Issue Analysis

The EPA's Fine Particulate Matter (PM2.5) Standards, Lung Disease, and Mortality:

A Failure of Epidemiology

by Jerome C. Arnett, Jr.

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EXECUTIVE SUMMARY

Congress passed the Clean Air Act of 1970 based on the belief that reducing air pollution levels saves lives and improves health. The Act mandated the Environmental Protection Agency (EPA) to base its regulatory policies on good science. In 1997, EPA promulgated standards for fine particulate matter that were the most stringent and expensive in the agency's 35-year history. The standards were widely criticized, and even EPA's own science advisory committee did not endorse them. Instead of preventing 20,000 deaths and saving $69 to $144 billion a year at a cost of $6.3 billion (for partial attainment), as claimed, the standards have cost at least $70 billion a year to implement, eliminated hundreds of thousands of jobs a year, and likely have cost lives (because of the huge cost) without providing any public health benefit.

One reason for this failure of public policy lies with the epidemiological environmental studies used. Two large studies served as the scientific basis for the standards promulgated—the 1993 Harvard Six Cities Study and the 1995 American Cancer Society Study. These and other studies showed only a weak association between exposure and disease or death—an increased relative risk of 1.26 and 1.17 respectively—and yielded several discrepant results.

Epidemiology is the study of health in populations. With air pollution studies, as a practical matter, exposure is estimated at the group level, while health outcomes are measured at the individual level. In addition, observational epidemiological studies, unless they show overwhelmingly strong associations—on the order of an increased relative risk of 3.0 or 4.0—do not indicate causation because of the inherent systematic errors that can overwhelm the weak associations found. These errors include confounding factors, methodological weaknesses, statistical model inconsistencies, and at least 56 different biases.

In order to show causation, environmental epidemiological studies showing *strong* associations must be accompanied by experimental animal toxicologic studies that provide evidence for a plausible biological mechanism. But dozens of such animal studies performed over the past 30 years all have been negative.

The introduction of causal assumptions into observational epidemiological studies that show only weak statistical associations is a problem that has been recognized for many years, and has been well documented in the literature. Since this process bypasses the scientific method, it has been labeled "statistical malpractice." In addition, the improper use of science to promote political agendas is rightly considered unethical.

EPA's PM2.5 regulations are a tragic failure of public policy that are shown to have no basis in science and thus are not saving lives or preventing illness. Instead they are imposing billions of dollars of net cost each year on the American people.

Introduction

Emphysema, or chronic obstructive pulmonary disease (COPD), is a chronic lung disease that is responsible for 8 million doctors' office visits, 726,000 hospital admissions, and 119,000 deaths annually. It was the fourth leading cause of death in 1997, and it costs over $32 billion each year.[1,2] Worldwide its prevalence and resulting mortality are increasing, largely because of cigarette smoking, which is its leading cause.[3]

But over the last several decades, air pollution has become suspect.[4] Air pollution is a complex mixture of particulate matter (PM) and gaseous co-pollutants, primarily ozone (O_3), carbon monoxide (CO), nitrous oxide (NO_2) and sulfur dioxide (SO_2). The most important component of PM is the fine particulate matter (PM2.5) that penetrates deep into the lungs. It originates largely from the combustion of fossil fuels. The sulfate fraction of PM2.5 is formed by the reaction of the gaseous co-pollutant, sulfur dioxide, with ammonium in the atmosphere, to form the sulfate particulates.

The standards were criticized by many of EPA's own science advisers as soon as they were proposed and before the final rule was published.

A large number of epidemiologic studies worldwide have identified particulate matter (PM) air pollution as a cause of excess illness, and of thousands of premature deaths per year.[5] The World Health Organization has estimated that 800,000 deaths a year worldwide are attributable to it.[6]

The belief that reducing air pollution levels would save lives led the U.S. Congress to pass the Clean Air Act of 1970. Amended in 1977 and in 1990, it has six "Titles" or sections spanning hundreds of pages to implement the legal requirements, and tens of thousands of pages of "guidance" documents explaining the regulations.

