THE GEORGE WASHINGTON UNIVERSITY

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Public Interest Comment[1] on

The Office of Management and Budget's
Draft 2014 Report to Congress on the Benefits and Costs of Federal Regulations

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The George Washington University Regulatory Studies Center works to improve regulatory policy through research, education, and outreach. As part of its mission, the Center conducts careful and independent analyses to assess rulemaking proposals from the perspective of the public interest. The following comments on the Office of Management and Budget's Draft 2014 Report to Congress are offered to help improve a document that already expresses several important points and caveats about the estimated benefits from regulations.

1. On p. 15, the draft report notes that "EPA, with the endorsement of its Clean Air Scientific Advisory Board, has determined that the weight of available epidemiological evidence supports a determination of causality." However, this mistakenly treats causality as if it were dichotomous: something that can be determined to be either present or not in interpreting epidemiological evidence. But this is an oversimplification. In reality, some *fraction* of an epidemiological association might be due to a causal impact of one variable on another. Another fraction might be explained by confounding; a third part might result from model specification error, a fourth from unmodeled uncertainties in estimated values of explanatory variables, and so forth. Thus, the true challenge facing regulators should not be represented as being to make a determination about *whether* evidence supports a causal interpretation (a yes-no determination), but rather to determine *what fraction* (if any) of an association is causal. For $PM_{2.5}$ and human health effects, this causal fraction has never been determined.

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Yet, without knowing it, policymakers lack the crucial factual information required to make well-informed policy decisions based on the probable health consequences of alternative choices.

2. Traditional weight-of-evidence considerations are neither necessary nor sufficient nor, in many cases, useful or trustworthy guides for drawing valid inferences about causation (Rothman et al., 2005). The fact that they are often used for this purpose by experts in epidemiology does not justify such usage (e.g., Freedman, 2004). For at least the past two decades, epidemiologists and commentators on scientific methods and results have raised concerns that current practices lead to too many false-positive findings and too often attribute causality to statistical associations (e.g., Lehrer, 2012, Sarewitz, 2012, Ottenbacher, 1998; Imberger et al., 2011). Limitations of human judgment and inference, such as confirmation bias (finding what we expect to find), motivated reasoning (concluding what it pays us to conclude), and overconfidence (mistakenly believing that our own beliefs are more accurate than they really are), do not spare health scientists. Investigators with expertise in the health effects of $PM_{2.5}$ or other pollutants are not necessarily also experts in causal analysis, and their causal conclusions are often mistaken, often with a bias toward finding "significant" effects where none actually exists (false positives) (Lehrer, 2012, Sarewitz, 2012, Ioannadis, 2005, *The Economist*, 2013).

3. The most recent evidence on air pollution health effects is ambiguous, and does not support a clear, objectively sound causal inference that reducing $PM_{2.5}$ causes reductions in elderly mortality rates, as assumed by EPA. To the contrary, recent and current advances in methodological understanding cast doubt on, and often outright refute, previously published causal interpretations of air pollution health effects data that EPA has cited and used (e.g., HEI, 2010; HEI, 2013). Table 1 provides some examples of important policy-relevant conclusions and doubts about their validity from the recent air pollution health effects literature. As noted by Dominici et al., (2014), "[A]nalyses of observational data have had a large impact on air-quality regulations and on the supporting analyses of their accompanying benefits, [but] associational approaches to inferring causal relations can be highly sensitive to the choice of the statistical model and set of available covariates that are used to adjust for confounding. … There is a growing consensus… that the associational or regression approach to inferring causal relations—on the basis of adjustment with observable confounders—is unreliable in many settings." The authors demonstrate that the choice of regression model can result in either statistically significant positive or statistically significant negative associations between air pollutant levels and mortality rates. This implies that implicit modeling choices can greatly affect—or even determine—the results presented to decision-makers and the public. Inviting selected experts to pass judgment on whether selected associations might be causal is not a satisfactory substitute for objective causal

analysis. It prone to the many limitations of expert opinions about causation, some of which have proved to be unwarranted (e.g., HEI, 2010; HEI, 2013); and, as remarked previously, it begs the question of what fraction of an association is causal.

