United States Senate
WASHINGTON, DC 20510

June 30, 2011

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue NW
Washington, D.C. 20460

Dear Administrator Jackson:

On June 15, 2011, you testified before the Senate Environment and Public Works Committee. At that hearing, we emphasized the serious nature of the scientific concerns raised by the National Academy of Sciences (NAS) in its recent critique of EPA's draft risk assessment for formaldehyde, *Review of the Environmental Protection Agency's Draft IRIS Assessment of Formaldehyde* ("NAS Formaldehyde Report"). The NAS report highlights that for over a decade EPA has continued to err in its risk assessments from issues such as a lack of information regarding study selection criteria, inconsistent methods for evaluating the strengths and weaknesses of studies, and the lack of a clear framework for evaluating the weight of evidence for establishing what causes adverse health effects. These problems have persisted despite numerous attempts by the NAS, National Research Council (NRC) and members of Congress to compel change.

From your testimony, you stated that EPA has been upgrading its science practices and that you have charged Dr. Anastas to respond to any unmet recommendations from the NAS. We look forward to seeing EPA's robust response to the NAS critique of the Formaldehyde Assessment and how EPA has fully implemented the recommendations contained in Chapter 7 of the report. We also look forward to your response to our May 10, letter which we have been awaiting for nearly two months.

While EPA may be attempting to make progress in correcting science deficiencies in the area of IRIS assessments, there remains fundamental problems to assuring high-quality, unbiased scientific results within other EPA programs. For example, the same scientific defects noted in the Formaldehyde Assessment are also present in EPA's evaluations of the science used to establish and revise National Ambient Air Quality Standards (NAAQS), including the ongoing reconsideration of the ozone standard, scheduled to be finalized in July. This should not come as a surprise. EPA's National Center for Environmental Assessment within the Office of Research and Development, performs this scientific work for both EPA's IRIS and NAAQS programs. One should therefore expect that scientific defects observed in EPA's IRIS program would also be observed in EPA's NAAQS program.

These scientific defects include, but are not limited to:

- Current methods for selecting studies appear to systematically exclude or discount well conducted, peer reviewed studies that show no adverse health effects from air pollution at or below current air quality standards.

- Current methods for evaluating studies appear to discount recent studies that report no association between ozone and cardiovascular morbidity and focus on a subset of increasingly outdated studies that support a positive relationship. Given the reported similarity between the designs of these studies, EPA's evaluation methods appear driven in large part by whether the studies show a positive association.

- Current methods for evaluating studies allow for conflicting interpretations of the same study based on which NAAQS standard is being reviewed and the study's results for that pollutant. EPA rejected the methods in one study as "notoriously unreliable" when it reported a negative result for one NAAQS pollutant, but placed a very high reliance on the same methods in the same study when it reported a positive result for another NAAQS pollutant. Apparently, a positive result is a sufficient basis to disregard the "notoriously unreliable" methodology.

- Current methods for weighing evidence allow EPA to discount multiple no-effect studies and rely instead on single studies showing an effect. One can only conclude that no-effect studies carry no weight under EPA's current weight of evidence approach, regardless of the number and quality of the no-effect studies.

- Current methods allow EPA to <u>assume</u> a causal relationship between PM exposure and mortality, without first establishing a causal framework, potentially leading to a subjective view of the overall data.

- Current methods allow EPA to calculate benefits from reducing $PM_{2.5}$ and ozone at levels far below exposure levels that CASAC has said are safe with an ample margin of safety. In fact, the majority of the calculated benefits for the $PM_{2.5}$ and ozone NAAQS are below the levels considered safe.

- Current practices do not provide for a comprehensive analysis of uncertainty and variability as a way to make risk assessments more useful for decision makers. In some cases, an error in one assumption can virtually eliminate all claimed benefits for $PM_{2.5}$ and ozone reductions.

EPA is scheduled to make a final decision regarding the ozone standard by the end of July. By EPA's own projections, this one rule could cost American manufacturers almost one trillion dollars over the next decade – the most expensive regulation ever imposed by EPA.

Given EPA's regulatory schedule and the significance of these issues to understand the potential value of these rules to the American public, I request that you respond to the attached questions by no later than July 8, 2011.