In 1997, the U.S. Environmental Protection Agency (EPA) promulgated standards for acceptable levels of atmospheric fine particulate matter. It estimated that costs for "partial attainment" of the standards would be $6.3 billion per year. It predicted that keeping levels at or below the standard would prevent 20,000 deaths per year, improve or prevent respiratory symptoms in hundreds of thousands of other citizens, and save between $69 and $144 billion a year.[7]

The standards were based on environmental epidemiologic studies from the nation's best schools of public health. These standards were published in prestigious medical and health journals. But the standards were criticized by many of EPA's own science advisers as soon as they were proposed and before the final rule was published.[8] They were so controversial that even the EPA's own advisory committee did not endorse them.[9]

Some studies have concluded that the standards, although they were the most stringent and expensive in the EPA's 35-year history, instead of providing significant benefits to society, are likely to have cost money and lives. One analysis found that the benefits are likely to be only $2 to $40 billion per year, while the cost is likely to be $70 to $150 billion a year, with hundreds of thousands of jobs eliminated.[10] According to one estimate, each $15 million in additional regulatory costs results in one additional induced death.[11] The EPA this year has proposed an additional

reduction of its 24-hour l PM2.5 standard, from 65 micrograms per cubic meter (ug/m^3) to 35ug/m^3.[12] This will mean even greater costs to the American people without any corresponding public health benefit.

Epidemiologic studies used by the EPA are not suitable for determining causation, since they are unable to control for their inherent systematic errors

This paper examines the reasons for this failure of public policy. It begins by explaining how the scientific method works. Next, it briefly reviews the environmental studies that were used by the EPA, notes the weak statistical associations they reported between exposure and death, and lists some of their discrepant results.

It order to provide a background in pulmonary physiology, it describes the development and anatomy of the normal lung, as well as the changes found when emphysema occurs, and it reviews the known risk factors for the development of the disease. It explains the importance of animal toxicologic studies and points out the negative findings from these studies, which means that no plausible biological mechanism has been found to explain how PM2.5 could cause emphysema or death.

It then shows that the epidemiologic studies used by the EPA are not suitable for determining causation, since they are unable to control for their inherent systematic errors—such as confounding factors, biases, methodological weaknesses, and statistical model inconsistencies. Finally, the paper discusses the improper introduction of causal assumptions into observational epidemiologic studies, explains how this bypasses the scientific method, and notes that this misuse of statistics has been documented in the literature for many years.

The Scientific Method

The idea that reducing the concentration of air pollutants might save lives is based on ideas first proposed by the 16th century physician Paracelsus, who rejected alchemy and introduced chemistry to medicine. He observed that the toxic effects on humans from poisonous plants and animals were due to specific chemicals and that our response to these chemicals depended, in part, on the dose of the chemical received. In 1620, Sir Francis Bacon proposed methods of generating and testing hypotheses that eventually became known as the scientific method.

The scientific method is the basic tool of science that has allowed us to discover universal laws governing the world around us. In order to organize or explain data, scientists develop hypotheses, which are tested by gathering appropriate data. If a hypothesis is found to be consistent with the available data and to predict the results of validation tests, it then becomes a "theory"—the best known explanation of any phenomenon. If the theory is confirmed by additional testing, it becomes scientific fact, or "law."

The scientific method, for example, uses the Henle-Koch criteria to confirm that a specific microorganism causes a specific disease that has been observed in an animal. These four criteria are:

1. The organism in question should be shown to be present in all

cases of affected animals, but be absent in healthy animals;

2. The organism should be isolated from the diseased animal and grown in a lab culture;
3. The organism grown in the lab should cause the same disease seen in the original animal when inoculated into a healthy laboratory animal; and,
4. The organism should be re-isolated in pure culture from the experimentally infected animal.

If these criteria are met, then the hypothesis—that the organism in question causes the disease seen in that animal—has been proven scientifically. But if any of the four criteria is not confirmed, the hypothesis cannot be used to explain the observation.

In medical research using the scientific method, the randomized clinical trial is the gold standard.[13] Subjects are assigned at random to test and control groups, the test group is exposed to the risk factor in question, and both groups are followed over time to learn the result.

These studies show only extremely weak statistical associations, or correlations, between increased levels of PM2.5 and disease or death. None has found a genuine causal relationship.