Table 1. Some conflicting claims about health effects known to be caused by air pollution

Pro (causal interpretation or claim)	Con (counter-interpretation or claim)
"Epidemiological evidence is used to quantitatively relate $PM_{2.5}$ exposure to risk of early death. We find that UK combustion emissions cause ∼13,000 premature deaths in the UK per year, while an additional ∼6000 deaths in the UK are caused by non-UK European Union (EU) combustion emissions" (Yim and Barrett, 2012).	"[A]lthough this sort of study can provide useful projections, its results are only estimates. In particular, although particulate matter has been associated with premature mortality in other studies, a definitive cause-and-effect link has not yet been demonstrated" (NHS, 2012)
"[A]bout 80,000 premature mortalities [per year] would be avoided by lowering $PM_{2.5}$ levels to 5 μg/m^3 nationwide" in the U.S. 2005 levels of $PM_{2.5}$ caused about 130,000 premature mortalities per year among people over age 29, with a simulation-based 95% confidence interval of 51,000 to 200,000 (Fann et al., 2012).	"Analysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence… However, epidemiological evidence alone cannot establish this causal link" (EPA, 2011, Table 6-11). Significant negative associations have also been reported between $PM_{2.5}$ (Krstić 2010) and short-term mortality and morbidity rates, as well as between levels of some other pollutants (e.g., NO_2 (Kelly et al., 2012) and ozone (Powell et al., 2012)) and short-term mortality and morbidity rates.
"Some of the data on the impact of improved air quality on children's health are provided, including… the reduction in the rates of childhood asthma events during the 1996 Summer Olympics in Atlanta, Georgia, due to a reduction in local motor vehicle traffic" (Buka et al., 2006). "During the Olympic Games, the number of asthma acute care events decreased 41.6% (4.23 vs. 2.47 daily events) in the Georgia Medicaid claims file," coincident with significant reductions in ozone and other pollutants (Friedman et al., 2001).	"In their primary analyses, which were adjusted for seasonal trends in air pollutant concentrations and health outcomes during the years before and after the Olympic Games, the investigators did not find significant reductions in the number of emergency department visits for respiratory or cardiovascular health outcomes in adults or children." In fact, "relative risk estimates for the longer time series were actually suggestive of increased ED [emergency department] visits during the Olympic Games" (HEI, 2010)
"An association between elevated PM_{10} levels and hospital admissions for pneumonia, pleurisy, bronchitis, and asthma was observed. During months when 24-hour PM_{10} levels exceeded 150 micrograms/m3, average admissions for children nearly tripled; in adults, the increase in admissions was 44 per cent." (Pope, 1989)	"Respiratory syncytial virus (RSV) activity was the single explanatory factor that consistently accounted for a statistically significant portion of the observed variations of pediatric respiratory hospitalizations. No coherent evidence of residual statistical associations between PM_{10} levels and hospitalizations was found for any age group or respiratory illness." (Lamm et al., 1996)
"Reductions in respiratory and cardiovascular death rates in Dublin suggest that control of particulate air pollution could substantially diminish daily death....Our findings suggest that control of particulate air pollution in Dublin led to an immediate reduction in cardiovascular and respiratory deaths." (Clancy et al., 2002) "The results could not be more clear, reducing particulate air pollution reduces the number of respiratory and cardiovascular related deaths immediately" (Harvard School of Public Health, 2002).	Mortality rates were already declining long before the ban, and occurred in areas not affected by it. "Serious epidemics and pronounced trends feign excess mortality previously attributed to heavy black-smoke exposure" (Wittmaack, 2007). "Thus, a causal link between the decline in mortality and the ban of coal sales cannot be established" (Pelucchi et al., 2009). "In contrast to the earlier study, there appeared to be no reductions in total mortality or in mortality from other causes, including cardiovascular disease, that could be attributed to any of the bans. That is, after correcting for background trends, similar reductions were seen in ban and non-ban areas." (HEI, 2013)

