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ON

EPA'S PROPOSED OZONE RULE: POTENTIAL IMPACTS ON MANUFACTURING

BEFORE THE

SUBCOMMITTEE ON ENERGY AND POWER AND THE SUBCOMMITTEE ON COMMERCE, MANUFACTURING, AND TRADE

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SUMMARY

To determine whether EPA's Proposed Ozone Rule would serve the public interest, it is important to understand (1) Whether and to what extent it would truly cause the improvements in public health that EPA predicts; and (2) How sure we are about the answer. However, EPA's own health effects risk assessment report for ozone admits that their estimation of health impacts uses inaccurate models for which they have been unable to quantify uncertainties in predictions and conclusions. This leaves policy makers and the public uninformed about an issue crucial for sound policy-making: How likely is it that the Rule will cause the public health benefits that EPA estimates, or that it will instead produce other outcomes, such as zero health benefits?

However, there is overwhelming evidence that EPA's predictions of public health benefits from the Proposed Ozone Rule are unwarranted and exaggerated. They are unwarranted because EPA's conclusions about the causal impacts of ozone reductions on public health are not derived from objective science or statistical analyses of causation. Instead, EPA's conclusions rely on unreliable subjective judgments of selected experts; on models that they concede are inaccurate and have large but unquantified uncertainties; and on mistakenly treating association or correlation as causality. None of these methods produces trustworthy conclusions.

We also know from extensive real-world experience that EPA's benefits estimates are exaggerated. Ozone levels have already fallen in recent decades by far more than the proposed amounts in many locations in the United States. Yet analysis of public health records shows that these large reductions in ozone levels have caused no detectable public health benefits. Thus, EPA's assumption that smaller future reductions in ozone will do so is unwarranted. There is no need to repeat the costly effort to obtain better public health by further reducing ozone levels when we already know from abundant historical experience that doing so does not work.

Introduction

Chairman Burgess and Members of the Subcommittee, thank you for inviting me to discuss the human health aspects of EPA's Proposed Ozone Rule. I am testifying on my own behalf today, understanding that well-informed policy making must consider the likely and foreseeable impacts of the proposed rule on human health, as well as on economic end points. I have provided the Committee members with a detailed CV describing my academic, publishing, professional, and consulting affiliations.

In evaluating whether costly proposed regulations are in the public interest, two questions stand out: (1) How well will a regulation work in reality, i.e., will it actually cause the desired benefits that motivate it?; and (2) How sure can we be? EPA's Proposed Ozone Rule is intended to protect and improve human health by reducing human mortality and morbidity risks, especially those from respiratory and cardiovascular illnesses. These projected benefits are to be caused by further reducing allowed ambient concentrations of ozone. It is therefore important to ask to what extent the proposed rule will produce these desired improvements in health, and how sure we can be that it will do so.

The rest of this testimony makes the following main points.

- **First, by EPA's own account, they have not quantified their very large uncertainty about the public health benefits that their models project.** This is unacceptable in a risk assessment prepared to inform public policy decision-making. The public health benefits that EPA predicts from lowering ozone levels are purely hypothetical results of models that EPA itself recognizes are inaccurate. A proper quantitative uncertainty analysis might conclude that, with something like 95% confidence, these heath benefits either do not exist or

are so much smaller than EPA has estimated that they cannot be found in massive amounts of past data (Cox and Popken, 2015).

- **EPA's conclusions about public health effects caused by ozone reductions are based on subjective opinions, not objective science.** EPA has relied on notoriously unreliable methods, including asking selected experts for their opinions, using models that are convenient but inaccurate, and assuming that correlation or association can be treated as causality, to reach its conclusions. None of these methods produces reliable or trustworthy conclusions.
- **EPA's Proposed Ozone Rule will not cause the benefits to public health that EPA models project – and we can be certain of this now.** If we look at actual data instead of at EPA's model-based predictions, it is clear that, in many places in the United States, much larger reductions in ozone levels have already occurred in recent decades than those that are now being proposed. Yet, these relatively large reductions in ozone levels have caused no detectable public health benefits. Therefore, EPA's assumption that future proposed reductions in ozone will do so is unwarranted. Such changes have been tried and they have not worked: their predicted public health benefits have not materialized.

1. EPA has not quantified large uncertainties about its predictions for public health risk reductions caused by lowering the ozone standard

EPA has been candid about some of the uncertainties in its modeling of predicted public health benefits from further reducing ozone levels. For example, it states that it has used a modeling approach that "is convenient for fitting the model, but is not accurate. The extent to

which this mis-specification affects the estimates of the… model and its predictions is not clear." (EPA, 2014, http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100KBUF.TXT). EPA further explains that "[I]t may be that selection bias has influenced the model parameter estimates. … [The] model is also sensitive to the exposure concentrations, but we have not quantified that sensitivity. …We are unable to properly estimate the true sensitivities or quantitatively assess the uncertainty… EPA staff have identified key sources of uncertainty with respect to the lung function risk estimates. … At this time we do not have quantitative estimates of uncertainty for any of these." In short, EPA uses a model that is known to be inaccurate to predict benefits from reducing ozone. EPA's ozone health risk assessment provides no quantitative assessment of uncertainty about whether or to what extent the projected human health benefits would actually occur if the proposed rule were implemented, thus depriving policy makers of the opportunity to see just how hypothetical and unlikely the projected health benefits really are. Failing to properly quantify uncertainties implies that the basic scientific and analytic work required to support well-informed and responsible policy making has not yet been done.