Environmental PM2.5 Studies Used by EPA

Randomized clinical trials of the health effects of air pollution would be unethical and would, in any case, be too expensive and impractical for studying environmental risk factors. Hundreds of thousands of individuals would need to be followed over many years to find the few subjects that become ill. Instead, government agencies such as EPA have relied on the much quicker and cheaper observational epidemiologic studies. Dozens of these studies have been published. They show only extremely weak statistical associations, or correlations, between increased levels of PM2.5 and disease or death. None has found a genuine causal relationship.[14] However, nearly all have implied or asserted that the weak association does imply causation, bypassing the scientific method. That many of their findings are contradictory is not surprising. A few of these findings will be reviewed here. Other examples can be found elsewhere.[15-19]

The EPA's 1997 PM2.5 standards were based primarily on findings from two large studies; the 1993 Harvard Six Cities Study (HSC),[20] and the 1995 American Cancer Society Study (ACS I).[21] The HSC study followed 8,111 subjects between 1974 and 1991. It reported that the mortality rate was 26 percent higher in the city with the highest, compared with that with the lowest, level of air pollution. Confusing association with causation, the authors improperly concluded, "fine particulate air pollution…contributes to excess mortality in certain U.S. cities." Moreover, for one group, subjects with more than a high-school education, it found no association of PM2.5 exposure with mortality and found a slight *decrease* in mortality due to respiratory disease. [22]

The much larger ACS I study compared PM2.5 levels with mortality in more than 500,000 people from 151 U.S. metropolitan areas

between 1982 and 1989. It found a statistically significant increase in all-cause mortality of 17 percent for PM2.5 and 15 percent for sulfate, in the most polluted compared with the least polluted cities. Surprisingly, lung cancer mortality was associated with exposure to increased sulfate but not to PM2.5. However, the study used limited risk factor data since health information was obtained only once, at entry into the study in 1982, and it considered only a few of the 300 known risk factors that have been associated with cardiovascular disease. None of the data obtained was verified by review of medical records or by other means. Confusing association with increased risk, the authors concluded: "Increased mortality is associated with sulfate and fine particulate air pollution at levels commonly found in U.S. cities. The increase in risk is not attributable to tobacco smoking, although other unmeasured correlates of pollution cannot be excluded with certainty."

Two re-analyses of the ACS I study were published after enactment of the EPA regulations, both of which included authors from the original studies. The Health Effects Institute (HEI), a research institute funded both by the EPA and by private industry, in 2000 reanalyzed both the HSC and ACS I studies.[23] Surprisingly, the HEI study found that the pollutant most strongly associated with all cause mortality in the ACS I study was not PM2.5 or sulfate, but the gaseous co-pollutant sulfur dioxide. And when sulfur dioxide was added to the statistical model containing either PM2.5 or sulfate, the association of both with mortality became statistically insignificant. [24]

Applying another statistical model to the HSC data, the HEI study factored in migration into and out of cities and found that the PM2.5 mortality became statistically insignificant. This is surprising since those who remain behind are likely less healthy and would be expected to suffer greater mortality from the air pollution. In addition, HEI found that the PM2.5 association with all-cause mortality was true only for those with less than a high school education. The authors, confusing association with causation, concluded that the "increased relative risk" of mortality "may be attributed to more than one component of the complex mix of ambient air pollutants in urban areas in the United States."

The second reanalysis by the American Cancer Society (ACS II) was published in 2002 and studied the same population from ACS I, but extended the follow-up period from 1982 to 1998. It reported that each increase of 10 ug/m^3 of PM2.5—between the city with highest and that with the lowest pollution level—was associated with mortality increases of 4 percent for all-cause, 6 percent for cardiopulmonary, and 8 percent for lung cancer.[25] For some reason, sulfur dioxide was not considered. In their conclusion, the authors confused association with causation, stating: "Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality." [26]

A number of other studies have reported negative findings.[28,29] One example is the large 2000 Veterans Study of 50,000 male veterans, all of whom had been diagnosed with hypertension.[30] That study group should have been more susceptible than the general population to the effects of

PM2.5. The researchers reported increased mortality of these patients from their hypertension—as expected—but the overall group demonstrated a statistically significant *decrease* in mortality associated with PM2.5 exposure.

The same types of findings and the same mistaken conclusions are found in studies of other environmental pollutants, including sulfur dioxide, ozone, and nitrogen dioxide.[31-34] All of these paradoxical results strongly suggest that air pollution alone does not explain the findings.

