive approach to model specification in regression analysis, since the relative standard error of a regression coefficient is affected by sample size and by the range of the variable, as typically indexed by its coefficient of variation (CV). A relatively constant pollutant would not be a good predictor in an epidemiological study, regardless of its underlying toxicity. The more episodic pollutants, like H^+, $PM_{2.5}$ or peak O_3, will tend to have larger CVs than, say NO_2 or coarse particles, and thus will have an advantage in regression analysis that is unrelated to potential toxicity.[32] Measurement error will tend to increase standard error and decrease significance. These attributes can become even more important in multiple-pollutant regressions involving correlated pollutants (as most of them are, after seasonal adjustment). Such effects can result in an apparent "transfer of causality."[33] Single-pollutant effect estimates may reflect their correlations with other pollutants that may be more toxic, rather than their intrinsic toxicity per se. For these reasons, selecting the real agent(s) from a field of candidates is not a straightforward process and certainly cannot be reliably based on statistical significance alone.

Are all particles alike? One of the important characteristics of PM is heterogeneity. PM is the only criteria air pollutant that has no chemical definition. The traditional (and current) regulatory approach is to treat everything that is collected by the Federal Reference Method the same, on a mass basis; a microgram of plutonium is given the same emphasis as a microgram of gypsum, for example. A recently retired EPA scientist chided the Agency for this counterintuitive approach: “Perhaps it is time for the Agency to consider fostering such a novel approach that actually incorporates the relative toxicity of ambient PM species into the standard setting process, as recommended by NRC….”[34]

Ambient particles not only vary substantially in their inherent toxicity, they vary in size, solubility, bioavailability, probability of exposure and cost of control. Lumping them all together makes it easy for the pollution control agencies, not only in terms of cheaper and easier measurements, but also in terms of devising strategies to achieve the overall ambient standard. With the previous PM_{10} standard, it was possible to trade a microgram of street dust against a microgram of wood smoke, even though these two substances may exhibit very different health effects. With the advent of the new $PM_{2.5}$ standard, some of this flexibility has been reduced, but such counterintuitive tradeoffs would still be allowed within PM size classes.

As mentioned above, data on the health effects of specific types of particles are sparse, in large part because of the lack of ambient monitoring data. However, some estimates have been made of the costs of control ($/additional ton removed for specific control technologies, relative to existing practice[35]). These estimates vary by several orders of magnitude and dramatically illustrate the need for such particle-specific information in order to design a cost-effective overall control strategy (Table 2).

Table 2 Some estimated costs of PM controls (1990$)

source	**technology**	**cost/ton of PM**	**remarks**
reformulated gasoline	fuel supply	$755,000	other pollutants are reduced
heavy-duty diesels	retrofit	25,500	
industrial boilers (gas)	fabric filter	13,300	
industrial boilers (oil)	fabric filter	10,700	
industrial boilers (coal)	fabric filter	4,200	
industrial boilers (wood)	precipitator	1,900	
utility boilers (coal)	fabric filter	2,700	
material handing (minerals)		1,800	
construction activities	dust control	3,600	
agricultural burning	using propane	5,200	
agricultural tilling	soil conservation	138	
residential wood burning	education	1,300	
paved road dust (vacuum)	road sweeping	160-1070	urban areas are cheapest
paving unpaved roads		570-2350	
sulfate from small boilers	wet scrubbing	20,000	based on 50% conversion of SO_2
sulfate from utility boilers[36]	wet scrubbing	420-970	based on 50% conversion of SO_2

Using the “all particles are alike” paradigm, the cheapest PM control strategy would thus involve soil conservation, urban street cleaning, and retrofitting wet scrubbers to large utility boilers. However, there is no reliable toxicological evidence showing that reducing ambient levels of those specific types of particles would actually improve public health. A rational pollution control policy must consider relative toxicity and population exposure in addition to these estimated control costs. Unfortunately, little of this type of information is currently available.

