Written Comments on the Policy Assessment for the Review of the National Ambient Air Quality Standards (NAAQS) for Particulate Matter (External Review Draft—September 2019)

John Dale Dunn MD JD Civilian Lecturer Emergency Medicine Carl R. Darnall Army Medical Center, Fort Hood, Texas, Policy and Science Advisor Heartland Institute, Chicago, American Council on Science and Health NYC

The EPA scandal that cannot be excused.

The issue of small particle air pollution human effects was discussed in a House of Representatives hearing in September 2011 by the U.S. EPA administrator, Lisa Jackson. In a colloquy with Representative Ed Markey (D-Mass.),

Jackson: "Yes sir. If – um – we could reduce particulate matter to levels that are healthy, we could have identical impacts to finding a cure for cancer." (Author note: Cancer kills a half-million Americans a year – 25 percent of all deaths in the U.S. annually).

The claim stated above by Ms. Jackson is the basis for the EPA's war on coal, fossil fuels, and internal combustion engines. All other criteria air pollutants are minimal concerns for the EPA. Surely small particles are a very toxic and lethal thing, as bad as cancer. Right?

EPA is discovered doing human experiments

That same month as Ms. Jackson's testimony, Steve Milloy discovered a report in *Environmental Health Perspectives*, a journal published online and in hard copy by the National Institutes of Health, that reported an experiment on a 57-year-old lady subjected to small particle air pollution much higher than the EPA says is safe, in a chamber at the University of North Carolina

The Nuremberg Code; the Helsinki Accords; the Belmont Report; and U.S. common law, statutes, and regulations, to include state laws and the Federal Code "Common Rule" and EPA rule 1000.17, all prohibit human experimentation that might cause harm to the subjects.

In 2011 and 2012, Milloy and Dunn wrote letters to the EPA, the NIH journal editor who published the article, the EPA inspector general, and the federal Office for Scientific Integrity. They wrote to all the physicians in Congress, all the deans of the ten domestic medical schools doing human experiments, and state medical boards in North Carolina and Michigan, all attempting to stop the human experiments.

EPA sponsorship of these studies at ten domestic and six foreign medical schools was admitted under oath by an EPA official, Wayne Cascio, M.D., and it is unethical and illegal. Senior EPA research scientist Robert Devlin, Ph.D. admitted in a sworn affidavit that the EPA epidemiology was unreliable, the reason for human experiments.

EPA hires the National Academy of Science

The EPA, in response to a congressional inquiry and negative inspector general report, engaged and paid the National Academy of Sciences (NAS) contract subdivision, the National Research Council (NRC), to provide a whitewash investigation.

The closeted investigation continued with closed meetings attended only by NRC staff, committee members, and the EPA and the docket by a year, June of 2016, had 50 documents, all submitted by the EPA or its allies. In May of 2016, a congressional aide ran across information about the existence of the committee and informed Milloy. Milloy demanded a hearing and allowance for submissions in June of 2016, which was granted by NRC officials. Milloy found that 13 out of 19 members of the committee were significant grantees of EPA, amounting to tens of millions of dollars received, with the most extreme example being Charles Driscoll, discussed here.

On August 11, 2016, an internet audio conference of the National Research Council Panel on EPA-sponsored human exposure experiments titled "Assessing Toxicological Risks to Human Subjects Used in Controlled Exposure Studies of Environmental Pollutants" was held, with two hours of testimony heard and submissions critical of the EPA human exposure experimentation. After that, nothing was heard from the committee.

The NRC committee published its news release and a 150-plus-page report on March 28, 2017, that exonerated the EPA human experiments on the theory that small particles are not toxic or lethal or carcinogenic acutely – that is, they do not have any acute toxic effects, but rather just long-term deleterious effects. The NRC committee said that, knowing the EPA asserted short-term acute death effects. So we have an example of deep state collusion on misconduct and cover-ups. An NRC committee creating cover for the EPA by misstating the EPA position on toxicity and lethality of small particles. That is clear from this quote from the National Academy of Sciences Report press release:

The EPA violations of epidemiological and toxicology scientific rules are a scandal that cannot be ignored. The Dockery 1993, Pope 1995, and Samet 2000 studies (see endnotes) and other studies of health effects of air pollution relied on by the EPA, all showed that large studies with adequate power could not demonstrate relative risk of any significance. The studies all showed effects less than ten percent, rather than the statistically and scientifically required 200 to 300 percent effect. It is astounding the EPA has the gall to announce an air pollution crisis and propose more stringent air quality standards when none of the studies the EPA relies on show and proof of health effects.

The EPA is obligated to educate the public on the clear evidence that air pollution may have aesthetic and cultural import, but that there is no air pollution health “crisis.” The EPA and its sponsored and supported health effects reasearchers are now just raising their voice in this debate instead of trying to use science. The EPA air pollution health effects science is an emperor with no clothes, as discussed below.

This commentary challenges the EPA to show one study that proves that one person has died due to air pollution in America in this past 20 years. People die for various reasons, suddenly and not so suddenly, as will be discussed below. That reality eludes the work of numbers crunchers who slave at desks over death certificate information like Pope and Dockery. One doesn’t die from an exposure to air pollution, one dies from failed medical therapy, arrhythmias caused by long term coronary disease, stroke, pulmonary embolism, which are not caused by air pollution. The Asthma problem is an increasing problem not related to air pollution, since the rate of asthma is increasing with decreasing air pollution. The deaths from asthma will be discussed below and have nothing to do with air pollution, it is a socioeconomic phenomenon. It is time to retire the air pollution health effects studies of crude death tallies and it’s time for the EPA to stand down from this repeated use of crisis talk and aggressive pursuit of pure air—a religious campaign disguised as science in the public interest.

As a last and compelling consideration, this author is familiar with death in America. As an emergency physician, much more familiar with what kills people than economists and public health officials who don’t know which is the business end of a ventilator and live in the world of death certificates and mortality data.

People die for many reasons and under many circumstances in America, but air pollution doesn't kill them, even the worst levels of outdoor air pollution one might imagine in America don't create a toxic level, which reveals the other major flaw in the EPA crisis rhetoric, junk science toxicology that completely disregards any effort to define toxin or toxicity. That subject will also be dealt with herein below.