Acute episodes of lung infection (exacerbations) worsen the symptoms of emphysema and are accompanied by increased inflammatory cells in the airways, but the role of these exacerbations in the long-term progression of the disease is uncertain.

Normal Lung Development and Changes Caused by Emphysema

The lung increases in size from birth until it reaches peak function at around the age of 20 years. Thereafter, its function slowly declines with age. The 300 million air sacs, or alveolae, in the lung, each surrounded by small blood vessels, expand to accept the oxygen-containing air inhaled through the mouth and delivered to them by the tubes, or bronchi, to which the air sacs are connected. The oxygen is then transferred to the hemoglobin of the red blood cells in the blood stream, and subsequently is circulated to all the body's tissues by the cardiovascular system.

Our current understanding is that when emphysema develops, unknown physiological factors interact with environmental exposures to cause the normal, slow decline in lung function over time to accelerate. Some of the air sacs are destroyed, and those remaining are damaged and cannot expand adequately to accept the inhaled air, so that stale air is trapped. And the tubes leading to the air sacs become narrowed, impeding the movement of air in and out of the lungs. This makes it more difficult for the patient to remove foreign particles or lung secretions by coughing. Along with these chronic structural changes the normal repair mechanisms, which help restore lung function when the lung has been injured, are impaired.

These physical changes all have been induced in experimental animal studies by exposure to toxic concentrations of inhaled foreign particles such as cigarette smoke, sand, dust, or chemicals—but not PM2.5.

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Risk Factors for Emphysema

The recognition of risk factors for a disease is important to facilitate its prevention. At present, since cigarette smoking is responsible for up to 80 percent of the cases, decreasing the incidence of cigarette smoking is the most effective strategy to prevent emphysema.[35] Cigarette smoking doubles or triples the rate of progression of the disease and is responsible for up to a 20-fold increase in the death rate.[36, 37] However,

since only 15 percent of smokers develop emphysema, other factors must play an important role.[38]

The most significant of these, undoubtedly, is genetic. Certain genes likely control cellular enzymes and other factors that lead to tissue destruction. The improper regulation of these destructive factors by defective genes may lead to the development of disease. Other genes act to protect the lungs from destructive processes. For example, researchers recently have identified a gene in mice that protects the lungs from the harmful effects of tobacco smoke and, by suppressing this gene, have increased the degree of inflammation produced by exposing the animals to cigarette smoke.[39]

No form of environmental PM, other than viruses, bacteria, and biochemical antigens, has been shown either experimentally or clinically to cause disease or death at concentrations close to U.S. ambient levels.

Less commonly known causes of emphysema are intense or prolonged exposure to occupational dusts and chemicals, such as those that occur with sand blasting or coal mining, and the exposure to allergens seen with agricultural workers. Other factors that may be associated, but whose causative role is unclear, include:

1. repeated acute lung infections, such as acute bronchitis or pneumonia;
2. exposure to environmental dust or other air pollution;
3. a deficiency of antioxidant vitamins, such as vitamins C and E; and
4. nutritional factors.

Animal Toxicologic Studies

No form of environmental PM, other than viruses, bacteria, and biochemical antigens, has been shown either experimentally or clinically to cause disease or death at concentrations close to U.S. ambient levels—even though hundreds of researchers have tried for years to demonstrate this. When environmental epidemiologic studies are used to show causation, two criteria are required: 1) a *strong* association between the disease and a risk factor (which means at least a two-fold increase in risk); and 2) a plausible biological mechanism. Animal toxicologic studies can provide the evidence for the latter.

An important principle of toxicology is that the dose makes the poison. If exposure to ambient particulate matter air pollution sickens or kills people, acute

exposure to concentrated ambient particulate matter (CAP) should sicken or kill lab

animals. Several animal species have been used in these studies, including mice, rats, guinea pigs, cats, and monkeys. A device that concentrates the pollutant 30 to 50 times greater than its level in ambient outdoor air delivers the CAP to the animal.

Dozens of these animal toxicologic studies over the past 30 years have searched for a biological mechanism, but none has supported the hypothesis that ambient PM2.5 air pollution, in the concentrations found

in the U.S., causes lung disease. Studies using animals with diseases such as asthma, bronchitis, cardiopulmonary disease, and even old age—that should make them even more susceptible to the CAP—have produced only slight effects, all of which were reversible within one day.[40] For example, exposing asthmatic mice to CAP for three days did not worsen their disease 24 or 48 hours after exposure, while exposing rats with cardiopulmonary disease to CAP produced a change in inflammatory cells that reverted to normal 24 hours after exposure.