2. EPA's conclusions about public health effects caused by ozone reductions are based on unreliable subjective opinions, not objective science

EPA's conclusions that current standards do not fully suffice to protect public health with an adequate margin of safety and that further reductions in ambient ozone would probably further reduce mortalities and morbidities in the population are derived from its judgment that short term O3 exposures are "causally related to respiratory effects, and likely causally related to cardiovascular effects;" and that long term O3 exposures are likely causally related to respiratory

effects (EPA, 2014 citing EPA, 2013). Remarkably, ***these key causal conclusions are not supported by any reliable, objective statistical tests for potential causality***. They are supported solely by the subjective judgments of selected experts applied to associational data that show that both ozone levels and adverse health effects are higher in some times and places than in others.

The track record of such expert judgments is poor. They are easily influenced by the biases and ideologies of the experts who are invited to give them (Kahneman, 2011). Experts who have opined that reducing pollution causes reductions in mortality or morbidity rates might have the opposite opinion if required to present objective statistical analyses supporting their judgments about causation. For example, a confident and influential causal expert judgment that banning coal burning reduced all-cause and cardiovascular mortality rates (Harvard School of Public Health, 2002) was recently replaced by a finding that there was no objective evidence of these causal effects based on a more objective statistical comparison of mortality rates inside and outside the affected area (Health Effects Institute, 2013).) EPA's health risk assessment for ozone depends critically on judgments about causality that are not supported by any objective statistical causal analyses. Such expert judgments are unreliable. They might well be reversed if different experts were used, or if the experts who have made them were required to use and display objective analyses rather than personal beliefs as a basis for their findings.

Perhaps even more importantly, ***there is now broad scientific consensus outside the EPA-funded air pollution health effects community that associational data – that is, data of the type relied on throughout EPA's health effects risk assessment for ozone – do not in general provide reliable information about causation.*** As stated in a 2014 paper in *Science*, "There is a growing consensus in economics, political science, statistics, and other fields that the associational or regression approach to inferring causal relations—on the basis of adjustment

with observable confounders—is unreliable in many settings" (Dominici et al., 2014). Yet, this is precisely the approach that EPA has taken to estimate health risks from ozone and to predict human health benefits from further reductions in ozone. Throughout EPA's health risk assessment and supporting documents (www.federalregister.gov/articles/2014/12/17/2014-28674/national-ambient-air-quality-standards-for-ozone#t-3; www.epa.gov/ttn/naaqs/standards/ozone/data/20140829healthrea.pdf; http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=247492#Download), associational and regression approaches are mistakenly treated as if they described causal relations. This ***fundamental error, treating correlation as causality, invalidates EPA's entire risk analysis and its conclusions.*** It is the same type of logical error as would be involved if one were to divide car accidents per year in a population by pounds of potatoes consumed per year in that population, and then were to predict on the basis of the resulting positive "slope factor" ratio of car-accidents-per-pound-of-potatoes that reducing potato consumption would reduce car accidents. In both this toy example and in EPA's real calculations of mortalities or morbidities avoided per ppb of ozone reduced, the fundamental error is to use the ratio as a basis for prediction without first showing that changes in the denominator cause any changes in the numerator.

Expert opinions that exposure-response associations or ratios are causal do not successfully address this problem. First, they not even try to address the question of what *fraction* of the association is causal – that is, what proportion of the slope factor ratio, if any, reflects a causal relation between the exposure in the denominator and the health effect in the numerator; and what proportion reflects non-causal sources of association, such as coincident historical trends (both exposure and effects are declining over time, apart from any causal relation between them), seasonal effects (e.g., both ozone and mortality rates are higher at some times of year than at others), or modeling choices (e.g., EPA's use of a convenient but

inaccurate, misspecified model). EPA asked its experts the wrong question, how probable it is that the statistical association between ozone and health effects is causal, rather than asking what fraction is causal. Second, answers to causal questions and opinions based on causal judgments are not warranted for associational data, such as that which EPA has relied on. Associations or ratios between historical health effects and historical exposure levels do not reveal how *future* changes in exposures would affect *future* changes in health effects, which is the question of practical and policy interest. Thus, EPA's prediction of human health benefits from further reductions in ozone reflects wishful thinking and bad statistics, but not sound science or sound analysis. Because no objective methods of causal analysis have been used in EPA's risk assessment that would allow valid predictions about how or whether further reductions in ozone will affect public health, there is no legitimate basis for projecting any human health benefits from the Proposed Ozone Rule.