The scientific epidemiological and toxicological criticisms of the EPA health effects studies and policy making are:

1. The Dockery 1993 and Pope 1995 studies did not show valid evidence of death effects, since they showed a death effects relative risk below 1.1, a negligible relative risk that is 10 percent of the minimal relative risk all epidemiologists consider necessary for proof of causation. A 200% or 300% change in death effect is the lower limit. Some epidemiologists require relative risk of 4 or a 400% effect when evaluating poorly controlled cohort studies.
2. This relative risk problem cannot be overcome by EPA and health effects researchers emphasizing the misleading use of the term statistical significance, which is not a proof test, but a statistical reliability test. One can be statistically confident and reliable but absolutely wrong.
3. The EPA and its health effects researchers have consistently and persistently ignored the lack of proof of health effects in these studies, and have made public announcements and allowed media reports to proclaim that thousands are dying in America due to air pollution when the studies do not show any proof of death effect at all. Lying for justice or an environmental ideal does not make the lie any less dishonest.
4. The health effects research used by the EPA has consistently ignored the basic rules for toxicology and the well-known phenomenon of threshold for toxicity. Only at the EPA does straight line toxicology have any status, mostly because it avoids serious science. Main stream toxicology science is still committed to the idea of threshold of effect and the old saying—the dose makes the toxin. The EPA scientists in house know the truth, but again politics and a commitment to a policy/environmental ideal results in lies.
5. Under no valid scientific analysis retro or prospectively, can the EPA use the methodologies or the results of the Pope, Dockery, McDonnell, or Lipfert (see endnotes) studies to justify one more burdensome air pollution regulation, but there is strong evidence for rescinding the last round of Air Quality Standards.
6. The EPA has a mandate to act only on the basis of acceptable scientific evidence of health effects, and is obligated to abandon the precautionary principle approach to regulatory policy, a pathetic substitute for legitimate science and clearly a principle founded in politics, not science.
7. The EPA could never convince a Federal Court, operating under Federal Rules of Evidence 702 and the court dicta for expert and scientific testimony that the EPA air pollution health effects science is valid proof of anything. The Pope, Dockery and Lipfert or Samet studies cannot be massaged or misrepresented enough to create any proof of air pollution health effects. The studies show trends within an insignificant range and "associations," that are not evidence of proof of health effects.
8. Precautionary principles that are used by the EPA as stand-alone policy justification, are nothing more than a dressed up version of anxiety, cannot pass muster for admissible scientific evidence in a Federal Court and ignore the reality of risk/benefit analysis.
9. Based on the information reviewed in this critique, the EPA must revisit old regs, forgo new, more onerous and expensive regulatory interventions, and the EPA must suspend its rule making in air pollution until it can find valid and reliable science on health effects.

Toxic air pollution existed in the past, and still may occasionally occur in some places on the planet as a local phenomenon, as particulate and other noxious air pollution in industrial areas, from various sources. Certainly air in big cities, Pittsburg, Los Angeles, Houston, New York was fouled in the past by air pollutants and even when not toxic, was smelly and visible, but trends in air pollution in the past 30 years as

reported and confirmed by the EPA, have all been positive, attributable to changes in industrial processes, regulatory efforts and cleaner petroleum and coal consumption. Any study or discussion of air pollution is focused on a moving, improving problem. However the public thinks the air is worse than ever and there is an air pollution health effects crisis, and that is the fault of the EPA, its favorite researchers, and the mass media, who love to scare the public, since EPA budgets and environmental organization budgets depend on the anxiety of the public.

The death and illness rates during smog and air pollution catastrophe periods in the past were affected by less effective medical management and heavier cigarette smoking but also significantly higher air pollution than exists anywhere in the United States today, for many reasons. Deaths from acute respiratory failure in the past were more common and less preventable, but that is an independent factor related to medical advances and not due to air pollution itself. Airway diseases, the main effect of any air pollution, were less treatable before the 1970s. Pulmonary Medicine has changed dramatically for the better since 1970. Many airway diseases were more dangerous in the past and medical therapies frequently failed to control disease and death. Medical expertise in respiratory illness and cardiovascular disease is changed, but Pope and Dockery still yearn for the good old days of killer air because it scares the public. Their research ignores the trends of the last 20 years and below I will discuss a conscious deception in the second half of the Pope research from NCI data. In addition the EPA air pollution researchers continue ignores the weakness of their findings, hoping to keep alive the "deadly air" panic talk alive.

People die for lots of reasons in America, but not due to air pollution. Air pollution health effects researchers know that, but act as though nothing has changed. The EPA should carefully reevaluate the number of deaths that researchers claim are due to air pollution in the last 20 years, but the EPA has a conflict of interest. No air pollution crisis might mean reduced EPA funding. No air pollution crisis might mean no funding for the researchers and their support organizations.

The air pollution health effects studies are based on weak epidemiologic relationships and trends carelessly described without definition as "associations," or "trends." Well ice cream consumption and drowning or boating accidents are associated by season, but ice cream eating doesn't cause water accidents. Associations are not proof, they are observations of phenomena--clusters of events that may or may not mean something. Epidemiologists know these things and should be careful when describing data associations and trends within insignificant ranges like less than relative risk of 2, so that the reader or reporter won't mislead the public or a politician. However, the definitions are not forthcoming from the scientists and researchers because saying that there is no crisis of air pollution means no publications for air pollution researchers, no invitations to swell events, no funding, no chance to pursue a political agenda and change the world, making your mother proud.

The uncertainties of the air pollution health effects studies, the weak relative risks and the methodological problems of the most influential of the health effects studies are so noticeable and remarkable that during this comment period the EPA should reassess what has gone wrong in air pollution health effects research. The EPA should assess how these weak studies have affected EPA policy and rule making. The EPA doesn't have the right to panic the public and political leaders with deceptive junk science in the service of religious and fanatic environmentalism.

DISCUSSION OF THE STATISTICAL AND METHODOLOGIC PROBLEMS OF THE SAMET, POPE, AND DOCKERY HEALTH EFFECT DEATH STUDIES.

Author's comments are in bold. Studies referenced are underlined and the cite is in the endnotes by name and year. Sorry to disappoint those who want numbered endnotes—not a formal paper.

J. Samet (Samet 2000) published in the *New England Journal of Medicine,* a study modeled after the studies of Pope (1995) and Dockery (1993). He compiled and studied deaths in twenty American cities over a period of years, and compared them with air pollution monitor reports for those cities.

Samet in this 2000 paper asserts the following:

--"the relative rate of death from all causes was 0.51 percent increase for each increase in the PM 10 (10 micron size particulates) of 10 micrograms per cubic meter." **This effect is not proof of anything, and Dr. Samet knows it. Less than a 1-% death effect is a nonsense result in a big cohort study.**

--"the relative rate of death from cardiovascular and respiratory diseases rises 0.68 percent for each increase of 10 micrograms per cubic meter" Trends **of less than 1% inside of a meaningless range of relative risk less than 1.05? A serious epidemiologist would snicker?**

--"we also analyzed the effects of levels of carbon monoxide, sulfur dioxide, and nitrogen dioxide in a fashion similar to that of the analysis of pm 10 levels. After adjustment for pm 10 and ozone levels we found little evidence that these pollutants had a significant effect on the relative rate of death." **Hold it, hold it, Samet says that he can't find an effect, even itsy bitsy effects from ozone precursor and carbon monoxide, something the other EPA favorite researchers say are killing thousands? Samet is not helping the EPA here. What about those dastardly pollutants? We scientists and particularly toxicologists are smiling to see Samet make a fool of himself and by adoption of this weak and deceptive epidemiology, the EPA doesn't look too good either. This is the kind of research the EPA has been using in air pollution regulatory policy now for years.**