Questions Regarding EPA Scientific Methods

Study Selection and Evaluation Criteria

One of the key concerns raised by the NAS is EPA's consistent failure to document how studies are selected for review, including EPA's criteria for selecting individual studies upon which to base either qualitative (empirical) or quantitative judgments regarding the risk from exposure:

> The committee did not find sufficient documentation of methods and criteria for identifying the epidemiologic evidence to be reviewed, for evaluating individual studies, for assessing weight of evidence, for selecting individual studies for derivation of toxicity and risk estimates, or for characterizing uncertainty and variability. (p19)

These same scientific concerns over study selection are also applicable to EPA's review and selection of epidemiology studies in establishing and revising NAAQS. A review of EPA's NAAQS decisions confirms that EPA's approach to selecting studies for purposes of assessing risk and developing standards appears to systematically exclude or discount well conducted, peer reviewed studies that show no adverse health effects from air pollution at or below current air quality standards. Please address the following questions.

- In assessing the evidence on the health effects of ozone, EPA has discounted or ignored studies reporting no significant association between current levels of ozone and asthma exacerbation. Examples include studies by Schildcrout et al. (2006) and Connor et al. (2008). Both of these studies were funded by EPA and include more accurate measurements of pulmonary function than the studies EPA ultimately selected for assessing the relationship between ozone and asthma. Despite EPA funding and the Agency's likely participation and approval of their design, these studies appear to play no meaningful role in EPA's qualitative or quantitative assessment of the effect of ozone exposure on asthma. While the Schildcrout et al. study was included in EPA's 2008 review, EPA had the opportunity to review the Connor et al study in its provisional assessment, but chose not to do so.
    - Why did EPA fund these studies?
    - Did EPA review and endorse the design of the studies prior to funding?
    - What are the relative strengths and weaknesses of these two studies compared to the other studies EPA relied upon its assessment of risk?
    - What role did these studies play in EPA's qualitative or quantitative assessment of the risk of ozone exposure on asthma exacerbation?
    - If EPA discounted or ignored these studies, what were the reasons for these decisions?

- How would EPA's qualitative or quantitative conclusions change if these studies had not been conducted?

- Similarly, EPA also appears to have discounted numerous recent studies that report no association between ozone and cardiovascular morbidity (Szyskowica 2009, Symons, 2006, Villeneouve, 2006, Wellenius 2005, Tolbert, 2007, Zanobettei and Schwartz 2006) to focus on a subset of increasingly outdated studies that support a positive relationship (Wong et al. (1999a,b), Wong et al. (1999b), and Ballester et al. (2006)). Given the reported similarity between the study designs of those studies showing a relationship and the studies that do not, EPA's selection appears driven in large part by whether the studies show a positive association.

  - Please explain what role the aforementioned studies that showed no effect played in EPA's qualitative or quantitative assessment that ozone causes cardiovascular morbidity?

  - Are the study designs of the no-association studies similar to the ones EPA relied upon? Are there any significant differences between the studies?

  - Did EPA effectively treat the no-effect studies for purposes of its assessment as if they had not been done?

- EPA's apparent biased approach to selecting studies for purposes of assessing risk is not unique to ozone. In reviewing the science for the fine particulate matter ($PM_{2.5}$,) NAAQS, EPA also appears to have discounted or ignored studies, such as Enstrom (2005), the case control results by Beelen et al. (2008), and the 2-polltuant (SO_2 and PM) by Krewski et al. (2000), all of which fail to find an association between exposure to $PM_{2.5}$ and chronic mortality at any exposure level.

  - Please explain what role, if any, these studies played in EPA's development of the staff recommendations for $PM_{2.5}$?

  - Does EPA believe that these studies suffer from methodological weaknesses that are not reflected in other studies?

  - What role does EPA believe well conducted epidemiology studies should play in EPA's assessment of risk from national ambient air quality pollutants if the studies do no show an association?

