"Comments on US EPA's projections of mortality reductions achieved by reducing levels of particulate (PM-2.5) in our ambient (outdoor) air"

Peter A. Valberg, Ph.D., Gradient, 20 University Road, Cambridge, MA 02138
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Gradient

Good morning, Mr. Chairman and members of the subcommittee. Thank you for inviting me to testify this morning. I'm Peter Valberg, principal at Gradient, an environmental consulting firm near Boston. I've worked for many years in public health and human health risk assessment. I've been a faculty member at the Harvard School of Public Health and I was a member of a National Academy of Sciences panel that worked on evaluating public health benefits of air-pollution regulations.

At the outset, we should remind ourselves that, by every public health measure, from infant mortality to life expectancy, we are healthier today, and exposed to fewer hazards, than ever before. Our present-day air is much cleaner now than years ago, thanks to EPA, and our air quality is among the best in the world.

I'm here today to address the method used by EPA in their projections of benefits from reductions in outdoor air particulate levels, called "PM" or ambient $PM_{2.5}$. The dollar value of EPA's calculated benefits is dominated by promised reductions in deaths that EPA assumes to be caused by breathing PM in our ambient air. Asthma is also monetized by EPA as an ambient-air concern.

In understanding health hazards, the solidity of our scientific knowledge, like the solidity of a three-legged stool, is supported by three legs of evidence: one leg is observational studies, or, epidemiology, another leg is experimental studies with lab animals, and the third leg is understanding of biological mechanism. If any leg is weak or missing, the reliability of our knowledge is compromised.

EPA uses the observational studies that examine statistics on two factors, which, in small part, seem to go up and down together. These studies correlate changes in mortality (either temporally, say, on a day-by-day basis, or geographically, say, on a city-by-city basis) with differences in levels of ambient PM from day-to-day, or from locale-to-locale. Statistical associations are indeed reported, and, EPA assumes PM-mortality associations are 100% caused by <u>outdoor</u> PM, no matter what PM levels you may breathe in your own home, car, or workplace.

My key points are (1) the mortality evidence doesn't add up, (2) most of our PM exposure is not from outdoor air, (3) the PM statistical studies cannot identify cause, and (4) outdoor PM is recognized as a minor, not major, contributor to asthma.

(1) The evidence doesn't add up. Lab experiments have carefully examined both human volunteers and animals breathing airborne dust, at PM levels hundreds of times greater than in outdoor air, without evidence of sudden death or life-threatening effects. Moreover, we've studied the chemicals that constitute particles in outdoor air, and no one has found a constituent that is lethal when breathed at levels we encounter outdoors. Remember that the basic science of poisons, toxicology, has shown us that "the dose makes the poison."

(2) Where do people get exposed to airborne dust? The majority of our time is spent indoors. Homes, restaurants, and malls have high levels of PM from cleaning, cooking, baking, and frying. When you clean out your attic or basement, you are breathing much higher PM levels than outdoors. We're exposed to high levels of PM when mowing lawns, raking leaves, or enjoying a fireplace. Yet, in spite of these vastly larger PM exposures, we've no case reports of people who died because of the dust they inhaled while cleaning or barbecuing. We can identify who died from car accidents, food poisoning, firearms, and infections, but, out of the tens of thousands of deaths that EPA attributes to our breathing PM in outdoor air, we can't pinpoint anyone who died from inhaling ambient PM.

(3) Intricate statistical manipulations are required to demonstrate the PM-mortality correlations. The computer models require many assumptions and adjustments, and the results you get depend on the model you use, how you set it up, and how many different tests you run. You need to correct for many non-PM pollutants as well as non-pollution factors that may confound the PM-mortality associations. It's not clear that all confounders have been taken into account, and mere associations cannot establish causality. For example, increased heat-stroke deaths are correlated with increased ice-cream sales, but none of us would suggest that ice cream causes heat stroke. In fact, there are many other examples where spurious associations have been observed.

(4) Finally, on asthma, medical researchers recognize that respiratory infections, mildew, dust mites, pet dander, and stress each play a far greater role in asthma than pollutants in ambient air. Among urban neighborhoods sharing the same outdoor air, both childhood and adult asthma vary considerably by location, and doctors investigating these patterns point to risk factors such as obesity, ethnicity, age of housing stock, and neighborhood violence. Most importantly, over past decades, asthma has gone up during the very same time period that levels of all air pollutants outdoors have markedly gone down. This is opposite to what you would expect if outdoor PM caused asthma.

Taken together, there are major questions about EPA's calculations of "lives saved" by small PM reductions in our outdoor air. Most importantly, neither the animal toxicology nor the human clinical data validate the statistical associations from the observational epidemiology studies. How can it be that lower levels of exposure to outdoor PM are killing large numbers of people when our everyday exposures to higher levels of PM are not?

