U.S. House of Representatives
Committee on Science, Space, and Technology
Subcommittee on Energy and Environment

Questions for the Record – Responses by Dr. Michael Honeycutt
July 6, 2012

Hearing Title: EPA's Impact on Jobs and Energy Affordability: Understanding the Real Costs and Benefits of Environmental Regulations

1. According to OIRA's Draft Report to Congress on the Benefits and Costs of Federal Regulations and Unfunded Mandates on State, Local, and Tribal Entities, the benefits from EPA air quality regulations that affect particulate matter represent almost 80 percent of <u>all</u> benefits from <u>all</u> regulations across the <u>entire</u> federal government. Do you find that claim to be credible?

I do not find this claim to be credible. This conclusion is based on the monetization of mortality risks attributed to $PM_{2.5}$ (fine particulate matter). The true (if any) relationship between $PM_{2.5}$ and premature mortality is obscured by:

(1) the choice of studies that support the proposed relationship between $PM_{2.5}$ and mortality (and exclusion of contradictory data). EPA relied on two studies that showed a statistically-significant association between $PM_{2.5}$ and premature mortality. If they had used any of the several equally well- or better-conducted studies that did not show a statistically significant association between $PM_{2.5}$ and premature mortality[1], then the monetized benefits would have been $0.

[1] Krewski *et al*.2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality, Part II: Sensitivity Analysis. Health Effects Institute. P129-293. See models that include SO2
McDonnell *et al.* 2002. Relationships of Mortality with the Fine and Coarse Fractions of Long-Term Ambient PM10 Concentrations in Nonsmokers. Journal of Exposure Analysis and Environmental Epidemiology. 10(5):427-36.
Koop and Tole. 2004. An Investigation of Thresholds in Air Pollution-Mortality Effects. Environmental Modeling and Software. 21(12):1662-1673.
Chen *et al.* 2005.The association between fatal coronary heart disease and ambient particulate air pollution: Are females at greater risk? Environmental Health Perspectives. 113(12):1723-1729. See data for males
Enstrom. 2005. Fine Particle Air Pollution and Total Mortality Among Elderly Californians, 1973-2002. Inhalation Toxicology. 17(14):803-16.
Lipfert *et al.* 2006. $PM_{2.5}$ Constituents and Related Air Quality Variables as Predictors of Survival in a Cohort of U.S. Military Veterans. Inhalation Toxicology. 18:643-657.
Franklin *et al.*. 2007. Association Between $PM_{2.5}$ and All-Cause and Specific-Cause Mortality in 27 U.S. Communities. Journal of Exposure Science and Environmental Epidemiology. 17(3):279-87. see lag 0 data.
Zeger *et al*.2008. Mortality in the Medicare Population and Chronic Exposure to Fine Particulate Air Pollution in Urban Centers (2000-2005). Environmental Health Perspectives. 116(12):1614-9. see data for Western U.S.
Krewski *et al*.2009. Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality. Research Report from the Health Effects Institute. 140:5-114. see 1972-2000 data.
Klemm *et al*.2011. The Impact of Frequency and Duration of Air Quality Monitoring: Atlanta, GA, Data Modeling of Air Pollution and Mortality. 61:1281-1291.
Tony Cox. 2011. Hormesis for Fine Particulate Matter ($PM_{2.5}$). Dose-Response. Pre-Press Article.

(2) the assumption of a no-threshold model which attributes risk to background levels of $PM_{2.5}$. This practice inflates the benefits because it calculates risk from $PM_{2.5}$ levels that are naturally-occurring and too small to be controlled by regulations.

(3) the application of a Weibull distribution to possible concentration-response functions leading to the exclusion of data that does not support the assumption of a cause-and-effect relationship between $PM_{2.5}$ and premature mortality. Again, this artificially inflates the benefits because it predicts premature mortality (and therefore monetized benefits) in parts of the country where the actual data shows premature mortality due to $PM_{2.5}$ exposure doesn't occur.

Based on the uncertainties regarding the limitations of observational epidemiology studies as well as the methodological issues noted above, the benefits estimated by EPA for rulemakings under the Clean Air Act are overstated.

2. The Subcommittee received a letter from the American Lung Association that stated that, in the case of the recently-finalized Mercury and Air Toxic Standards, "For every dollar spent to reduce air toxics pollution, Americans receive $3-9 in health benefits." Do you agree with this characterization?