In summary, none of the experimental animal studies has found any plausible biological mechanism to support the hypothesis that exposure to ambient atmospheric PM2.5 produces or significantly aggravates disease or causes death.

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Types Of Epidemiologic Studies and their Limitations

Epidemiology is the study of health in populations.[41] Epidemiologists collect data with poorly controlled observational studies and evaluate it using statistical models. These observational studies are best used to discover the patterns of disease that occur in human populations.[42] For example, they can find unexpectedly high rates of rare diseases such as lung cancer in cigarette smokers. They are not appropriate for the study of weak associations among the general population where a small effect must be differentiated from no effect at all, or where a small risk must be identified in the presence of strong confounding factors.

Four types of epidemiologic studies are available. They are—in descending order of reliability—randomized clinical trials, cohort studies, case control studies, and ecologic studies. Each type has a particular utility, but all search for a statistical association between an exposure of interest and a particular disease. The reliability of each depends on its level of control over the study data.

Randomized clinical trials, cohort studies, and case control studies all use data collected about individual study subjects. The most reliable is the randomized clinical trial because random assignment of subjects to test and control groups inherently controls for all effects other than those of the drug or toxin under study. The other three are merely observational in nature and cannot control for the confounding effects of exposure to unknown substances. This means that the researcher needs to sort out the effect of the exposure of interest—in this case one or more air pollutants—from the known and unknown, measured and unmeasured, effects of all other factors that impact the health of the subjects.

In the second type, the cohort study, the researcher identifies a group of subjects, obtains relevant information about them, and follows them over time.

The third type, the case-control study, is a kind of cohort study in reverse. It retrospectively studies subjects who already have the disease of interest, and aims to identify a history of exposure to the agent of interest.

Fancy mathematical techniques are misleading because they purport to address data weaknesses that they cannot correct.

The fourth type, the ecologic study design, is the least reliable type and only should be used to develop ideas for further research. Its problems have been recognized for decades.[43,44] Instead of measuring data collected on individuals, it measures only data collected about populations. Both the level of exposure and the health effects are measured at the group level, so the study cannot identify who was exposed to what. It makes the mistake of misapplying group data to individual instances, a problem known as the "ecological fallacy." [45 - 47]

That an association is statistically significant means only that the association found is not likely to be due to random variation in the data. In the meantime, systematic errors that can overwhelm the association are ignored. These can include confounding factors, at least 56 different biases, methodological weaknesses, and model inconsistencies.[48-57] Confounding factors are the single most important problem and include hidden variables such as population shifts, lifestyle changes, unrecognized past history of exposure, unrecognized exposure to co-pollutant(s), and unrecognized health risk factors unrelated to air pollution.

The biases include problems inherent with the study designs themselves, such as the proper choice of controls in a case-control study or inadvertent interviewer bias. By far the most important bias occurs when exposure to a particular risk factor is assessed. The presence of the pollutant does not necessarily mean that exposure has occurred, and exposure does not equal toxicity. For example, the subjects studied may have several pounds of salt in their homes but have little exposure to it. Even if they consume relatively large amounts in their diet, this will not be causative if the disease studied is lung cancer rather than hypertension.

Ever more sophisticated statistical techniques are introduced into these epidemiologic studies, but they cannot compensate for the limitations of the data. These fancy mathematical techniques are misleading because they purport to address data weaknesses that they cannot correct. [58]

The necessarily poor design of epidemiologic studies means that finding a relative risk of less than 3 or 4 (a 200 percent increased risk) probably is not representative of a real causal link but instead is a result of bias in the data and in the methods used to analyze it.[59-61] The National Cancer Institute has recognized this, and the director of drug evaluation at the Food and Drug Administration once stated, "My basic rule is, if the relative risk isn't at least 3 or 4, forget it." [62,63]

Despite the claims of some studies to be cohort—such as the HSC and ACS I studies—all of the environmental studies of PM2.5 have been semi-ecologic because air pollution exposure can, as a practical matter, be measured only at the group level via fixed regional monitoring sites. In other words, health outcomes have been measured at the individual level, but pollution exposure has been estimated only at the group level. As noted above, implying causal association at the group level cannot indicate the cause of disease in individual subjects. [64]

From Association to Causation or Science by Sleight-of-Hand

Cost-effective public policy cannot be promulgated—and disease cannot be prevented—without accurate knowledge of the causes of disease, which scientists determine by using the scientific method to test hypotheses.[65] The scientist first establishes a specific association between the suspected cause and the disease. Then a tightly controlled experiment is performed to determine whether the association in question has identified a *necessary* cause.

