

**STATEMENT OF**

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**ON**

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## Summary

Several recent high-profile reports by scientists at NASA, EPA, and elsewhere, have stated that further reducing air pollution emissions would slow climate change and create significant human health benefits, including reducing death rates among the elderly. These predictions ignore the known effects of temperature on elderly mortality rates (cooler winter temperatures increase, and warmer temperatures decrease, average elderly mortality rates), and instead focus on model-projected public health benefits from cleaner air. However, these claimed health benefits are illusory. They rest on a technically unsound approach that consists of finding (or creating or simply assuming) positive statistical associations between historical levels of pollutants and adverse health effects; using the opinions of selected experts to interpret these associations as causal; and then using the assumed (but not factually established) causal relations to project how reducing future exposures will reduce future health risks. EPA, among others, constrains the questions asked and the allowed answers to guarantee predicted positive health benefits from further regulation. This process is not a reliable guide to the truth. Historical associations do not provide a sound basis for predicting health effects caused by future changes in exposures. Real-world data typically contradict the assumptions and conclusions of models that predict that reducing emissions will cause significant health benefits.

It is possible to develop more credible and accurate predictions of health effects by applying relatively objective, reliable methods for causal analysis of data to air pollution and health effects data. Doing so typically reverses conclusions based on expert opinions and associations, showing that claimed significant health benefits from reduced emissions disappear when examined rigorously. At present, strong claims and exciting headlines about the health benefits of reduced emissions should be regarded as products of wishful thinking and poor statistics. More objective methods of causal analysis can and should be used instead.

U.S. House Committee on Energy & Commerce

Subcommittee on Energy and Power

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Chairman Whitfield and Members of the Subcommittee, thank you for inviting me to discuss health risk analysis aspects of greenhouse gas regulations. One way in which policies to reduce greenhouse gas emissions and other air pollution emissions have been promoted recently is to claim that they will not only slow climate change, but that they will also create significant human health benefits from cleaner air (e.g., [Shindell et al., 2012](#); [EPA, 2011](#); [Fann et al., 2012](#); [Yim and Barrett, 2012](#), [www.prnewswire.com/news-releases/air-pollutant-levels-of-particulates-and-ozone-add-to-public-health-burden-138843874.html](http://www.prnewswire.com/news-releases/air-pollutant-levels-of-particulates-and-ozone-add-to-public-health-burden-138843874.html), [NHS, 2012](#)). This claim has been advanced in prestigious scientific journals and by prominent scientists. It tells policy makers and the public that they can confidently expect a double win – health benefits and climate goals – by more tightly regulating emissions. This encouraging prediction is based on selected statistical and computer models which project that significant health benefits, including reductions in mortality rates, will almost immediately following reductions in air pollution. I will address what we know about the truth and certainty of this assertion.

*False Positives and Exaggerated Benefits Claims are Common in Health Effects Research*

Unfortunately, the projected health benefits in these studies and press releases are firmly based on wishful thinking and bad statistics. They do not reflect what *does* happen in the real world,

but only what *would* happen if the assumptions and beliefs of selected experts were correct. They ignore the real effects on human health of changes in temperature – cooler winter temperatures increase mortality rates among the elderly, and warmer temperatures reduce elderly mortality rates (e.g., [Mercer et al., 2003](#)) – to focus instead on model-based predictions of health effects from pollution. Real-world data contradict the assumptions and conclusions of the models that predict further health benefits from further reductions in emissions (e.g., [Wittmaack, 2007](#), [Moore et al., 2012](#)). The frequently repeated causal claim that tighter regulation will produce further health benefits does not hold up when rigorous methods of causal analysis and hypothesis testing are used to test it. Such tests have too seldom been used in the recent scientific literature on projected health effects of air pollutants.

Misplaced optimism that various interventions will cause health benefits has become widespread and well-documented in recent years ([Ottenbacher, 1998](#); [Imberger et al., 2011](#), [Ioannadis, 2005](#)). It is part of an epidemic of false-positive conclusions and optimistically biased claims in scientific articles, discussed last month in the top science journal, *Nature* ([Sarewitz, 2012](#)). That piece warns that, since the early 1990s, “Science’s internal controls on bias were failing, and bias and error were trending in the same direction – towards the pervasive over-selection and over-reporting of false positive results.” As a possible explanation for this trend, the author notes that, “Scientists are rewarded both intellectually and professionally, science administrators are empowered and the public desire for a better world is answered” by these overly optimistic beliefs and claims. These warnings apply forcefully to recent work by EPA and others in projecting health benefits from further reductions in air pollutants ([EPA, 2011](#); [Fann et al., 2012](#); [Shindell et al., 2012](#); [Yim and Barrett, 2012](#)).

