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JUNK SCIENCE ON SOOT

Flawed Study Can't Justify Clean Smokestacks Bill

Summary: A new study in the *Journal of the American Medical Association* alleges a significant increase in lung cancer risk for those exposed to high-levels of particulate matter, commonly called soot. In North Carolina, the news media and others have cited the study to boost support for the proposed Clean Smokestacks bill. But according to expert analysis, the study is so flawed that it should have been rejected by the journal. Moreover, it does not establish a case for new regulation.

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Dirty air, cancer risk tied." This was the headline on March 3, 2002 in the Raleigh *News & Observer*. The article discussed a recently published paper by lead author C. Arden Pope and six co-authors (referred to in the remainder of this paper as "Pope, et al.")[1] which appeared in the *Journal of the American Medical Association* (JAMA). The *News & Observer* article opened by saying that "Researchers…have established that long term exposure to fine particles from air pollution from coal fired power plants, factories and diesel trucks can greatly increase an individual's risk of dying from lung cancer." This statement is based on a conclusion in the paper, namely that "long-term exposure to combustion-related fine particulate air pollution *is* an important environmental risk factor for cardiopulmonary and lung cancer mortality" (emphasis added). Subsequent stories and editorials in the *N&O*, *The Charlotte Observer*, and other publications cited this risk factor as another reason for North Carolina lawmakers to enact a proposed set of power-plant regulations called the "Clean Smokestacks" bill.

In reality, the study does not support such a conclusion and, when held to well-recognized standards of statistical analysis, is a clear example of junk science.

Furthermore, the many blatant inconsistencies between the reported results in this study and previous analyses by some of the same authors strongly challenge its credibility. The American Medical Association should be concerned about the apparent failure of its peer review and editorial process in not rejecting the Pope, et al. paper.

What is PM 2.5?

Particulate Matter (PM) 2.5 is a measure of the fine particulate concentration of air pollutants found in the air we breathe. Particles below 2.5 microns in diameter (1/

10,000th of an inch) are transported and deposited into the lung while particles greater than 2.5 microns generally are not. Air monitoring for PM 2.5 involves sampling of the air through a device that separates the particles into two fractions, which are captured on filters and then weighed. The sampling time is 24 hours, usually every six days. These site-by-site data are then combined to obtain the annual average.

Historically, the national health standard was based upon total particle concentrations and since 1987 has been based on particles less than 10 microns. The proposed PM 2.5 standard of 15 micrograms per cubic meter of air (annual average) has been the subject of scientific debate as well as litigation all the way to the U.S. Supreme Court. This author along with Ben Lieberman has reported on the history of this scientific and legal controversy.[2]

Not Science but Bad Statistics

Studies like Pope, et al. are called epidemiological studies and can only identify possible *associations* between, say, air pollution and some health indicator. They cannot define *causation*. The authors claim to be showing that there is a statistical association with certain concentrations of PM 2.5 and mortality rates. There is nothing in this paper that links, in a medical sense, PM 2.5 with any diseases and no such causal connection has been made in actual scientific research.

With respect to the kind of statistical analysis done in the Pope, et al. paper, epidemiology practitioners typically reject or ignore associations showing a risk ratio (RR) of less than 2.[2] This would mean that the Pope, et al. paper would have to show a 100 percent increase, or a doubling of the health impacts of PM 2.5, before it could be considered a meaningful increase in a statistical sense. Note that even without further scrutiny, Pope, et al. reported a maximum RR of 1.14, which is only a 14 percent increase for lung cancer. They also cited other reported values well below this insignificant increase as being "robust" associations. On this basis alone, the analysis should have been rejected by JAMA's editors.

The fact is that Pope, et al.'s basic conclusion — that "…fine particle air pollution *is* an important risk factor…" — is not supported by their study. Not only is such a statement not scientifically meaningful within the context of this kind of study, as noted above, but nowhere in the paper is a causal connection between PM2.5 and lung cancer proven, defended, or even discussed. Unfortunately, this is the line that has been played up by the press in North Carolina and elsewhere.

The Flaws in the Study

A detailed presentation regarding all of the flaws I have found in the Pope, et al. paper would be extensive. However, some very simple but major problems are evident by inspection of their own results, which appear in a graph toward the end of their study.[3] These results are not only deemphasized in the text of the paper, but are in large part ignored. Based on standard rules of scientific acceptability defining what is considered to be a *significant* association[4] the graphed but unnoted results in Pope, et al.'s analysis reveals that:

1. There is no significant association between lung cancer mortality and PM 2.5 among women.
2. There is no association between lung cancer mortality and PM 2.5 for persons under the age of 60 and over the age of 70, but only in the age interval of 60-70.
3. There is no association between lung cancer mortality and PM 2.5 for persons with more than a high school education.
4. There is no association between lung cancer mortality and PM 2.5 for persons who are current smokers or have never been smokers, but only for former smokers.