- Perhaps the most obvious demonstration of the extent of EPA's bias against studies showing no association can be seen in the conflicting interpretations EPA has applied to the results of a single study. In reviewing the health effects evidence for exposure to nitrogen dioxide (NO_2), EPA concluded in the 2007 Integrated Scientific Assessment (ISA) that the numerous studies reporting no association between NO_2 and self-reported peak expiratory flow (PEF) were not relevant because studies relying on PEF as a pulmonary lung function measurement are "notoriously unreliable " (EPA, 2007 ISA p 3-16). One of the studies EPA discounted

due to its reliance on PEF in the NO_2 2007 ISA is Mortimer et al. (2004). However, when evaluating the same study for assessing risks from ozone exposure as part of the 2008 ozone NAAQS review and Reconsideration (using the same PEF pulmonary lung function measurement), EPA places very high reliance on this study without even attempting to explain the inconsistency (EPA 2010a pg. 2951). Apparently, positive results constitute a sufficient basis to disregard the "notoriously unreliable" nature of the self-reported PEF measurement.

- Please explain why self-reported PEF measurements can be reliable.
- What are the reasons for this inconsistent interpretation of studies using this same measure of pulmonary function?
- Is there any difference in how the measurements were taken with regard to ozone versus NO_2?
- Was the NO_2 scientific review conducted by the same staff as the ozone review?
- Does EPA have criteria that would clearly detail how EPA should evaluate studies with PEF measurements compared to those that do not rely on such measurements?

- Unfortunately, EPA's willingness to interpret a study differently based on its results is not an isolated event. For example, Schildcrout et al. (2006) reports asthma symptoms for NO_2 but not ozone. In the NO_2 NAAQS review, EPA states that this study provides strong evidence for the health effects of NO_2 (EPA 2010b pg 6485) whereas in the 2008 Ozone NAAQS review and Reconsideration, EPA notes many reasons why the results of the study should be ignored, including the fact that only 12 children per day were evaluated and that the authors did not clearly define the severity of asthma in the study subjects (EPA 2009, pg. 14).

  - Please explain the reasons for the inconsistency in interpreting the results of this same study.

  - On what basis does your Agency believe that the EPA cited deficits in the ozone review do not apply to the NO_2 conclusions?

  - Did EPA rely on the study in the context of the NO_2 review simply because the study provided a positive result?

- EPA's apparent bias in selecting epidemiology studies and even in interpreting the same study differently based on its results for different pollutants appears to be a clear bias toward interpreting scientific data based not on merit but on the desired results. As reported by the NAS in the context of formaldehyde, it also confirms that EPA does not document and may not have clear methods and criteria for selecting and critically evaluating individual epidemiological studies. This weakness clearly extends to the NAAQS assessment process and EPA's selection and evaluation of epidemiology studies.

  - Does EPA adhere to a clear set of criteria in determining which studies should be relied upon in assessing potential risk? If not, why not?

- Does EPA believe that its mandate to protect public health allows the Agency to disregard or ignore well-conducted studies that fail to show an effect?

- Does EPA believe that a policy of consistently disregarding studies misinforms the public and leads to inflated and highly uncertain estimates of public health risks?

- Does EPA believe that it fairly educates the public on the extent and potential implication of its bias in communicating risks and benefits?

Weight of Evidence

In the context of the formaldehyde review, the NAS review also notes that EPA does not contain documentation on the methods and criteria for "assessing the weight of evidence" (3-4) – how EPA should weigh multiple studies or even different types of evidence in reaching a conclusion. This problem is also very apparent in the EPA NAAQS science reviews where EPA has not only discounted equal numbers of studies showing no association, but has at times, discounted multiple no-effect studies to rely instead on single studies showing an effect.

For example, in the ongoing review of the science of the 2013 ozone NAAQS, EPA is using the results of a single study (Jerrett et al. 2009) to draw the conclusion that ozone causes chronic mortality despite the existence of nine other major studies that report no association. No associations were reported for cardio-pulmonary mortality in two updates of the Harvard Six Cities Study (Dockery *et al.* ,1993 and Krewski et al. 2000), three updates of the American Cancer Society (ACS) study (Pope *et al.* 2002, 2004 and Krewski et al. 2000), three updates of the Adventist Health Study of Smog (AHSMOG) (Abbey *et al.* 1999, Chen et al. 2005, and Beesen et al. 1998), the latest update of the Veterans Affairs cohort study (Lipfert et al. 2006), and an Australian cohort study by Wang et al. (2009). EPA's approach of discounting the results of the Harvard Six Cities Study by Dockery et al. is especially problematic given that EPA places significant weight on this very same study when assessing the risks of particulate matter. In addition to being another clear example of EPA's willingness to interpret the same study differently based on whether the results support a positive relationship, it also shows that no-association studies carry no weight under EPA's current weight of evidence approach, regardless of the number and quality of the no-association studies and EPA's reliance on the same studies in other assessments.