--"We did not find an effect of ozone levels on the overall rate of death from all causes or from cardiovascular and respiratory causes during the full year periods. Ozone levels were positively associated with mortality rates during the summer months when ozone levels were highest, although the 95 percent posterior interval extended into the range indicating no effect of ozone levels on mortality." **Might this non-Johns Hopkins man who owns no jacket with arm patches translate for the benighted—Samet says even ozone doesn't have a death effect in his study. Score so far on this paper—rational skeptics for people in search of truth 3, EPA and Samet 0.**

--"We found no evidence that key socioeconomic factors such as low socioeconomic status affect the association between PM10 and the risk of death in linear regression models." **Some might be surprised to know that Samet works at a School of Public Health and all Public Health research for the last 20 years has shown clearly that there is a socioeconomic effect that produces premature deaths. Skeptics now 4 and running away, EPA and Samet still 0. Socioeconomic noise cancels out air pollution effects; that's the way the epidemiologists put it.**

--"Our analysis also did not address the extent to which life is shortened in association with daily exposure to the various pollutants." **Well golly Dr. Samet, everyone dies, how can you talk about death effects if you don't measure whether deaths are premature? Skeptics 5, Samet and EPA still 0.**

Additional comments by this author:

1. The rate of death changes in Samet's studies are less than 1%, which is epidemiologically meaningless and shows no respect for the relative risk of 2 (100%) or more, that all cohort studies have to show in order to be able to assert effect. Little effects, even in studies with good confidence intervals and lots of power, are still empty studies, make work exercises. Samet's study was a nothing, yet it got published in the *New England Journal,* so one must wonder about

political and environmentalist agendas up in Boston. I suppose they are neutral on the environment and always demand valid research in support of political agendas. I suppose.

2. The study fails to age/sex adjust for the important analysis—premature death. How did Samet get published? Samet is asserting proof of effect at less than one two hundredth of what is required in epidemiology. Then he says he didn't bother with measuring whether air pollution caused premature deaths. This research is about acute death affects? At non-toxic pollution levels? There is no plausible biologic science to support the idea that non-toxic air pollution kills people. Samet is beyond redemption. He's in scientific denial, or he works for the EPA agenda and he will be funded until he is old and gray.
3. Low relative risks, below 1.2, are the results in Samet's studies and all the other EPA health effects studies. One study goes above 1.2, the Dockery 1993 smaller study at 1.26, since recalculated by Enstrom in his article, Enstrom 2005 to 1.13. Such weak and minimal findings are unacceptable for publication, much less serious EPA policy making. The EPA and the studies misuse the term statistical significance, trends or association if they mean proof. There is no proof in any of these studies of an air pollution health effect. These studies prove nothing in the relative risk ranges of less than 1.3, particularly in cohort studies of death certificates that are subject to serious confounding.
4. The failure by Samet to find any effect, even these minimal effects, from other air pollutants like nitrous and sulfur oxides (ozone precursors), ozone, and carbon monoxide should give the EPA cause to wonder about any further attempts to impose new ambient air standards. The EPA has noticeably ignored Samet conclusions about these pollutants, why?
5. Samet's assertion that socio economics do not effect death rates is a an extraordinarily faulty conclusion for a public health researcher, since his study only looked at average area incomes for the twenty cities; and there is a vast body of public health research that shows that socioeconomics independently are a significant factor in life expectancy. (Wong 2002, Fitzpatrick 2001, Lantz 1998).
6. Socioeconomics is a factor and would nullify the signal from air pollution effect, and could even be a cofactor in another way by causing poor indoor air quality from substandard housing and a higher rate of smoking along with a higher rate of underreported smoking. For example the poor have outdoor jobs where they can smoke more, and culturally they may be much heavier smokers with more inhaling, a potential confounder. Such confounding might explain the Ohio and West Virginia data from Pope 1995. That's why relative risk has to be set high, to avoid the effect of confounders not seen or understood.

The Samet article includes cautionary notes on the limitations of the study's methodology. His caveats are applicable to the all the previously mentioned Pope and Dockery, favorite EPA studies on air pollution health effects:

1. "For the pollutants measured on an hourly basis we calculated the 24-hour average." **Toxicologists cringe at that one.**

2. "If the pollutants were measured at multiple locations in a metropolitan area, we averaged the data." **Remember the basic principles of toxicology; if you're downwind from an air pollutant you're safe, how can he say these things with a straight face? You have to know the patient and the toxin and the dose to know anything much about the science. Population studies are very crude at non-toxic levels of exposure.**

3. "Since the Environmental Protection Agency requires levels of PM 10 to be measured only every six days, data for ozone and other pollutants were generally more available on more days." **Good grief, this is a sham, a toxicology study with exposures every so often in sub toxic ranges.**

4. "We analyzed the effect of the day on which the pollution data were obtained (the current day, the day before, or two days before) on the association with mortality rates. The overall effect did not vary with the lag interval selected. We report data for a one day lag between pollution variables and mortality." **This is the place where Dr. Samet shows he doesn't know anything about death. You could be sick to death in a hospital and I can keep you alive indefinitely until the family gives up—where do those cases fit in Dr. Samet's arbitrary lag time of one day? What about people who die in a bed at a nursing home and haven't been outside in two years? These public health wonks and economists who hate dirty air do research as if a death certificate signed by the local GP is a piece of reliable data on the health effects of air pollution. They are in dreamland.**

Then Samet says they found a temporal-causal relationship -- astounding! He didn't find a causal relationship, but he can find a temporal relationship. Did he dredge and dredge until he found something to point at? What's he talking about? Who's to know when the blips in the data are differences of less than 1%? That's not about cause of death, that's about political agendas and a polemic dressed up as science that causes public anxiety.

The good Doctor continues.

5. "Data on levels of PM 2.5 (small particulates) are not yet available nationally, since a monitoring network for particles in this size range is currently being implemented." **This writer believes that Dr. Samet is working the agenda for the "annuity." Small particulates are an annuity for the EPA and air pollution researchers because, along with ozone, dust will never go away. Those air pollution demons assure EPA power into the distant future and more regs and anxiety. Dust is bad. Dust is always going to be there. It's the perfect air pollutant for the EPA.**

Samet and others in the air pollution junk science club just use the PM 10-micron data that is measured every six days as a surrogate for PM 2.5. The supportive press and academic colleagues forgive such a lapse since they are working on the agreed upon agenda.

6. "Our analyses also did not address the extent to which life is shortened in association with daily exposure to the various pollutants."

Extraordinary. If the endpoint is a death effect, then the study must analyze premature death in mortal man and assess acute events as a measure of effect and endpoint for acute and/or chronic disease. To determine premature death effect, age and sex adjusted death rates are the accepted methodology, but Samet is just doing death rates and he gets published in the *New England Journal of Medicine*? Politics and the right agenda trump science and peer review?

7. "The finding that the association between PM 10 levels and the risk of death was strongest for cardiovascular and respiratory causes of death is consistent with the hypothesis that persons made frail by advanced heart and lung disease are more susceptible to the adverse effects of air pollution."