## *Unjustified Causal Conclusions are Created by Asking Experts to Interpret Associations*

A standard approach to generating false-positive results has developed in air pollution health effects research, and it is widely followed. Its two main steps are as follows.

- Step 1 is to *identify a positive statistical association between exposures and health effects*. This can always be done. It is easy for a statistician or epidemiologist to select models and assumptions that guarantee a positive result. For example, dividing a positive number of deaths or illnesses observed in a city during some interval of time by a positive average concentration of a pollutant over that interval will necessarily give a positive estimated ratio of deaths per unit of concentration, even if there is no causal relation between them. As another example, if both heart attack risks and pollutant levels are trending down in a city, due to independent causal reasons such as better prevention and treatment of one and tighter regulatory standards for the other, then comparing heart attack risk rates when pollutant levels were high to rates some years later, when both are lower, will show that risk decreases with pollutant levels. It would take to be a bold leap to interpret such arithmetic as telling us anything about causality, but many health effects researchers are willing to make such leaps, and to publish and cite the results as clearly demonstrating causality (e.g., [Clancy et al., 2002](#), [Harvard School of Public Health, 2002](#), [Hůnová et al., 2012](#)). The statistical models and methods deployed are usually (but not always) more sophisticated than in these examples, but the basic logic is the same: choosing a statistical model and some assumptions can always produce positive statistical associations.
- Step 2 is to *ask selected experts whether they believe the statistical associations are causal* ([EPA, 2011](#), [Fann et al., 2012b](#)). If the question is framed the right way, and the right experts are selected, they will usually say yes, or provide high subjective confidence levels that the answer is yes, even if there is no objective basis in the data for an opinion about causality. Experts who have devoted substantial parts of their careers to air pollution health

effects research may feel that if pollutant concentrations are lower than they used to be and mortality rates are lower than they used to be, then it is perfectly clear that one caused the other (e.g., [Harvard School of Public Health, 2002](#)), with no need for further inquiry or rigorous statistical tests of causal hypotheses ([Fann et al., 2012b](#)). These are the experts who tend to end up providing expert opinions about causality to EPA. Even if more cautious scholars subsequently showed that the decline in mortality rates was unrelated to pollutant reductions, proceeding just as quickly with or without them ([Wittmaack, 2007](#)), so that a valid causal link cannot be established from the data ([Pelucchi et al., 2009](#), [NHS, 2012](#)), the myth that one has been established persists ([Moshhammer, 2010](#)). Calculations of disease burdens said to be caused by or attributable to air pollution, based on the original flawed causal interpretations, soon propagate in the literature (e.g., [Röösli et al., 2005](#)), and other experts advising EPA continue to cite the flawed study as a valuable source of evidence (e.g., [Pope, 2010](#)). But all that is really happening is that experts in health effects research literature are offering and citing their own and each other's opinions, without any objective basis in data for supporting such causal conclusions.

These two steps base policy-relevant causal conclusions and predictions on selected expert opinions about *statistical associations*, rather than on proved or rigorously tested *causal relationships*. But, logically, statistical associations only describe whether past observations of exposures and health effects tended to be high or low together. They do not in general correctly predict how a future change in one would affect the other (e.g., [Freedman, 2004](#), [Moore et al., 2012](#)). Yet, this is precisely the practical question of greatest interest to policy makers. Addressing it using statistical associations (as in [EPA, 2011](#); [Fann et al., 2012](#); [Shindell et al., 2012](#); and [Yim and Barrett, 2012](#)) is technically incorrect and misleading: considerable knowledge of causal mechanisms, rather than extrapolation from historical associations, is required to correctly answer how changes in exposures will affect future risks ([Freedman,](#)

[2004](#)). At present, the few rigorous statistical tests of hypothesized causal mechanisms that have been performed for air pollution health effects show that predicted changes did not occur, suggesting that the gap between association and true causation is wide ([Kaufman et al., 2007](#)).