Not at all. This statement by the American Lung Association is taken from the Regulatory Impact Analysis for the Mercury and Air Toxic Standards (MATS) rule, which relies almost exclusively upon $PM_{2.5}$ co-benefits (see response to question 1 above). The EPA's economic analysis misrepresented the actual benefits of the rule. Benefits should be based on direct health benefits associated with reductions of the HAPs rather than including co-benefits associated with emission reductions of non-HAP pollutants. More than 90% of the represented health benefits are based on particulate matter reductions and not the HAPs that are the focus of the rule. Particulate matter is not a HAP and is regulated under other EPA air quality programs. If EPA confined its analysis only to the specific HAPs that pose a hazard to public health, any health benefits would be insubstantial compared to cost of the regulation.

EPA was not able to quantify health benefits for reductions of actual HAPS regulated by the MATS rule except for mercury. EPA's quantified health benefits of $4 to $6 million reflecting mercury reductions are questionable, because that amount is based on the assumed economic value of a total of 511 intelligence quotient (IQ) points. EPA multiplied the average loss of 0.00209 IQ points per prenatally exposed child by 244,268 children assumed to be exposed to mercury via their mothers' consumption of freshwater fish[2]. This is akin to requiring 10 vehicles to reduce their speed by five mph per vehicle and then saying the resulting total decrease in speed is 50 mph. An IQ reduction of 0.00209 points cannot be measured. Also, EPA assumed no lag time in the response of methyl mercury levels in fish due to MATS, and if a lag was (correctly) assumed monetized benefits would be significantly lower.

[2] Please refer to additional testimony given 10/4/2011 before the Subcommittee on Energy and Environment - "Quality Science for Quality Air": http://science.house.gov/sites/republicans.science.house.gov/files/documents /hearings/100411_Honeycutt.pdf

3. What did you mean when you said that there is legal guidance for establishing causal relations and that relative risks less than 2.0 should not be considered? How does this affect EPA's association between particulate matter and mortality?

There is scientific as well as legal precedence indicating that relative risks below 2.0 should not be considered to support a hypothesized relationship (Federal Judicial Center Reference Manual on Scientific Evidence Second Edition (2000) and NCI/IARC/WHO[3],[4]). This is because relative risks less than 2.0 can often be explained by confounding variables, i.e. factors that were not considered, but that are responsible for the observed effect. For example, cholesterol levels were not measured in the Pope *et al.* 2002 study but might explain the observed cardiovascular disease mortality rates. The relative risks for $PM_{2.5}$ and premature death reported to date are considerably lower than 2.0. For the two studies most often cited by the EPA, the relative risks are 1.06 (Pope *et al.* 2002[5]) and 1.16 (Laden *et al.* 2006[6]), and therefore may actually be due to confounding variables. In fact, a recent report indicates that confounding likely plays a significant role in the statistical findings of positive $PM_{2.5}$-mortality associations.[7]

4. Aren't all of the assumptions within EPA's regulatory analysis and decision making designed to be health-protective? Isn't that a good thing?

In recent years, the EPA has approached policy decisions with an overabundance of caution, leading to excessively conservative regulations not fully supported by the best available science. The application of this precautionary principle, without regard to the extent of population exposure or risk, conflicts with best practices of science-based risk assessment. In defense of this approach, some have argued that EPA always overestimates risks in order to provide adequate protection. While this may or may not be sound regulatory policy, systematic over-estimation of benefits renders the cost-benefit process useless. Worst-case estimation of risks (and the benefits of avoiding those risks) without any indication that they represent very unlikely or even impossible scenarios, is highly misleading to the public and to elected representatives evaluating proposed policies and regulations.

Contrary to what its advocates claim, this principle does not provide a prudent guide to developing public health measures. Harvard law professor Cass Sunstein, who currently serves as administrator of the Office of Information and Regulatory Affairs has said, *"The precautionary principle, for all its rhetorical appeal, is deeply incoherent. It is of course true that we should take*

[3] http://benchmarks.cancer.gov/2002/07/epidemiology-in-a-nutshell/ "Relative risks or odds ratios less than 2.00 are viewed with caution."
[4] WHO/IARC Breslow and Day (1980). *Statistical methods in cancer research. Vol. 1. The analysis of case control studies.* IARC Sci. Publ. No. 32, Lyon, p. 36. "Relative risks of less than 2.0 may readily reflect some unperceived bias or confounding factor, those over 5.0 are unlikely to do so."
[5] Pope CA III, RT Burnett, MJ Thun, EE Calle, D Krewski, K Ito, and GD Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association. 287:1132-1141.
[6] Laden F, J Schwartz, FE Speizer and DW Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. American Journal of Respiratory and Critical Care medicine. 173:667-672.
[7] Greven *et al.* 2011. An Approach to the Estimation of Chronic Air Pollution Effects Using Spatio-Temporal Information. Journal of the American Statistical Association. 106(494):396-406.

precautions against some speculative dangers. But there are always risks on both sides of a decision; inaction can bring danger, but so can action. Precautions, in other words, themselves create risks—and hence the principle bans what it simultaneously requires."[8]

The result of utilizing the precautionary principle is highly uncertain estimation of the benefits of Clean Air Act rules. This is because regulating pollutants without proof they are indeed harmful at relevant doses negates the accurate quantification of what harm has been prevented. Further, especially in times of resource scarcity, focusing attention and regulation on unnecessary risks can result in inadequate attention or resources to address real health effect risks or problems.