Again they didn't show that at all, they showed less than a 1% effect on death rates. I thought these people were dying of air pollution caused illness, not acute effects of air pollution, which at current levels couldn't kill a canary. What gives? What gives is that Dr. Samet is clueless because he's a numbers cruncher for the EPA in cahoots with his friends in the spic and span air society. I know why people die and it isn't from air in America, or even from Air America.

Air pollution comes in many forms but we are obligated to live with toxicology science, not anxiety. Living organisms don't die for the thought of a smoggy day or from a bad smell. Dr. Samet and his cottage clack of air pollution hand wringers should go to a hospital and see how and why people die before they do these desk analyses of death certificates.

Despite these caveats the Samet research group asserts in the conclusion of their paper: "Our analyses provide evidence that particulate air pollution continues to have an adverse effect on the public's health and strengthen the rationale for limiting levels of respirable particles in outdoor air." **Samet says nothing about the significance of their research showing no death effect from ozone, carbon monoxide, sulfur and nitrous oxides. That would certainly disrupt current EPA policy, and he avoids an admission that the relative risks and death rate changes he found do not reach epidemiologic significance.**

This study by Samet is sham epidemiology/science, junk science with lipstick, and the deception and "newspeak" harkens back to junk science in the service of the King or the current tyrant. Pope, Dockery and Samet are the officials/magicians/astrologers/conjurers in the EPA court, providing the EPA regent with needed "expertise" to justify the latest edict.

Briefly we will discuss below Dr. Samet's mentors, the EPA's favorite air pollution haters, Drs. Dockery and Pope, who work together and change places on the author's lists of their papers.

The Six City and Pope Studies?

Dockery (1993) and Pope (1995) did studies that were the model for the Samet study discussed above. The studies did do better than Samet, in that they measured relative risk of premature death by studying death rate with age sex adjusting. Both Dockery and Pope were unable to show significant relative risk of health effect. The Pope and Dockery studies were used in the mid 1990s to justify EPA Director Browner's "emergency" new ambient air quality standards on ozone and other pollutants. The resulting cost was estimated by the Center for Study of American Business at Washington University, St. Louis, at more than 100 billion. The Browner action was taken unilaterally, in spite of protests from many agencies within the government and without the approval or support of EPA internal experts. This action was taken without proof of a health effect, since Pope and Dockery never showed an acceptable relative risk. They were limited again to Samet's "associations" and trends within meaningless ranges below a relative risk of 1.3.

There is a greater relative risk of whole milk causing lung cancer than the relative risk that the EPA has shown for air pollution. One might say that's because of some confounder—well duuuh, that's why relative risk has to be above a threshold of 2 and some say 3, so confounders don't make the epidemiologist look confounded. Samet, Pope, Dockery don't care, they're on a roll and have the support of the environmentalist zealots, and the EPA (whoops, that's redundant). Call public relations, the research shows air pollution is killing thousands. It causes CANCER.

This paper points out that the EPA and the researchers are cheatin', and Dr. K. Popper, famous philosopher of science favorably cited by the Supreme Court in the Daubert decision, says that science must be more serious and reliable than politics. Popper asserts that science must be based on proofs that are reliable. Popper even talks about what the air pollution research by Pope, Dockery and Samet and the spic and span society is—Popper says some "science" is so bad it can't be falsified. How does one falsify something that means nothing? Associations at the edge of or in the midst of nothingness is what Pope's and the other health effects studies assert should be the basis for society wide regulatory regimes. Breathtaking—no pun intended.

The EPA says that air pollution kills thousands, because air pollution kills thousands. That is a tautalogy, a common tool for junk scientists. IT IS BECAUSE IT IS. I write here to tell the EPA that their anxious pursuit of clean air is more about politics and power and anger with modern industrial society that is already cleaning up the air, more about the religion of environmentalism. That's why the crisis, without the deaths or the science is a political or a polemic tool, not science. Not nice to fool with science that way, particularly when there is a Federal mandate that the EPA insist on scientific integrity for policy making. The EPA should not be in the business of ginning up false crises and scaring mothers that their kids are going to suffer from the air just so that the bureaucracy will thrive at the Federal and State level.

The EPA cannot claim to be unaware of the failure to prove health effects by the insignificant level of relative risk in the Pope and Dockery studies. These are the most basic of epidemiologic rules. And no subsequent studies have rehabilitated the failures of the Pope and Dockery studies. Samet, as described above, just repeated the same mistakes and came to up with the same lack of proof of health effect, unjustified conclusions and excessive and activist recommendations.

The barriers to a good study on health effects of air pollution for Dockery and Pope were the same as for Samet,

1. mobile populations,
2. unreliable, non-continuous and fixed monitor information,
3. no monitor information on some pollutants all the time (2.5 micron particles for example) or part of the time (10 micron and others),
4. an attempt to assess long term chronic health effects of air pollution by death studies, an acute phenomenon,
5. death certificates and raw death data used without autopsies,
6. inside air quality ignored for populations living indoors, particularly during old age, advanced medical illness, and terminal illness,
7. But most of all, no biological plausibility because the deaths are in the setting of non-toxic levels of air pollution (the inane straight line effect toxicology of the EPA cannot continue to get a pass—it is advocacy at the expense of science).

The EPA in assessing the air pollution effects studies must revive Bradford-Hill Criteria for toxicology

The Bradford Hill (BH) criteria for toxicology are elementary, and establish biological plausibility for toxin effects. They require the toxicologist to establish plausibility, dose effect, reproducibility, time relationship, and a pattern of predictable and observable effects. Sounds like good science, but that's only part of it. Karl Popper was referenced above as the guru of the philosophy of science, and master or curator of scientific principles. The Popper legacy of science rules are referred to reverently in the Supreme Court opinion in the Daubert v. Merrill Dow Case [509 U.S. 579 (1993)] on admissibility of scientific testimony. Falsifiability is the key. To be true science one must submit to the test of being proven wrong. Pope and Dockery study results can't be falsified because they don't even allow a legitimate assertion of proof. They are tools in the game of politics, not in the game of toxicology. The EPA is required by common sense and federal statute to apply the BH criteria in air pollution studies, and all other toxicology work, but instead this wildly deceptive use of small changes within insignificant ranges of effect is souped-up to become the reason the EPA must act, now, immediately, to save lives. The EPA is saving itself, but the air pollution regulations are not saving any lives because the research would show the lives lost with valid epidemiology, and it doesn't.

The only reason that the EPA can create a crisis from the Pope or Dockery studies if it holds its nose and just projects to the whole population of the United States, then relative risk of less than 5% becomes thousands of deaths, even though it fails to show proof of one death caused by the toxicity of air pollution. Not one death.

If the biological plausibility of air pollution causing disease and death consistent with the BH criteria was established or could be established, then EPA and air pollution health effects researchers like Pope, Dockery and Samet could rest with their laurels. If air really were a killer or a toxin, we wouldn't see these weak cohort studies from the EPA with itsy-bitsy relative risks, and the argument would be over.