Source: Adapted from Cox (2013)

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4. It is possible, and relatively straightforward, to use more sound and objective methods of causal analysis and inference to quantify the extent to which reducing pollution might reduce future adverse health effects, such as elderly mortality rates. Table 2 lists several quantitative methods for causal hypothesis testing, modeling, and analysis that have been extensively developed and applied over the past six decades (Cox, 2013). Various advantages of these techniques, as compared to qualitative causal criteria (Rothman et al., 2005) such as the traditional Hill considerations and other weight-of-evidence and associational methods, are well explained and illustrated in the references for Table 2 (e.g., Greenland and Brumback, 2002), along with their limitations (e.g., Freedman, 2004). Prominent among these advantages is the development of empirically testable implications of causal hypotheses, such as conditional independence implications, timing implications, information-theoretic implications, and exogeneity implications, with conditional probability distributions of some variables being determined by the values of others. These testable implications capture the asymmetry inherent in the notion of causation, unlike correlations or other symmetric measures of association. They can be tested statistically using publicly available standard computer codes, such as those in R and Python/NumPy. This enables different investigators, perhaps with very different prior beliefs, to reach the same conclusions from the same data. This points the way toward greater objectivity and definitiveness in determining via such tests the extent to which data do or do not support causal hypotheses, based on their testable implications. Other reasons why modern methods of quantitative causal analysis should be (and increasingly are) included among current approaches in the epidemiologist's tool kit are discussed in modern epidemiology textbooks and monographs (e.g., Hernan and Robbins, 2011) and in the references to Table 2.

5. In the relatively very few cases where relatively sound, objective methods of causal analysis (e.g., Granger causality tests, other methods in Table 2) have been applied, there has turned out to be no objective evidence that reducing $PM_{2.5}$ levels in recent decades has caused any corresponding reductions in all-cause or CVD elderly mortality rates (e.g., Cox et al. 2012, 2013), or that other reductions in air pollution have caused the health benefits that have often been attributed to or predicted for them using questionable association-based methods (Dominici et al., 2014) and weight-of-evidence or other expert judgments (Table 1). Thus, there is very high value-of-information to reassessing using rigorous causal analysis whether the Clean Air Act Amendments have caused any of the benefits that EPA has attributed to them—and, if so, what fraction of these projected benefits have actually been caused by Clean Air Act regulations (as opposed, for example, to coincident historical trends in prevention, early diagnosis, and treatment of CVD).