Pope, et al. offers no explanations for these results. Logic tells us that a lung is a lung and if PM 2.5 causes lung cancer or other lung related disease then the study should show significant positive associations under all hypothetical tests, *assuming* that the underlying data and analytical protocol are sound. This is clearly not the case with the study in question. The hypothetical associations failed to show a significant PM 2.5 association in five of the 11 models run for cardiopulmonary mortality and failed in six of 11 models run for lung cancer mortality for a combined score of 11 failures in 22 cases. These are the same odds as coming up with heads in coin flipping.

On the other hand, the current study did show a positive association between mortality from all causes and sulfur dioxide (SO_2) air pollution in a single-pollutant model — that is, a model that looks at one pollutant in isolation from all others. This is important because two of the authors on this paper did previous work arguing that in a "multi-pollutant model," which considers several pollutants simultaneously, the association between PM 2.5 and mortality disappears. Furthermore, it is replaced by an association with SO_2.

Some background on this issue is important. This is not Pope's first analysis of the statistical relationship between PM 2.5 and mortality. He performed an earlier study for the American Cancer Society (ACS). The current paper in JAMA is a rehash

of the original paper and presents essentially the same results. The ACS study was reviewed and reanalyzed for the Health Effects Institute (HEI) by two of the authors of the JAMA study in question, Daniel Krewski and Richard Burnett. It is in this reanalysis of the original Pope ACS study where the multi-pollutant model was used and the association between mortality and PM 2.5 disappeared.[5]

Since this multi-pollutant model was readily available, the question arises why wasn't it used in this follow up study? This would have been a reasonable way to discover whether the health effects being observed might be associated with pollutants other than PM 2.5. Clearly, a more open-minded inquiry would have allowed for more investigation of this possibility.

The fact is that, despite all its flaws, if we were to take the results of Pope, et al. at face value, it would still mean very little from a public health standpoint. This author has been closely involved as a critical reviewer of the PM 2.5 standard setting process and the use of Pope's earlier research as a reference in establishing that standard.[6] An analysis was conducted to estimate the added health risk to the U.S. population over 30 years of age (the baseline for both the current JAMA study and the original ACS Study) based on current national mortality statistics [7] and EPA PM 2.5 air quality data.[8]

Understanding the Statistics on Cancer Risk

The Pope, et al. study alleged that high levels of exposure to PM2.5 increased the chances of contracting lung cancer by 14 percent. But as one expert put it:

"Increases in relative risk on the order of 100 percent and less . . . should be viewed suspiciously. The esteemed epidemiologist Ernst Wynder even said that relative risks under [200 percent] are suspect. Given the nature of the epidemiologic method and the quality of the data, such small differences could easily be explained by the quality of the data. A 100 percent increase in risk may sound like a lot, but in epidemiology, it's not . . . U.S. smokers have rates of lung cancer that are 10 to 20 times greater than those of nonsmokers — a relatively high rate of a rare disease."

From Steven J. Milloy, Junk Science Judo, *Cato Institute, 2001*

Out of a total of 320 standard metropolitan statistical areas (SMSAs) in the US, 20 have levels of PM 2.5 that are above the EPA's threshold. Using Pope's analysis, and ignoring all of its flaws, we found that the deaths associated with PM 2.5 levels that are above the EPA standard for these 20 metropolitan areas were increased by only 0.31 percent of the total reported deaths for this segment of the total population.[9] In general, 96 percent of all deaths occur in the above-30 age group. Heart disease and cancer account for 53 percent of all deaths. The 0.31 percent calculated based on Pope's dubious results is not even a blip on the radar screen of public health concerns.

Scientific Incest

Clearly the authorship of the Pope, et al. paper raises some questions regarding the independence of their work. As noted, Krewski and Burnett were hired by the Health Effects Institute as part of a Reanalysis Team to critically peer review the original Pope ACS study[10] which forms the basis for the current paper in JAMA. This reanalysis included one other long-term health effects study known as The Harvard Six Cities Study, and cost approximately one million dollars[11] (paid for by the taxpayers through the EPA and interested industry groups).