The EPA is not the national agency or institute for the arts, culture, pleasantness and good smells, it has a serious public health responsibility and a federal mandate to find toxins with legitimate science, promulgate appropriate solutions for the public benefit and then assess the effectiveness of what it has done. None of those steps are being taken in the air pollution policy making of the EPA.

The air pollution health effects studies in America will never be able to show the required relative risk of 2 or 3. What was the EPA role in such deception?

The idea that seems to control the EPA policy making on air pollution in the past 15 years is--ignore methodology and statistical problems, science be damned, move on to the grand program of air purification. Find the ultimate terrible pollutant that will never go away, even with all our regulations. That is why small particulates are so promising for the EPA, enough so that these health effects studies talk about small particulates without measuring them, or measuring them in only one part of the study and not everywhere. The project of demonizing small particulates is reflected in the Samet study. He makes strong assertions with extraordinarily weak evidence, but he goes to the meetings, he knows what the EPA is concerned about. With EPA leading and frequently funding the crusade—science and truth casualties are acceptable. Small particulates are the worst crisis in the history of air pollution, they might cause CANCER.

I grew up and still live on a farm. I consider dust a reality that cannot be regulated away, just like ozone is part of the Smoky Mountains. There is a form of air pollution that is now being generated by the EPA in its ozone and small particulates crisis project—it is composed of dust, water, methane, and biological particulates.

Joseph Schumpeter said that the first casualty of a commitment to an ideal is the truth. The second casualty, this author asserts, is the unwary taxpayer and public that depends on responsible government. Solzhenitsyn said "The simple step of a courageous individual is not to take part in the lie. One word of truth outweighs the world." The EPA has become a slave to the lie of junk science in health effects research because the agency is devoted to its own importance and the importance of its religious and political agendas. EPA dredges up and makes icons of the precautionary principle, the small numbers/large projections lie, small trends within meaningless relative risks in population studies, the refusal to recognize basic toxicology concepts. The EPA is a rogue agency in need of a stand down and close internal inspection.

The Killer Smog

In *The New England Journal of Medicine, Dr.* C. Arden Pope, clean air activist, and one of the EPA's all time favorite air pollution health effects researchers, describes killer air in Belgium in 1930, Pennsylvania in 1948, and London in 1952 -- and uses those incidents as examples of why he thinks there is good reason to pay attention to a study in that issue of the *Journal* that claims to show a causal relationship between non-toxic air pollution and children's pulmonary functions. Again the study he is supportive of shows no epidemiological proof, just "associations," which are nothing more than statistical cluster puffs in

population studies subject, as pointed out above, to bias and confounders. But the key is the study includes two important things for environmentalist zealots, children, and air pollution. Most importantly this study, like all the air pollution health effect studies, is working in insignificant causation ranges of effects so Pope and the EPA can talk about little bitsy trends and associations and urge that something be done before children die on playgrounds. They talk of these numbers exercises like they foretell an apocalypse. *Gather the elderly and children and go seek shelter from the air, says Dr. Pope, an economist who got in the air pollution health effects business because he hated the air in Utah—imagine if he had lived in New Jersey. Dr. Pope advises---Stop breathing, if you must.*

People do not go out into the streets of America, choke and die. The days of the people of London and Pittsburgh wearing dark clothes to mask the effect of soot and smoke are gone. The public health hanky battalion wants Americans to think air is killing their children and old folks, but in America ambient air pollution did not kill anyone, last week, last year, or in the last ten years. The panicky talk has to stop and the EPA must stop being the sponsor of the lie. The medical journals have to put their scientist hats back on and stop wringing their hands about nonsense environmental crises. The EPA is so busy these days frightening people about their rat studies and the imagined effects of so many things. Hardly enough time in the day to pursue air pollution, except the EPA has lots of staff and lots of money and much energy and religious devotion to the cause.

EPA Policy and Regulation Activity

Fredrick Bastiat is known for his "law of unintended consequences," best exemplified as the analysis of the Paris shopkeeper's broken window. Bastiat made a common sense observation that when government or individuals choose to spend money or act, it produces desired and undesired effects, always making a ripple within the society and economy.

Let us propose to the EPA that if asthma deaths are predominately in young adult black males in America because of poor compliance (McFadden 1997), due to cost and availability of asthma treatment for disadvantaged adult black males or some other socio-economic or political problem, the EPA would be foolish to work on parsing senseless air quality regulations in preference to better asthma health care. The EPA would not be a party to such nonsense, would it, to relieve the anxiety of anxious environmentalists or satisfy the EPA staff's need for power and control?

There are no free regulatory actions. Every choice has multiple consequences, and government interventions have effects unforeseen. The EPA takes taxpayer dollars for every jot and tittle, every phone call, every new grand idea of every zealous bureaucrat. Every dollar spent for the EPA's ideal of pure air comes from somewhere and is taken from somewhere else.

The EPA is charged with responsible health effects research and policy making. The questions raised in the mid 1990s and now are the same:

1. If relative risk is a well-known measure of cause and effect in epidemiology, why does the EPA allow relative risk below acceptable levels of proof to influence policy making?
2. Considering that EPA regulatory activity is tremendous burden to the economy, and the air regulations have a cost effect measured in billions per year taken from the taxpayer. If socioeconomic factors are an undeniable influence on quality of life and life expectancy, then can weak and unacceptable health effects epidemiology as described above, be excused for some abstract ideal of pure air?
3. Can studies that measure acute events in any way be considered studies of cumulative health effects? Are these death studies that Pope and the other air pollution researchers insist on basically flawed and deceptive? The answer is yes.

4. If some of the studies can't eliminate confounders, does the EPA have the authority to impose an onerous regulatory regime on the American society on the theory that cleaner air is a worthwhile, even if it doesn't have any effect on health?

Enstrom Particulate Air pollution Health Effects Study of 50,000 elderly Californians.

Dr. James Enstrom, in the attached article found in appendix A, studied deaths in elderly Californians in 25 counties. He found that the relationship between fine particulates and mortality was very weak during the 1973-2002, particularly after 1982. He also reviewed the cohort studies on health effects of fine particulates and mortality by Pope, Dockery, McDonnell, and Lipfert, and found that their results were fairly similar to his, with the weakest health effects being present during the most recent years.

Enstrom finds:

1. The relative risks, age and sex adjusted and homogenized, are close to 1.00 in his and the other death studies (Pope, Dockery, McDonnell, Lipfert) he reviews in Table 10—there is no proof of health effect shown from particulate air pollution in his or the other studies.
2. Pope's year 2000 16 year follow up to the earlier (Pope 1995) study of the same cohort (Pope 2002) shows a declining cumulative risk from 1.07 to 1.04, first half to second That means to all but the innumerate that the relative risk in the second decade is well below 1.04. **Hello Dr. Pope, Helloooo EPA.**
3. Enstrom points out there are substantial geographic variation between the California populations of his study and Pope's Ohio, Kentucky and West Virginia data. The potential for confounders should be considered. **I know something about that, and people in those states aren't the same as people in Enstrom's study. They might live different lives from their fellow citizens in Lala land. That's what homogenizing and sampling in epidemiology is all about. Without the data from those three states, Pope's studies would be more epidemiologically insignificant than they are, if that were possible. So much for avoiding cherry picking and the admonition to chip off the edges of the data to norm a cohort analysis.**

The important points of the Enstrom study:

.