3. **EPA's Proposed Ozone Rule will not cause the benefits to public health that EPA models project – and we can be certain of this now.**

Fortunately, it is possible to do much better. More objective, reliable statistical methods od causal analysis that depend on data rather than on expert judgments have been extensively developed and applied in areas such as econometrics and social statistics (see references in Harris et al., 2004 and 2006 and Hipel, 1978), neuroscience (Vincente et al., 2011), epidemiology (Joffe et al., 2012; Robins et al., 2000), physics (Runge et al., 2012), artificial intelligence (Voortman et al., 2008), and machine learning (Sun, 2008). Major companies such as Microsoft and Google, that make or lose money depending on how well they understand the

causal relation between what they do and how people respond, have contributed to a growing body of high-quality statistical algorithms and software for testing causal hypotheses and estimating causal impacts (https://google.github.io/CausalImpact/CausalImpact.html). Modern methods of causal analysis apply sound, objective principles, such as that information flows from causes to their effects. These principles lead to independently reproducible and verifiable quantitative tests and conclusions about causality, rather than to subjective qualitative judgments that may differ from expert to expert.

There is thus no need to rely on EPA's inaccurate models, or on mistaken assumptions that historical association can be substituted for future causation, or on the opinions and judgments of selected experts, in order to determine how changes in ozone levels affect changes in human health. In effect, the experiment of reducing ozone levels and seeing what happens to public health has now already been done many times, as ozone levels have fallen dramatically and health statistics have been maintained for decades in many locations throughout the United States. Examining the historical record using objective statistical methods for causal analysis answers the question of what really happens to public health when ozone levels are reduced.

In contrast to the expert opinions relied on by EPA, relatively objective and reliable statistical methods reveal ***no causal relation between past ozone reductions and past improvements in public health***, such as reductions in asthma-related hospitalizations (Moore et al., 2012) or reductions in all-cause or in cardiovascular mortality rates (Cox and Popken, 2015) or reductions in asthma-related emergency room use (Health Effects Institute, 2010). To be sure, there is indeed a positive statistical *association* between past levels of ozone and past mortality rates, with both declining over time in many locations. What is missing is any evidence that the association is causal. To the contrary, mortality rates declined just as quickly and just as much

between 2000 and 2010 in counties where ozone level increased as in counties where it decreased, vividly illustrating the real-world irrelevance of increases or decreases in ambient ozone levels for public health. Similar findings hold for short-run effects as well. For example, 20%-30% reductions in ozone concentrations, far larger than those now being proposed, have been *associated* with large (42%) reductions in asthma acute care events, but were subsequently found to have *caused* no detectable reductions in such events or in emergency department visits for respiratory or cardiovascular health outcomes in either adults or children (Health Effects Institute, 2010). (The association turned out to be explained by seasonal effects, rather than causal impacts of ozone on asthma.)

In summary, plentiful data on ozone levels and public health at the individual county or city level in recent decades make it possible to directly examine how and whether changes in ozone cause any detectable changes in public health. They do not. Modern methods of causal analysis make reliance on expert judgments and inaccurate predictive models unnecessary. When such unreliable methods are not used, EPA's claim that further reducing ozone will cause substantial public health benefits can no longer be sustained.

4. Conclusions and Recommendations: Doing better

EPA's health effects risk assessment (HERA) for ozone does not emphasize or explain the absence of any detectable causal impact of past ozone reductions on public health. Instead, it focuses on predicting substantial future human health benefits from future reductions in ozone, in part using new and admittedly inaccurate models for which, in EPA's words, "We are unable to properly estimate the true sensitivities or quantitatively assess the uncertainty." Policy makers

and the public interest would be better served by abandoning such models, along with other unreliable methods such as the judgments of selected experts, and instead insisting on a more rigorous, reliable and scientific approach to predicting human health effects of the Proposed Ozone Rule. This can easily be done by applying objective statistical methods of causal analysis to available data. Such an improved approach might start by explaining why past substantial reductions in ozone have not produced the public health effects that EPA predicts from its proposed reductions in ozone levels, and by modifying its health risk assessment for ozone to be more consistent with past data.

Whether environmental regulations in the United States should be based on the judgments of selected experts or on independently reproducible and verifiable statistical analyses of causation, when the two conflict, raises important questions about what relation we want between science and policy-making. In principle, expert judgments would not conflict with relatively reliable and objective statistical methods for causal analysis, but would be informed by them. In practice, EPA's insistence that further reducing ozone standards is necessary to protect and improve human health contrasts with decades of experience revealing that no such benefits actually occur. What to do next will say much about what role if any, we collectively want science and objective causal analysis of data to play in shaping environmental and public health risk management policies and regulations.

Thank you for your attention.

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