Table 2. Some formal methods for modeling and testing causal hypotheses

Method and References	Basic Idea	Appropriate study design
Quasi-experimental design and analysis (Campbell and Stanley, 1966)	Can control group comparisons refute alternative (non-causal) explanations for observed associations between hypothesized causes and effects, e.g., coincident trends and regression to the mean? If so, this strengthens causal interpretation.	Observational data on subjects exposed and not exposed to interventions that change the hypothesized cause(s) of effects.
Conditional independence tests (Freedman, 2004, Friedman and Goldszmidt, 1998)	Is hypothesized effect (e.g., cardiovascular disease (CVD) mortality rate) statistically independent of hypothesized cause (e.g., $PM_{2.5}$ concentration), given values of other variables (e.g., education and income)? If so, this undermines causal interpretation.	Cross-sectional data; Can also be applied to multi-period data (e.g., in dynamic Bayesian networks)
Panel data analysis (Angrist and Pischke, 2009, Stebbings, 1976)	Are changes in exposures followed by changes in the effects that they are hypothesized to help cause? If not, this undermines causal interpretation; if so, this strengthens causal interpretation. Example: Are reductions in $PM_{2.5}$ levels followed (but not preceded) by corresponding changes in CVD mortality rates?	Panel data study: Collect a sequence of observations on same subjects or units of observation (e.g., counties) over time
Granger causality test (Eichler and Didelez, 2010)	Does the history of the hypothesized cause improve ability to predict the future of the hypothesized effect? If so, this strengthens causal interpretation; otherwise, it undermines causal interpretation. Example: Can CVD mortality rates be predicted better from time series histories of $PM_{2.5}$ levels and mortality rates than from the time series history of mortality rates alone?	Time series data on hypothesized causes and effects
Intervention analysis and change point analysis (Helfenstein, 1991; Gilmour et al., 2006)	Does the best-fitting model of the observed data change significantly at or following the time of an intervention? If so, this strengthens causal interpretation. Do the quantitative changes in hypothesized causes predict and explain the subsequently observed quantitative changes in hypothesized effects? If so, this strengthens causal interpretation. Example: Do mortality rates fall faster in counties where pollutant levels fall faster than in other counties?	Time series observations on hypothesized effects, and knowledge of timing of intervention(s) Quantitative time series data for hypothesized causes and effects
Counterfactual and potential outcome models (Moore et al., 2012)	Do exposed individuals have significantly different response probabilities than they would have had if they had not been exposed? Example: Do people have lower mortality risk after historical exposure reductions than they would have had otherwise?	Cross-sectional and/or longitudinal data, with selection biases and feedback among variables allowed
Causal network, path analysis, and structural equations models of change propagation (Hack et al., 2010)	Do changes in exposures (or other causes) create a cascade of changes through a network of causal mechanisms (represented by equations), resulting in changes in the effect variables? Example: Do relatively large variations in daily levels of fine particulate matter ($PM_{2.5}$) air pollution create corresponding variations in markers of oxidative stress in the lungs?	Observations of variables in a dynamic system out of equilibrium
Negative controls (for exposures or for effects) (Lipsitch et al., 2010)	Do exposures predict health effects better than they predict effects that cannot be caused by exposures? Example: Do pollutant levels predict cardiovascular mortality rates better than they explain car accident mortality rates? If not, this weakens causal interpretation of the CVD associations.	Observational studies

Source: Adapted from Cox (2013)

6. OMB would serve the public interest and the needs of public decision-makers by emphasizing that any responsible discussion of uncertainties in EPA's benefits estimates should include *discrete* uncertainties—e.g., the probability that the correct value for health benefits is actually zero. EPA has used an approach to uncertainty analysis (based in part on Weibull uncertainty distributions) that implicitly assumes a 100% probability of positive benefits. This is stacking the deck. When the possibility of discrete uncertainties is modeled using discrete probabilities (rather than best guesses and surrounding confidence intervals based on Weibull or other continuous uncertainty models), it appears that the probability that the true health benefits are zero could well exceed 90% (Cox, 2012). This is very decision-relevant information that has not been emphasized in EPA's presentations to Congress or other public decision-makers.

7. On page 18 of the draft report, OMB mentions the assumption that "The value of mortality risk reduction, which is taken largely from studies of the willingness to accept risk in the labor market is an accurate reflection of what people would be willing to pay for incremental reductions in mortality risk from air pollution exposure and these values are uniform for people in different stages of life or with differing health status." Yet, EPA's calculations show most benefit as accruing to the elderly. Valuing a mortality risk reduction the same for an 85-year old as for a 25-year old worker lacks any sound foundation.[3] Moreover, the arithmetic of EPA's calculation is problematic, since it emphasizes a count of premature deaths avoided rather than any type of life-years gained or age-specific shift in survival probabilities. These points are discussed in more detail in Cox (2012).

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[3] See, for example, "Public Interest Comment on The Office of Management and Budget's Draft 2014 Report to Congress on the Benefits and Costs of Federal Regulations" by Susan Dudley, Brian Mannix and Sofie Miller. The George Washington University Regulatory Studies Center. September 2, 2014. Available at: https://regulatorystudies.columbian.gwu.edu/sites/regulatorystudies.columbian.gwu.edu/files/downloads/2014-OMB-Report-to-Congress.pdf

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