- Please describe EPA's current weight of evidence approach to evaluating health evidence and why the above nine cited negative studies were discounted in favor of the single study by Jerrett et al. 2009.

- How does EPA factor a no-association result from a well conducted study in its assessment of public health risk? Does EPA place any weight on these studies?

- Please explain why EPA places high reliance on the Harvard Six Cities Study for PM but discounts the results of this same study in the case of ozone?

- Can EPA cite any examples where no- association studies were given weight in EPA's decisions to establish a NAAQS? Please explain.

Causality

One of the NAS's most striking criticisms of EPA's draft formaldehyde assessment concerns EPA's conclusions regarding the risk from exposure to formaldehyde. According to the NAS, the "conclusions appear to be based on a subjective view of the overall data, and the absence of a causal framework". (p83). Given that EPA's main criteria for selecting and interpreting epidemiology studies in the NAAQS review process appears to be whether the results show an association, this same criticism may also be applicable to EPA's conclusions that ozone and fine particulate matter ($PM_{2.5}$) cause premature mortality. EPA's main criteria for placing weight on a study should not be how large the estimated result is from the study, or if the results are positive or negative, but rather how well designed the study is and how well it explains the observed data.

Concerns over the legitimacy of EPA's conclusion that exposure to $PM_{2.5}$ causes mortality is a not a new concern. In 1998, the National Research Council in its report on "Research Priorities for Airborne Particulate Matter" questioning whether the observed statistical associations are a result of model selection (p 90). In addition to ignoring well-conducted studies that show no association (such as Enstrom 2005 and Beelen et al. 2008), EPA has also negated suggested methodological advances in the interpretation of epidemiology results. More recent research evaluating $PM_{2.5}$ mortality allows for a more even handed, objective consideration of multiple models and results that present a good fit of the observed data (rather than the highest positive result). This research does not confirm EPA's conclusions regarding causality. In fact the authors conclude that it "is unclear whether any of the pollutants has an appreciate effect on mortality." (Koop and Tole, 2004). Similarly, EPA also ignores the significant methodological issues raised most recently by Janes et al. (2007). In this study, the authors evaluated local or county results separately from results at the national scale due to the fact any national association between PM2.5 and mortality is more likely to be confounded by other factors. When the association between PM2.5 and mortality at the national scale is set aside, there is little evidence of an association between exposure to PM2.5 and mortality. This led Janes et al to the conclude that we should be very careful about interpreting long-term trends of improving air quality and trends in reduced mortality as causal.

EPA's assumption that exposure to fine particulate matter causes mortality is critical. In Table 5-11 of EPA's March 2011 Benefits Report, *The Benefits and Costs of the Clean Air Act from 1990 to 2020,* EPA states that it "assumes a causal relationship between PM exposure and premature mortality", and that "If the PM/mortality relationship is not causal, it would lead to a significant overestimation of net benefits". In fact, over $1.9 trillion of the $2 trillion in benefits cited by EPA in the Benefits Report would vanish if this one key assumption proves incorrect. Nowhere does EPA expand on this significant uncertainty to explain to policymakers the consequences of this critical assumption being incorrect. Given the highly dependent nature of EPA's overall benefit analysis on this one assumption, one could question whether EPA is conducting an objective analysis of the data.

- Has EPA reviewed the analysis conducted by Koop and Tole, 2004?

- Why does EPA reject their proposal to conduct model averaging to determine the best fit the observed data?

- Similarly, did EPA review Janes et al. 2007? Does EPA believe the authors have a legitimate concern regarding the greater role of confounders at the national scale compared to local or county air quality results?

- Does EPA believe the lack of an association at the country or local level undermines the case for causality?