*EPA and Others Constrain All Estimated Health Effects of Regulation to be Positive*

The use of statistical associations to address causal questions about health effects of regulation is not only technically incorrect, but, as practiced by EPA and others, is also highly misleading to policy makers. A positive statistical association between exposures and health effects may exist, or be created by selecting models and assumptions, even if the causal relation between them is non-existent or negative. For example, in assessing human health benefits of the 1990 Clean Air Act Amendments, [EPA \(2011\)](#) selected technical methods that guaranteed positive answers. (Technically, they chose a Weibull uncertainty distribution to quantify the size of the uncertain reduction in mortality caused by a unit reduction in the concentration of fine particulate matter. The Weibull distribution assigns 100% probability to positive values and zero probability to negative (or zero) values, no matter what the data show ([Cox, 2012](#).) Thus, EPA guaranteed that the only possible conclusion would be that reductions in air pollution saved lives, even if rigorous causal analysis of data showed no effect at all. This positive conclusion was the one that EPA announced to the press and to Congress. The possibility that no causal relation exists, so that pollutant reductions would not cause the human health benefits being attributed to them, was briefly acknowledged in one table of the report ([EPA, 2011](#), Table 5-11), but was disregarded in the quantitative uncertainty analysis and in subsequent public presentations and discussions of these and related results ([Fann et al., 2012, 2012b](#)).

Artificial selection of positive findings has flooded the scientific and policy literatures with confident, but unjustified, projections of significant human health benefits from additional regulation of air pollutants. Although analyses such as EPA's insist on disregarding all negative

findings, and consider only all positive values ([EPA, 2011](#)), many significant negative associations have been found between levels of various criteria air pollutants (e.g., NO<sub>2</sub> ([Kelly et al., 2012](#)), PM<sub>2.5</sub> ([Krstić 2010](#)) and ozone ([Powell et al., 2012](#))) and short-term mortality and morbidity rates. As noted by [Powell et al. \(2012\)](#), “The health risks associated with short-term exposure to air pollution have been the focus of much recent research, most of which has considered linear Concentration-Response Functions (CRFs) between ambient concentrations of pollution and a health response. A much smaller number of studies have relaxed this assumption of linearity, and allowed the shape of the function to be estimated from the data. However, this increased flexibility has resulted in CRFs being estimated that appear unfeasible, often showing decreases in the risk to health with increasing concentrations.” Various health effects researchers therefore recommend constraining statistical modeling to show only non-negative associations between air pollution exposures and adverse health effects, no matter what the data show (e.g. [Powell et al., 2012](#), [Roberts, 2004](#)).

A simpler way to guarantee positive findings is to simply assume that risk is proportional to exposure, or that a positive fraction of adverse health outcomes is “attributable” to exposures ([Ezzati et al., 2006](#)). This is now the standard approach used to assess air pollution health risk burdens, leading to conclusions that even low levels of pollution create substantial burdens of mortality and morbidity (e.g., [Elliott and Copes, 2011](#)). However, such “findings” only reflect the starting assumptions that exposure causes risk, and that an increase or decrease in exposure will cause a proportional increase or decrease in health risks. Projections of health benefits based on such circular reasoning are liable to prove disappointing when true causal relations do not match assumptions. In practice, the assumptions that lead to predictions of health benefits from further regulation of emissions are contradicted by experience. Even substantial reductions in pollutants have had no detectable effect on mortality rates or other health effects (contrary to predictions, subjective interpretations, and some earlier statistical modeling) (e.g., [Wittmaack,](#)

[2007](#), [Moore et al., 2012](#)) and have failed to produce the physiological changes that they were expected and hypothesized to cause ([Kaufman, 2007](#)). Belief in further health benefits from further reductions in emissions is premature.

*Doing Better: More Objective Tests for Causality Are Readily Available and Should be Used*

The current literature on health effects of air pollution abounds with false positives, unjustified causal claims, and unrealistic projections of health benefits of air pollution regulation and emissions reductions (e.g., [Fann et al., 2012b](#), [Shindell et al., 2012](#); and [Yim and Barrett, 2012](#)). These claims are technically unsupportable and that should never have been made ([Cox, 2012](#)).