Integrating the state of the science. An important question that transcends epidemiology per se is:

What assurance can we have that reducing ambient PM will improve public health?

This question is seldom addressed, even though it provides context for the epidemiology studies. Time-series studies reflect conditions at a given place for a specified period and are not likely to be transferable to another time or place. In general, the considerable heterogeneity among studies remains poorly explained, even at the same location. Cross-sectional studies, including cohort studies, reflect spatial gradients over a given period. Comparing the two ACS studies,[15,17] the Veterans' study,[19] and the recent ecological study[20] shows that such results are also not transferable in time or place. Longitudinal intervention studies that investigate responses to specific pollution abatement campaigns are needed to answer this question. Such studies are entirely lacking in the draft CD.[3]

Some “Paths to Peace”

It appears from the above that the nation has now progressed to an improved environmental status that no longer permits easy decisions on how to balance improving environmental quality for its own sake against the most cost effective ways to benefit overall public health. However, we believe that all the protagonists in the “particle wars” are well intentioned, differing mainly in their perceptions of the best ways to serve the public interest. Resolving the current sources of conflict could allow them to work together in this regard. The public wants clean air, but they also want affordable essential services.

Currently, the U.S. is experiencing a demand for increased organizational accountability at all levels. A reasonable outlook on regulation may be one that was first proposed 25 years ago: “…in a complex industrial society, a substantial measure of regulation may be necessary. We are committed, nonetheless, that this regulation should be sensible, cost-effective, and as unburdensome as the nature of its objectives will allow.”[37] These concepts lead to some “guideposts along the path to peace”:

Fair and efficient regulation. It has been generally accepted that the “polluter pays,” but this rule must also be applied fairly and efficiently to get the largest health benefit for a given overall cost to society. This means that the possible administrative and political convenience of focusing on a few large pollution sources must give way to finding those sources with the most toxic emissions that affect the most people, regardless of their size or ownership. Residential wood burning and older, heavy-polluting vehicles come to mind as primary targets.

Consideration of all relevant information. Not only must all of the relevant health studies be given fair consideration, including those that fail to find significant health effects of PM, but the possibilities of unintended consequences must also be considered. These include higher costs of essential services (electricity, transportation), possible effects on climate (which can be in either direction, depending on the type of particle), and neglecting possible health effects from pollutants other than PM.

Involvement of mainstream U.S. medical science. Thus far, the claims of serious health effects of air pollution have been proposed by a relatively small group of researchers, funded mainly by EPA. Most of these studies have been directed at specific pollutants (PM in this case), and the considerations given to confounding co-pollutants have varied widely. However, the findings of these studies have not always been interpreted the same way for all pollutants. For example, while acute studies of PM effects on mortality are viewed with alarm, the same types of studies that find similar effects for CO have been largely ignored in the U.S. Given the stakes involved here, environmental research should be refocused on specific diseases rather than on specific pollutants, so that the relevant NIH Institutes might pass judgment on the environmental claims. In effect, what is needed is to move the “trials” into an unprejudiced venue.

Accountability for the results of regulation. EPA’s mission has been focused on pollution control as an important societal good in its own right. By most accounts, it has been successful in pursuing that goal and the environment is now the better for it. But currently, the question is “how clean is clean enough?” and accountability for the underlying (health-related) goals of regulation must be considered. Failure to do so will perpetuate a never-ending cycle of conflicts and regulations that benefit only bureaucrats and purveyors of pollution control equipment. Some extreme examples are given by the future air pollution abatement scenarios proposed by the Southern Appalachian Mountains Initiative, costing up to $100 billion.[38] This amount would be enough to triple the public secondary education budgets in the eight states involved, which would surely be a better use of the funds.