1. Deaths and air pollution relative risks were assessed for 25 California counties, a cohort of 50,000 elderly Californians, and 39,000 dead before the end of the study in 2002. The relative risks were measured with proper confidence and homogeneity.
2. Relative risk found was extremely small and insignificant, 1.04 in the first part of the study (1973-1982), and then relative risk of death from air pollution disappeared altogether in the second part of the study (1983-2002). **Which will it be EPA, a crisis or salvation from killer air?**
3. For the entire period the relative risk was 1.01 **Pulleeez, 1-% risk? That's a relative risk of 1.01. I am closer than that to being rich and good looking, like Michael Jordan. The results would have to be 2.00 to be proof of any health effect, 1.00 is no effect**.)
4. **This Enstrom study, like all the other studies that the EPA uses to analyze health effects, and supposedly to study small particulate effects, is limited by the lack of PM 2.5 micron monitors before 1979 and only limited monitors after.**
5. No increased death effects of any kind were shown in the counties with higher levels of air pollution, eliminating any dose response effect (a favorite rhetorical tool of the EPA researcher group), that, some of the higher pollution counties had lower relative risks. **So is air pollution good for you if you live in California? In this range of relative risk absence of trend is meaningless but Dr. Enstrom does the prescribed exercise, since the air pollution cabal likes to do trending and associations. The idea of a trend within an insignificance is interesting to consider, for fun, but not for science.)**
6. Table 10 in Enstrom's paper shows a comprehensive review of comparable relative risks from large (Pope, Enstrom) and small (Dockery, McDonnell, Lipfert) studies, showing that only the Dockery study published in 1993 in a small cohort shows a relative risk above 1.1 at 1.15. All the other studies show relative risk similar to Enstrom, in the range of 1.07 or less.

7. In table 10 a number of the confidence intervals cross 1.0, the cumulative relative risk of the Pope study for the second half is lost in the failure to separate out the second half, indicating there is a reason to believe that in the second half of his study 1990-98, Pope had a relative risk approaching an insignificant 1.01. **I worry, sort of, about Pope hiding this bad trend downward of an already weak relative risk. Could one suppose he has revealed this problem to his friends at the EPA?**

Suresh Moolgavkar

It would not be practical here to cover all the writings of Suresh Moolgavkar on the epidemiologic and methodology problems he identifies in the EPA air pollution health effects research and policy making, and this writer does not understand some of the subtleties. Dr. M's brain and pen are too capable for an adequate treatment here, by a mere emergency physician. Dr. Moolgavkar's recent in depth review and critique of EPA particulate and air pollution research and policy making is in Appendix B.

Moolgavkar 2005 wrote a commentary on Enstrom's paper for *Inhalation Toxicology* discussed above (see second part of App. A). He asked the rhetorical question "Can contemporary epidemiological and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders?" (**Dr. Moolgavkar is too kind. He politely avoids exposing the junk science, the obvious, that miniscule risks in a cohort study like the results in the Pope, Dockery and other studies show no health effects at all and talking about trends in those ranges is silly.)**

Moolgavkar objects to the methodology of proportional hazards modeling because "it is highly unlikely that proportionality of hazards would hold over the entire period of time covered by these studies." (The long term air pollution health affects studies). He asserts that it can be argued that "the SO 2 effect wipes out the PM signal in joint pollutant models." He does not even address the Samet study showing no SO 2 effect, so even that problem may ignore the more basic one that is so apparent—there is no detectable causal effect between air pollution and death. Dr. M is operating with the assumption that SO2 still is on the top of the list of bad pollutants. No doubt it is more toxic than others, but again, we must repeat the toxicology commandment—**the dose makes the toxin.** The air pollution health effect studies relied on by the EPA are ridiculously weak and are used as silly substitutes for a lack of laboratory proof that the current air conditions cause disease. The health effects research of Pope Dockery and Samet is just an exercise in the traditional deception of the "data dredge," the tool of crisismongers.

What is the point of quibbling about miniscule, below threshold of proof, differences in a cohort death study, some slavish devotion to arithmetic? I benefit, I suppose from not liking higher math, in this circumstances, that's why I focus on the medicine and the proper analysis of death studies and why people die.

Moolgavkar (2005 See App. B) wrote a lengthy review and criticism of EPA policy in *Regulatory Toxicology and Pharmacology* that exposes the epidemiologic and toxicological problems of the EPA air pollution health effects research discussed above.

Moolgavkar asserts: "evidence fell far short of supporting a causal association between particle mass concentration and human health." He goes on "the results of observational epidemiology studies can be seriously biased, particularly when estimated risks are small, as is the case with studies of air pollution. The Agency (EPA) has largely ignored these issues." "I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health."

Although Moolgavkar allows that the EPA may be bending the science in an attempt to pursue the precautionary principle on particulates, the precautionary principle under a mandate of good science in the public interest is not good policy. It is the default position for making concerns, feelings and aesthetics into the basis for regulatory actions that cost society billions for compliance. However no sandal-footed environmentalist gang of enviro-religious concerned citizens can allow the EPA to reject science.

The EPA is prohibited by federal mandate from ignoring science in the pursuit of the precautionary principle. The precautionary principle is anti-science and irrational by definition. Health effects not shown scientifically trumps

feeling, concern and governmental overreach. The EPA is mandated by federal law to halt the overreach of the air pollution crisis crusade until it can resuscitate science in the public interest.

Author--John Dale Dunn MD JD

Civilian staff lecturer Emergency Medicine Residency, Darnall Army Community Hospital, Fort Hood, Texas,
Public Health Authority Brownwood/Brown County, Texas 1991-2005,
Instructor, Environmental Law, Howard Payne University, Brownwood, Texas 1989-93,
Research Fellow in Environmental Regulation, Health Care Policy, Texas Public Policy Foundation, 1998 -2003,
Policy and Science advisor Heartland Institute, Chicago, American Council on Science and Health, NYC.
Member of Louisiana and Texas Bars (inactive).