Krewski and Burnett did not reach the same conclusion about the original Pope ACS paper that they now attest to in this paper. First, the reanalysis team headed by Krewski concluded that the study did not demonstrate causation between mortality and PM 2.5 and that the observed deaths were associated with a mix of pollutants that included PM2.5 but was not limited to it. The reanalysis emphasizes that "it is important to bear in mind that the results of our reanalysis alone are insufficient to identify *causal* relations with mortality. Rather, we can conclude only that *urban* air pollution is associated with increased mortality in these *two* important investigations"[12] (emphasis added). The HEI also appointed a Special Panel of the HEI Health Review Committee to review and comment on the Reanalysis Team's findings. The panel agreed with the reanalysis team's finding that mortality may be attributable to more than one component of the complex mix of ambient air pollutants. They also clearly stated that, "No single epidemiological study can be the basis for determining a causal relation between air pollution and mortality." [13]

As co-authors of the current paper in JAMA, Krewski and Burnett now ignore their own findings. It is also interesting to note that Pope, along with the principal authors of the Harvard Six Cities Study that was part of the HEI reanalysis, was not pleased with the reanalysis. These authors stated that, "Much of the elegance has been lost in the reanalysis…an attempt to

bludgeon the data until they succumbed…We think they went too far."[14] It seems that this "strange bedfellows" authorship is related to a mutual advocacy for more government regulation rather than for sound science.

In what appears to be a blatant attempt to draw attention away from studies that are inconsistent with their conclusions, Pope, et al. ignore discussing a more recent and important third study by Lipfert, et al.[15] This study showed *no* association between PM 2.5 and mortality among a highly sensitive and susceptible cohort of 90,000 male veterans living in 32 cities across the nation. Indeed, Pope, et al. cite only other studies that support their conclusions. In particular they cite only two long-term studies they authored or peer reviewed. In ignoring the broad base of literature in this area and citing only studies that they had personal involvement with, Pope, et al. are not only being incestuous but scientifically irresponsible.

Conclusion

The conclusion drawn by Pope, et al. regarding the mortality implications of fine particle exposure across the U.S. is without scientific merit and should not be taken seriously by policymakers considering air pollution regulations. In spite of this, the news media are reporting the study as gospel and without any further investigation or comment from critics. Furthermore, environmental advocacy groups are using its conclusions to lobby for additional regulations such as the Clean Smokestacks bill that is pending before the North Carolina General Assembly. The paper should have been rejected for publication by JAMA. It is clear, based on this author's knowledge of the historical controversy surrounding Pope's work, that a proper peer review was not conducted by the JAMA before the printing of this paper. It is unfortunate that the popular press sensationalizes such junk science and that legislators often take it seriously.

— Dr. Kay Jones is the head of Zephyr Consulting, a Seattle-based environmental consulting firm. Dr. Jones was a senior advisor on air quality to the President's Council on Environmental Quality during the Ford and Carter administrations. He also served as a senior technical advisor and research manager at the Environmental Protection Agency. His technical comments on the Pope, et al. study will be published in a forthcoming issue of JAMA.

Notes

[1] C. Arden Pope III, Richard Burnett, et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution" *The Journal of the American Medical Association*, March 6, 2002, Vol. 287, No. 9, pp. 1132-1141.
[2] Kay Jones and Ben Lieberman, "The Ongoing Clean-Air Debate, The Science Behind EPA's Rule on Soot," Competitive Enterprise Institute, June 2000.
[3] Op Cit, note 1, p. 1139.
[4] In the language of statistical analysis, the lower error boundary (or confidence interval) around a synthesized risk ratio (RR) must be above 1.0 to be considered a significant association otherwise it is a "due to chance" finding. For an excellent discussion of this issue in non-technical language see Steven Milloy, *Junk Science Judo: Self-Defense Against Health Scares and Scams*, (Washington, D.C.: The Cato Institute), 2001. See also, "Principles for Evaluating Epidemiological Data in Regulatory Assessment," *Federal Focus*, Wash. D.C., Aug. 1996.
[5] Daniel Krewski, Richard Burnett, Mark Goldberg, Kristin Hoover, Jack Siemiatycki, Michael Jarrett, Michael Abrahmowicz, and Warren White, "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality," Health Effects Institute, July 2000. See Table 46, p 207.
[6] See Kay Jones, "Is EPA Misleading the Public About the Health Effects From PM 2.5: An Analysis of the Science Behind EPA's PM 2.5 Standard," Citizens for a Sound Economy Foundation, May 1997 and Kay Jones, Michael Gough, and Peter Van Doren, "Addendum to 'Is EPA misleading the Public about the Health Risks from PM 2.5'," June 1997.
[7] National Vital Statistics Report, National Center for Health Statistics, Vol. 49, No. 11.
[8] Air Quality Statistics by City, 2000, Office of Air and Radiation, U.S. EPA.
[9] See Jones, May 1997 at note 6 for a detailed discussion of the methodology used in these calculations.
[10] Op Cit at note 5.
[11] Ibid., Sensitivity Analysis, Part II, p. 236.
[12] Op Cit at note 5, p. 236
[13] Synopsis for the Particle Epidemiology Project, HEI, July, 2000, p.4.
[14] Op Cit at note 5, "Comments on the Reanalysis," p. 278.
[15] Fred Lipfert et al, "The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results," *Inhalation Toxicology*, Vol. 12, 2000.