This state of affairs is unnecessary. Excellent methods for answering causal questions more correctly and objectively have been developed over the past six decades, primarily outside the air pollution health effects literature. Air pollution health effects researchers should make greater use of them. Conversely, they should stop relying on expert interpretations of ambiguous (real or assumed) statistical associations as a basis for making health claims. Unlike expert judgments about the causal interpretation of statistical associations, modern technical methods of causal analysis can be independently replicated by others based on data, using standard statistical methods and software. They address the following key factual questions.

1. *Is there objective evidence of an effect (e.g., a significant change in a health effects time series following a change in exposures)?* (Modern technical methods for answering this question include change-point analysis ([Friede et al., 2006](#)), intervention analysis ([Helfenstein, 1991](#); [Gilmour et al., 2006](#)), and panel data analysis ([Stebbing, 1976](#))). Such formal tests may give very different answers from subjective expert interpretations of the same data ([Wittmaack, 2007](#)). *Panel data analysis* examines how well changes in

explanatory variables predict changes in responses. The results are often very different from those predicted by regression models of health effects of air pollutants ([Stebbing, 1976](#)).

2. *If an effect exists, how large is it?* (This may be assessed via intervention analysis, change-point models, panel data, or quasi-experimental pre-post comparisons ([Campbell and Stanley, 1966](#)), with counterfactual causal models untangling the effects of confounders and estimating the remaining effect specifically caused by exposures ([Moore et al., 2012](#)).)
3. *Can changes in health effects over time be explained or predicted as well without knowledge of a pollutant levels as with it?* (This key question can be answered using objective statistical tests for causal hypotheses, such as Granger tests ([Eichler and Didelez, 2010](#)) for multiple time series; conditional independence tests for time series or cross-sectional data ([Freedman, 2004](#), [Friedman and Goldszmidt, 1998](#)), and quasi-experimental analyses to refute other (non-causal) explanations that threaten valid causal inference ([Campbell and Stanley, 1966](#); [Maclure, 1990](#)).)
4. *Are changes in causal predecessors predicted by hypothesized causal mechanisms actually observed?* This can be addressed using causal graph models and panel data analysis applied to biomarker data ([Hack et al., 2010](#)).

Such methods of causal analysis are vastly more powerful guides to objective truth than the current procedure of asking selected experts to offer opinions about causality based on (usually ambiguous and non-causal) statistical associations.

### *Conclusions*

Taking seriously the need to apply more objective methods to assess causality in air pollution health effects research suggests the following policy-relevant conclusions.

- *Expert judgment-based assessments of causality, and causal interpretations of statistical associations, (used at EPA, WHO, and elsewhere) are unreliable and prone to error and bias.* The warnings of methodologists ([Ioannidis, 2005](#); [Sarewitz, 2012](#)) about prevalent biases toward false-positives and inflated estimates of the benefits caused by interventions, appear to apply forcefully to air pollution health effects and accountability research. They should be heeded.
- *It is possible and practical to do better.* More objective methods for causal analysis are now readily available. Using them can eliminate much of the current speculation and ambiguity surrounding causation in health effects research, and correct erroneous preconceptions.
- *The credibility of expert opinions or analyses leading to conclusions about causation, and the credibility of health benefits projections based on them, should be assessed based on how well they provide sound, independently reproducible, answers to specific causal questions.* These questions include whether changes in exposures do in fact precede, and uniquely help to explain or predict, changes in health effects. Passionate or confident beliefs asserted by regulators and subject matter experts who have not yet answered these questions using data and independently reproducible analyses should be regarded as expressions of personal belief, not answers to scientific questions.

These recommendations could promote future health benefits estimates for emissions reductions that are more realistic, and more solidly based on reproducible science and data, than those driving headlines and calls for further regulation today. They would reduce needless controversies over the interpretation of ambiguous statistical associations; focus attention on the sizes of demonstrable real-world causal impacts; and shift the emphasis of health effects claims for emissions reductions toward more objective and independently verifiable risk analysis. Health benefits from emissions reductions might well turn out to be undetectably small in some cases ([Wittmaack, 2007](#)). Using sound causal analysis is the best way to find out.

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