Endnotes

Samet 2000 Samet JM, Dominici F, Curriero FC, et.al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. *NEJM 2000; 343:1742-9.*
Wong 2002 Wong JD, Shapiro MF, Boscardin WJ, et. al. Contribution of major diseases to disparities in mortality. *N Engl J Med* 2002;347:1585-92.
Fitzpatrick 2001 Fitzpatrick R. Ed. Social status and mortality. *Ann Intern Med* 2001 134;10:1001-2.
Lantz 1998 Lantz PM, Lepkowski JM et. al. Low income was an independent risk factor for premature death after controlling for health behaviors. *JAMA* 1998; 279:1703-8.
Dockery 1993 Dockery DW, Pope CA 3d, Xu X, et. al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753-9.
Pope 1995 Pope CA, Thun MJ, Manboodiri MM, et. al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med 1995;151:669-74.*
Pope 2002 Pope CA, Burnett RT, Thun MJ et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 2002; 287:1132-41.
McDonnell 2000 McDonnell WF, Nishino-Ishikawa N, Petersen FF, et.al. Relationship of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in non-smokers. *J Exper Environ Epidemiol* 2000;10:427-436.
Lipfert 2000 Lipfert FW, Perry HM, Miller JP, et.al. The Washington University—EPRI veteran's cohort mortality study: preliminary results. Inhal. Toxicol. 12 S4:41-73.
Pope 2004 Pope CA. Ed. Air pollution and health -- good news and bad. *N Engl J Med* 2004 351;1132-1134.
McFadden 1997 McFadden ER jr., Warren EL. Observations on asthma mortality. *Ann Intern Med* 1997;127:142-7.
Enstrom 2005 Enstrom J. Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. *Inhalation Toxicology* 2005; 17:803-16.
Moolgavkar 2005 Moolgavkar S. Let. Fine particles and mortality. *Inhalation Toxicology 2006;18:93-4.*
Moolgavkar 2005 Moolgavkar S. A review and critique of the EPA's rationale for a fine particle standard. *Reg Tox Pharm* 2005; 42:123-44.

App. A

Abstract only with one table added.

Inhalation Toxicology, 17:803–816, 2005

ISSN: 0895-8378 print / 1091-7691 online
DOI: 10.1080/08958370500240413

Fine Particulate Air Pollution and Total Mortality Among Elderly Californians, 1973–2002

James E. Enstrom
Jonsson Comprehensive Cancer Center, University of California, Los Angeles, California, USA, and Scientific Integrity Institute, Los Angeles, California, USA

Fine particulate air pollution has been associated with increases in long-term mortality in selected cohort studies, and this association has been influential in the establishment of air quality regulations for fine particles ($PM_{2.5}$).However, this epidemiologic evidence has been questioned because of methodological issues, conflicting findings, and lack of an accepted causal mechanism. To further evaluate this association, the long-term relation between fine particulate air pollution and total mortality was examined in a cohort of 49,975 elderly Californians, with a mean age of 65 yr as of 1973. These subjects, who resided in 25 California counties, were enrolled in 1959, recontacted in 1972, and followed from 1973 through 2002; 39,846 deaths were identified. Proportional hazards regression models were used to determine their relative risk of death (RR) and 95% confidence interval (CI) during 1973–2002 by county of residence. The models adjusted for age, sex, cigarette smoking, race, education, marital status, body mass index, occupational exposure, exercise, and a dietary factor. For the 35,789 subjects residing in 11 of these counties, county-wide exposure to fine particles was estimated from outdoor ambient concentrations measured during 1979–1983 and RRs were calculated as a function of these $PM_{2.5}$ levels (mean of 23.4 $\mu g/m^3$). For the initial period, 1973–1982, a small positive risk was found: RR was 1.04 (1.01–1.07) for a 10-$\mu g/m^3$ increase in $PM_{2.5}$.For the subsequent period, 1983–2002, this risk was no longer present: RR was 1.00 (0.98–1.02). For the entire follow-up period, RR was 1.01 (0.99–1.03). The RRs varied somewhat among major subgroups defined by sex, age, education level, smoking status, and health status. None of the subgroups that had significantly elevated RRs during 1973–1982 had significantly elevated RRs during 1983–2002. The RRs showed no substantial variation by county of residence during any of the three follow-up periods. Subjects in the two counties with the highest $PM_{2.5}$ levels (mean of 36.1 $\mu g/m^3$) had no greater risk of death than those in the two counties with the lowest $PM_{2.5}$ levels (mean of 13.1 $\mu g/m^3$). These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983.

Table ten is present as originally published in a pdf file of the article. Attached.

TABLE 10 Relative risk (RR) and 95% confidence interval (CI) for long- term all- cause mortality per 10- μ g/ m^3 increase in $PM_{2.5}$ for U. S. cohort studies based on $PM_{2.5}$ data, circa 1980

$PM_{2.5}$ Study characteristics							
Study (author, year)	Data period/ Mean (range)/ (μ g/ m^3)/ Cohort geographic definition/ Follow- up period/ Mean entry age for period/ Number entered in cohort/ Deaths in follow-up period/ RR (95% CI)						
Males							
Dockery et al., 1993	1979– 1985	19 (11– 30)	6 U. S. cities	1975– 1989	_	50	3671 [a]
	830 [a]	1.15 (1.02– 1.30) [b]					
Pope et al., 1995	1979– 1981	18 (9– 34)	50 U. S. SMSAs	1982– 1989	57	130,310 [a]	_
	12,400 [a]	1.07 (1.03– 1.11) [b]					
McDonnell et al., 2000	1973– 1977	32 (17– 45)	9 CA airsheds	1976– 1992	58	_	1347
_	375	1.09 (0.98– 1.21) [b]					
Lipfert et al., 2000	1979– 1981	24 (6– 42)	42 U. S. counties	1975– 1981	51	26,067	_
	4600 [c]	0.95 (0.89– 1.01) [c]					
	1982– 1984	22 (8– 41)	1982– 1988	57	_	21,467	_
	6100 [c]	0.94 (0.90– 0.98) [c]					
	1982– 1984	22 (8– 41)	1989– 1996	63	_	15,367	_
	5765 [c]	0.89 (0.85– 0.95) [c]					

Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 159,000 [a] _ 36,000 [a] 1.05 (1.01– 1.10)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 66 15,573 4701 1.03 (0.99– 1.07)
1979– 1983 24 (11– 42) 1983– 2002 74 10,872 8831 0.97 (0.95– 1.00)
Females
Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 4440 [a] 599 [a] 1.12 (0.96– 1.30) [b]
Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 164,913 [a] _ 8365 [a] 1.06 (1.01– 1.12) [b]
McDonnell et al., 2000 1973– 1977 32 (17– 45) 9 CA airsheds 1976– 1992 58 _ 2422 _ 568 _ 1.00 (assumed)
Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 200,000 [a] _ 24,000 [a] 1.02 (0.98– 1.06)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 20,210 4094 1.05 (1.01– 1.10)
1979– 1983 24 (11– 42) 1983– 2002 73 16,116 10,815 1.02 (0.99– 1.04)
Both Sexes
Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 8111 1430 1.13 (1.04– 1.23) [b]
Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 295,223 20,765 1.07 (1.04– 1.10) [b]
Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 359,000 _ 60,000 1.04 (1.01– 1.08)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 35,783 8795 1.04 (1.01– 1.07)
1979– 1983 24 (11– 42) 1983– 2002 73 26,988 19,646 1.00 (0.98– 1.02)
[a] Obtained from supplementary data (Krewski et al., 2000). [b] Recalculated from published data (US EPA, 2004). [c] Obtained from the author.