Thank you for the opportunity to testify today and I look forward to answering your questions.

Biographical Summary

Peter A. Valberg, Ph.D., Principal

Dr. Valberg is an expert in human health risk assessment, inhalation toxicology, and modeling of human exposure to environmental chemicals. He has 30 years of experience on the faculty of the Harvard School of Public Health and at Gradient. Dr. Valberg has provided air quality expertise to the Department of Justice, the US Environmental Protection Agency, and the National Academy of Sciences. He is the author of more than 100 scientific articles on biological effects of environmental exposures on humans and animals. Dr. Valberg's risk assessment expertise covers air pollutants, chemical exposures, biologicals, radionuclides, and EMF (including power lines, radio waves, and cellular telephones). Recent projects have included evaluating health impacts of airborne particulate matter, diesel exhaust, metals, asbestos, sulfuric acid, and TCE. Dr. Valberg is frequently called upon to prepare and interpret health-risk findings for a variety of audiences, and he helps apply research results to the regulatory, litigation, and public policy arenas.

Representative Projects

Health Risk Evaluation for Air Emissions: Evaluated health risks based on stack emissions estimates, air dispersion modeling, comparative dose from different sources, and multiple-pathway health risk assessment.

Hexavalent Chromium: Prepared an in-depth analysis of a risk assessment prepared for exposure to Cr6+ in surface water and groundwater. Compared how different regulatory agencies approach Cr6+ risk assessment. Provided an integrative perspective on how risk calculated for Cr6+ exposure compared to background, everyday risks.

Toxicity of Arsenic in Soils: Evaluated the scientific and epidemiological basis for arsenic toxicity and related toxicity to site-specific arsenic bioavailability. Recalculated how the cancer potency factor for arsenic is affected by water intake assumptions.

Environmental Electric and Magnetic Fields (EMFs): Reviewed and analyzed the various mechanisms by which biological systems may be affected by EMFs. Organized a workshop on EMF and leukemia, with subsequent publication in *Environmental Health Perspectives*.

Radioactive Risks: Used various US EPA and DOE models to evaluate the implications of radioactive substance migration from a contaminated site and assessed the health impact of radioisotopes, including uptake of radioactivity into plants, and, hence, into food.

Assessment of Carbon Black (CB): Evaluated the epidemiology of workers in the CB industry. Identified weight of evidence for CB toxicity for exposure *via* inhalation and ingestion. Reviewed data on the carcinogenicity of CB, and evaluated likelihood of human carcinogenicity for CB.

Airborne Sulfur Dioxide and Sulfuric Acid: Evaluated health impacts from short-term, acute air releases of H_2SO_4 and SO_2, as well as health risks arising from long-term, chronic exposures to these compounds.

Practice Areas & Expertise

- Inhalation Toxicology
- Exposure Modeling
- Airborne Pollutants & Particulates
- Electromagnetic Fields (EMF & RF)
- Radiation & Radionuclide Risk
- Risk Communication & Relative Risk
- Nanotechnology

Education

Ph.D., Physics, Harvard University

M.A., Physics, Harvard University

M.S., Human Physiology and Inhalation Toxicology, Harvard School of Public Health

B.A., Physics and Mathematics, Taylor University

Selected Publications

Hesterberg, TW; Long, CM; Lapin, C; Hamade, A; Valberg, PA. 2010. "Diesel exhaust particulate and nanoparticle exposures: What do human clinical studies tell us about potential human health hazards of nanoparticles?" *Inhalation Toxicology.* 22:679-69

Valberg, PA; Van Deventer, TE; Repacholi, MH. 2007. "Base stations and wireless networks. Radiofrequency (RF) exposures and health consequences." *Environmental Health Perspect.* 115:416-424.

Valberg, PA. 2004. "Is PM more toxic than the sum of its parts? Risk-assessment toxicity factors versus PM-mortality 'effect functions.'" *Inhalation Toxicology.* 16(Suppl. 1):19-29.

Valberg, PA. 2003. "Ambient Particulates and Health Effects." In *A Practical Approach to Occupational and Environmental Medicine* (Ed.: McCunney, RJ), Lippincott Williams & Wilkins, Philadelphia, PA, p835-850.

Valberg, PA; Watson, AY. 2000. "Lack of concordance between reported lung-cancer risk levels and occupation-specific diesel-exhaust exposure." *Inhalation Toxicology* 12(Suppl. 1):199-208.

Gradient

20 University Road
Cambridge, MA 02138
Phone (617) 395-5000
Fax (617) 395-5001

pvalberg@gradientcorp.com
www.gradientcorp.com