Risk factor epidemiology, by equating statistics with science, expands statistics beyond its legitimate use and improperly applies it to the imperatives of health policy and management.

However, the observational studies used by epidemiologists cannot test hypotheses and cannot prove causation.[66] For example, many dozens of studies over more than 30 years have examined the association of coffee drinking with coronary heart disease, and with cancers of the bladder, pancreas, breast, colon, rectum, and ovary.[67] Coffee drinking even has been proposed as a risk factor for hip fractures.[68] Increased dietary fat intake has been associated with breast cancer in women, and electromagnetic field exposure with different types of cancer. But none of these epidemiologic studies has been able to show that the associations are causal. [69]

The introduction of causal assumptions into studies that show only weak associations has been described as "risk factor" or "black box" epidemiology. The "black box" links the exposure and the disease in a causal sequence. [70, 71] Since this bypasses the procedures necessary to scientifically demonstrate a causal link, it is "performing science by sleight-of-hand." [72] It also has been labeled "statistical malpractice." [73]

Statistics is a tool of science. But risk factor epidemiology, by equating statistics with science, expands statistics beyond its legitimate use and improperly applies it to the imperatives of health policy and management. An example of this is the last sentence of the discussion section of the HSC study: "This study, therefore, provides added impetus to the development of strategies to reduce urban air pollution."

The misuse of studies to promote political agendas has been recognized for years and is documented in the literature.[74] Not only is it improper science, but insofar as the proponents claim the authority of science for their preferred policies, it is unethical as well. [75]

The Office of Management and Budget (OMB) reports that the 45 major rules it reviewed during 1994-2004 generated estimated annual benefits of $68.1 billion to $259.6 billion, with estimated costs of $34.8 billion to $39.4 billion.[76] This suggests that federal regulation is a good investment of public and private resources. However, to arrive at those figures, OMB simply aggregated or compiled various agency estimates; it neither reviewed nor endorsed all the "varied methodologies" agencies use to derive cost and benefit estimates.[77] Moreover, of the $68.l billion to $259.6 billion in estimated regulatory benefits, anywhere from 71 percent to 77 percent—$48.8 billion to $215.6 billion—come from EPA benefit estimates of its regulation of a single pollutant: fine particulate matter. As OMB acknowledges, EPA's benefit estimates are subject to a "large" degree of "scientific uncertainty." [78]

Not only was EPA's prediction of the annual benefits from its

PM2.5 standard flawed, its estimate of avoided premature deaths is not known to have any basis in reality.[79] Instead of billions of dollars and thousands of lives per year saved, the standard likely has imposed an annual net cost to the American people of billions of dollars and hundreds of thousands of jobs lost.

The idea that human lives will be saved by reductions in contemporary PM2.5 air pollution has been shown to have no basis in science.

The potential danger from the misuse of statistics was recognized many years before EPA was created. Especially with regard to the agency's PM2.5 standards, the prediction that "there may be greater danger to the public welfare from statistical dishonesty than from almost any other form of dishonesty" has proven prescient. [80]

Conclusion

Congress mandated that EPA base its regulatory policies on good science. But the idea that human lives will be saved by reductions in contemporary PM2.5 air pollution has been shown to have no basis in science. No biologic mechanism has been found to explain how the lungs might be damaged. The epidemiologic studies used to justify the agency's PM2.5 standard have failed to show that air pollution causes disease or death. Instead of supporting causation, their weak associations likely are fortuitous and simply represent normal variation or methodological artifacts and biases.

The present paper calls into question more than just the scientific basis for EPA's PM2.5 standard. If PM2.5 is not killing people, then the EPA is not improving public health or saving lives, but instead is imposing billions of dollars of net costs each year on the American people.

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Notes

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