Research and monitoring resources commensurate with pollution control costs. EPA is formally a regulatory agency; research and monitoring activities are designed to support that mission. These programs have been evaluated in terms of the Agency’s overall budget and not in terms of the overall costs of such regulation to society and to the economy. Institutional changes should be considered to reform this situation. Better information obtained at the cost of millions of dollars could save unnecessary control costs measured in the billions. A good place to start would be with the expansion of population-oriented air quality monitoring, including daily PM and its main constituents.

Separation of risk from blame. The “particle wars” are only one of many such environmental conflicts. Mary Douglas, an anthropologist interested in risk, has developed some interesting insights into such conflicts,[39] centered around the recent conversion of the term “risk” to equal “danger” and comparing “pollution” to “religious defilement.” She points out that, “Dangers to the body, to children, to nature are available as so many weapons to use in the struggle for ideological domination.” “It would be strangely innocent nowadays to imagine a society in which the discourse on risk is not politicized.” (p. 13) “Disasters that befoul the air and soil and poison the water are generally turned to political account: someone already unpopular is going to be blamed for it.” (p.5) “Every death and most illnesses will give scope for defining blameworthiness.” (p. 6) “The language of danger, now turned into the language of risk, often makes spurious claim to be scientific.” (p. 14) “The political pressure is not explicitly against taking risks, but against exposing others to risks.” “Of the different types of blaming systems that we can find in a tribal society, the one we are in now is almost ready to treat every death as chargeable to someone’s account, every accident as caused by someone’s criminal negligence, every sickness a threatened prosecution.” (p. 15) As in most conflicts, control of information is key. Douglas points out that “Disputes about risk have become endemic and self-generating.” (p. 14) “It is very much in the spirit of cultural theory to treat the institutions themselves as the monitors which determine what is going to count as information. … News that is going to be accepted as information has to be wearing a badge of loyalty to the particular political regime which the person supports; the rest is suspect, deliberately censored, or unconsciously ignored.” (pp. 18-19)

This reasoning explains why the debates over EPA’s official “Criteria Documents” have become so acrimonious (the latest such report on particulate matter is headed for its fourth try for public review and subsequent approval, after major revision) and why it is now so urgent to examine whether a regulatory agency should be charged and entrusted with the task of defining the supporting science.

Where Do We Go from Here?

Since the institutional reforms suggested above would not be achieved easily, what are some realistic options? If EPA has its way, it will proceed as planned to control PM as if all particles were equally harmful; any information to the contrary will have been received too late to matter, given the current pace of research and monitoring. This gives the Agency the flexibility to mandate further controls according to regulatory convenience or political expediency without regard to economic efficiency or public health benefits, since EPA is not accountable for public health. The public interest would be better served by accelerating the pace and enlarging the scope of research, increasing the coverage of detailed air quality monitoring of PM by particle size and chemistry, and by delaying implementation of any stricter ambient standards until this information has been disseminated and understood. Such a delay could be accomplished simply by setting the new ambient standard to conform with current ambient air quality levels. Accountability could be achieved by separating the research and regulatory functions, for example, by transferring environmental health research responsibilities to an agency that is formally charged with public health responsibilities. Such reforms are necessary for the survival of a credible environmental movement in the United States.

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Fred Lipfert is an independent consultant who was formerly with the Department of Applied Science at Brookhaven National Laboratory (BNL). He has degrees in mechanical engineering, aerospace engineering, fluid mechanics, and environmental studies, and has an extensive technical background in energy/environmental interactions, with emphasis on air quality. He has published over 150 scientific papers and reports, including work on air pollution effects, source emissions characteristics, energy use topics, air pollutant dispersion and modeling, and air quality characterization. His recent work has emphasized the health effects of air pollution and includes a book published by Van Nostrand Reinhold (now John Wiley and Sons) in 1994. His consulting activities have included expert testimony, analysis of health effects and risk assessment issues, and research on atmospheric damage to materials. Clients have included Federal and State agencies and industrial concerns in the U.S. and Canada. Lipfert was part of a National Research Council committee that reviewed the research program of the U.S. Department of Energy on fine particulate matter.

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