Commentary

Inhalation Toxicology, 18:93–94, 2006

ISSN: 0895-8378 print / 1091-7691 online
DOI: 10.1080/08958370500419207

Fine Particles and Mortality

Suresh H. Moolgavkar

Fred Hutchinson Cancer Research Center Seattle, Washington, USA

In an interesting paper in a recent issue (vol 17, issue 14) of the journal, Enstrom examined the association between fine particulate matter (PM) pollution and mortality in a cohort of elderly Californians. The analyses used proportional hazards regression and after adjusting for age, sex, cigarette smoking, and other potential confounders, Enstrom concluded, "These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983." Enstrom's analyses were based on a sub-cohort of individuals enrolled in the first Cancer Prevention Study (CPS I) conducted by the American Cancer Society (ACS). Enstrom's conclusion is consistent with the conclusions of a cohort study among veterans conducted by Lipfert et al. (2000), but is at odds with the results from analyses of the second ACS cohort (CPS II) by Pope and others (Pope et al., 1995, 2002; Krewski et al., 2000), which reported statistically significant associations between fine particulate pollution and mortality.

Every epidemiological study has weaknesses and limitations and, undoubtedly, both proponents and skeptics of the 'fine par-ticles cause death' thesis will find much to criticize in the studies that do not support their conclusions. These discrepant results raise an important question, however. Can contemporary epidemiological and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders?

All the cohort studies referred to above use proportional haz-ards modeling for data analyses. But is proportional hazards really the appropriate tool for these analyses? First, it is highly unlikely that proportionality of hazards would hold over the en-tire period of time covered by these studies. Statistical tests for departures from proportionality of hazards have low power. En-strom states that, in his analyses, these tests failed to reject pro-portionality of hazards. However, his finding of a higher relative risk associated with fine particles over the period 1973–1982 is inconsistent with proportionality of hazards over the entire

Address correspondence to S. H. Moolgavkar, Fred Hutchinson Cancer Research Center Seattle, WA 98109, USA. E-mail: smool-gav@fhcrc.org

period of the study. Even if proportionality of hazards were to hold for exposure to fine particles, we know that it most defi-nitely does not hold for cigarette smoking, a strong risk factor and a potential confounder of the PM mortality association. For example, we know that, for a given daily level of smoking, the relative risk of lung cancer is strongly dependent on duration of smoking. Moreover, when smokers quit, the relative risk for mortality declines over a period of many years, and not virtually instantly as is assumed by proportional hazards. What influence does this manifestly incorrect model for a strong confounder have on the estimates of air pollution effects? Similarly the use of a proportional hazards model to adjust for age of entry into studies is also suspect.

I have discussed the original CPS II study (Pope et al., 1995) and reanalyses (Krewski et al., 2000; Pope et al., 2002) in de-tail elsewhere (Moolgavkar, 2005). I note here, however, that the reanalysis by Krewski et al. (2000) of the original (Pope et al., 1995) study (which considered no pollutant other than PM), showed quite clearly that the pollutant most strongly as-sociated with mortality was not PM but SO_2.In fact, when SO_2 was considered along with PM in the model for all-cause mor-tality, the coefficient for sulfates was reduced to less than a third of its original value, that for fine particles was reduced to a sixth of its original value, and both became statistically insignif-icant. It is also of interest to note that consideration of spatial correlations attenuated the PM coefficients to a much greater extent than the coefficients for SO_2.Given the much stronger and more robust association of SO_2 with mortality in the CPS II reanalyses, I find it surprising that this study continues to be taken as providing strong support for the PM mortality asso-ciation. It can be plausibly argued on biological grounds that SO_2 could not be causally associated with mortality. But that still does not explain why SO_2 wipes out the PM signal in joint pollutant models. This awkward fact has simply been dismissed as being irrelevant. In a more recent study of the CPS II cohort that doubles the follow-up time and triples the number of deaths, Pope et al (2002) reported significant associations between fine particles and oxides of sulfur with all-cause, cardiovascular and lung cancer mortality. Surprisingly, despite the findings in the Krewski analyses that SO_2 was the pollutant most strongly as-sociated with mortality, no joint pollutant analyses were carried

App. B

Abstract only

A review and critique of the EPA's rationale for a fine particle standard

Suresh H. Moolgavkar
Sciences International, Inc., King Street Station, 1800 Diagonal Road, Suite 500, Alexandria, VA 22314, USA
Received 18 November 2004
Available online 24 March 2005
Abstract
I review the rationale for the Environmental Protection Agency_s 1996 fine particle standard, which was based almost entirely on the epidemiological data with neither support from Toxicology nor understanding of mechanism. While many epidemiological papers available in 1996 reported associations between ambient particles and adverse effects on human health, many others did not and the evidence fell far short of supporting a causal association between particle mass concentration and human health. The literature appearing after 1996 further complicates the picture. The large studies that have appeared after 1996, such as National Mortality Morbidity and Air Pollution Study, and the reanalyses of the American Cancer Society II study, report risks that are substantially smaller than the risks reported in the 1996 Criteria Document and Staff Paper. Moreover, concerns about confounding by weather, temporal trends and co-pollutants remain unresolved. Other issues having to do with model choice have resurfaced as a result of reanalyses of critical data to address a glitch in a widely used software package for time-series epidemiology studies of air pollution. Finally, contemporary examples show that the results of observational epidemiology studies can be seriously biased, par-ticularly when estimated risks are small, as is the case with studies of air pollution. The Agency has largely ignored these issues. I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health. Such a standard may be justifiable on the basis of the precautionary principle, however. The Agency could argue that the Science raises concerns about current levels of air pollution, and that reduction of ambient fine par-ticulate matter mass, if it could be achieved without an increase in the level of the ultrafines, could have positive effects on human health. If the Agency justifies a particulate matter mass standard on these grounds then the debate over the form and level of the standard will, for all practical purposes, belong strictly in the Policy arena.

Keywords: Air Pollution; Particulate matter; Criteria document; Staff paper

Here below is the chapter on Air Quality with a particular emphasis on small (fine) particles pollution, claimed by the EPA to be lethal, harmful and carcinogenic—which is not true—the EPA has never proved any of those claims.

The Chapter is Chapter Seven of a much larger enterprise, of more than 900 pages, challenging what the EPA is doing in many areas of environmental science and policy making. The EPA has become, unfortunately an industry that uses junk science to promote political agendas.

I am the lead author of the chapter, that debunks the EPA claims on small particles in particular with help from two individuals who testified to the CASAC in this session, Steve Milloy and James Enstrom, but there were many others as listed in the credits at the beginning of the chapter. The many contributors are not listed but are referenced.

The Chapter is comprehensive and provides a thorough thrashing of EPA conduct in the matter of small particles air pollution, the serial and unrelenting deceptions of EPA sponsored scientists in matters of junk epidemiology and toxicology.

http://climatechangereconsidered.org/wp-content/uploads/2018/12/6-Air-Quality-final.pdf