Risks from Pristine Air

EPA's calculation of health benefits includes not only to $PM_{2.5}$ and ozone exposures at levels above current standards, but also to levels far below ambient levels that EPA and CASAC consider to be protective of human health with an adequate margin of safety. In fact, the majority of EPA's calculated benefits from reducing $PM_{2.5}$ and ozone are from exposure levels far below levels that EPA and CASAC have said are safe with an ample margin of safety.

Buried on page 6c-5 of EPA' 2008 Ozone Regulatory Impact Statement (RIA) is a table that shows the distribution of claimed $PM_{2.5}$ "co-benefits" based on exposure level. Over 67 percent of the claimed $PM_{2.5}$ co-benefits occur at exposure levels at or below 10 micrograms per cubic meter, an exposure level that falls far below both the current $PM_{2.5}$ standard of 15 micrograms per cubic meter and below the recently recommended CASAC range for revising the standard. EPA further notes in this table that the benefit estimate becomes more uncertain at lower $PM_{2.5}$ concentrations. In light of this table, please answer the following questions:

What percent of the $PM_{2.5}$ mortality and morbidity estimates included in the March 2011 Benefits Report is based on reductions in exposure levels that occur below the current $PM_{2.5}$ annual standards? Please provide a table showing the percent of benefits of at and below 10, 11, 12, 13, and 14 micrograms per cubic meter.

Why did EPA indicate in table 6c-2 of the 2008 Ozone RIA that the benefits become less certain at lower exposure concentrations? Please describe how EPA accounted for this increased uncertainty in their quantitative estimate of benefits. Did EPA consider the quantitative impact of these factors in estimating the $PM_{2.5}$ mortality benefits in the March 2011 Benefits Report?

- Does EPA believe that the risk from exposure to $PM_{2.5}$ at levels between 1 and 10 ug/m^3 of $PM_{2.5}$ is equivalent to risks from exposures above 15 ug/m^3? If so, how can EPA set a NAAQS that is protective of public health above10 ug/m^3 if EPA believes that close to two-thirds of the public's exposure and risk comes from exposure to $PM_{2.5}$ at or below this level?

Comprehensive Uncertainty Analysis/Uncertainties Not Quantified

As discussed above, the NAS Formaldehyde report also notes that your Agency appears to have largely ignored repeated recommendations from the National Research Council, beginning as far back as 1983, to conduct more comprehensive analysis of uncertainty and variability as way to make risk assessments more useful for decision makers: "Those and other reports have consistently highlighted the necessity for comprehensive assessment of evidence and characterization of uncertainty and variability, and the Silver Book emphasizes assessment of uncertainty and variability appropriate to the decision to be made." (p113).

In estimating the risk from exposure to ozone and $PM_{2.5}$, EPA either fails or significantly underestimates the importance of a number of key uncertainties. These include: 1) whether or not the various health effects, in particular PM and ozone mortality, are causal; 2) uncertainties in the statistical modeled used to derive Concentration Response Functions (CRF) for key health effects (e.g. PM chronic mortality); 3) the uncertainty associated with extrapolating health effects far below the range of the underlying studies and to background levels.

Changing any *one* of EPA's key assumptions regarding causality, concentration response function (CRF), and extrapolation of health effects far below the level of the standards could significantly lower EPA's estimate of the risk and the resulting benefit estimates from reducing exposure to PM2.5. Changing more than one of the estimates could virtually eliminate most if not all of the claimed benefits from regulation. This Congress, policymakers, and the American public should understand how dependent EPA's benefit claims are to a number of highly questionable assumptions.

Why has EPA repeatedly failed to conduct a comprehensive assessment of the uncertainties given the significance of these rules?

- What steps is EPA taking to address the NAS and NRC recommendations on conducting a comprehensive uncertainty analysis?

- Why has EPA failed to conduct a quantitative uncertainty analysis that would allow policy makers and the public to see an integrated assessment of the impact of these uncertainties on EPA's estimates of public health risks and benefits rather than depending on a qualitative discussion?

Given the timeframe with which we are dealing the need for your prompt response to these important questions of scientific integrity cannot be understated. The economy and many of our fellow Americans are suffering. To further perpetuate the problems of high unemployment and poverty without strong scientific and economic support for EPA's calculated efforts would be unwise.

Sincerely,

David Vitter
United States Senator

James Inhofe
United States Senator

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