5. Why shouldn't EPA claim regulatory benefits association with incidental particulate matter reductions from non-PM rules?

In 2011, President Barack Obama issued Executive Order 13563, which states that agencies should strive to reduce regulatory requirements that are *"redundant, inconsistent, or overlapping."*

Including $PM_{2.5}$ co-benefits in multiple non-$PM_{2.5}$ rules is *redundant*. Section 109 of the Clean Air Act requires that each NAAQS be set at a level that protects public health with "an adequate margin of safety" (i.e. no additional public health improvements would be gained by tightening the standard any further). If those concentrations are safe, then it is not appropriate to calculate co-benefits for $PM_{2.5}$ below this level to justify non-$PM_{2.5}$ regulations. More broadly, co-benefits from any pollutant that is regulated as a criteria pollutant with a NAAQS that conforms to the requirements of CAA should not be included in the Regulatory Impact Analysis (RIA) of any other pollutant. Including $PM_{2.5}$ co-benefits in other RIAs not only results in double-counting of benefits, but also prevents identification of ways to reduce regulatory burdens while still meeting air quality objectives.

Baseline calculations for proposed rules are *inconsistent* with best practices. EPA has argued that it does not double-count the $PM_{2.5}$ benefits because it includes all existing regulations in the baseline of emissions for each of its RIAs for another rule; however, this is not the case for the following reasons: (1) multiple RIAs are prepared simultaneously; this creates a constant potential for double-counting; (2) review of recent RIAs released by EPA indicates that all applicable CAA-related rules are not, in fact, included in the baseline calculations for these standards (e.g. see RIAs for ozone, SO_2, and NO_2 NAAQS); and (3) the baseline calculations are based on monitored levels of $PM_{2.5}$, and it is impossible to distinguish the effect of each rule on ambient levels of $PM_{2.5}$. Furthermore, each rule seeks to lower the same observed levels of ambient $PM_{2.5}$, resulting in double counting of estimated benefits across multiple rules.

The consequences of these *overlapping* rules include lack of transparency and miscommunication with the public and policy makers. $PM_{2.5}$ co-benefits are reported as part of the total benefits in the executive summary of an RIA and also in public announcements about the proposed rule. This

[8] Cass R. Sunstein, "Throwing Precaution to the Wind: Why the 'Safe' Choice Can Be Dangerous," Boston Globe, July 13, 2008. For a more extensive critique, see Cass R. Sunstein, The Laws of Fear: Beyond the Precautionary Principle (Cambridge: Cambridge University Press, 2005).

creates confusion for audiences who often fail to realize that these total benefits are mostly due to reductions in $PM_{2.5}$. Furthermore, $PM_{2.5}$-related benefits would be more effectively and appropriately obtained through revision to the $PM_{2.5}$ NAAQS than through non-$PM_{2.5}$ rules. Moreover, reliance on $PM_{2.5}$ co-benefits undercuts the practical value of RIAs and allows EPA to avoid improvements to its methods for characterizing and quantifying health and welfare benefits for other pollutants.

6. The Texas Commission on Environmental Quality is the 2nd largest environmental agency in the world. From your experience at TCEQ, are there ways that EPA could improve its cost-benefit analysis and stakeholder outreach process?

Generally speaking, risk assessments that serve to inform cost benefit analysis should include the following steps:

- Consider all available appropriate and relevant studies, not just studies that present positive results.
- Report comprehensive weight-of-evidence based analyses, including positive and negative data.
- Perform extensive sensitivity analyses to determine how confounding affects the analysis.
- Select health endpoints based on toxicological grounds rather than on post-hoc statistical grounds.
- Focus on studies with exposure data collected for individuals instead of groups (i.e. the studies by Pope *et al.*[9] and Laden *et al.*[10] do not determine personal exposure to PM – it was assumed to be equal for all individuals within a metropolitan area).
- Use Cox proportional hazards models as the exposure-response models.
- Do not use splines in statistical models, especially smoothing splines, as they have the effect of making the data fit the model instead of choosing an appropriate model to fit the data.
- Consider including thresholds and nonlinear relationships in the exposure-response models.
- Estimate risks using best estimates of individual exposure rather than extreme characterizations of population exposures.
- Estimate risks for the general population in addition to the "most sensitive" subpopulation.
- Clearly state the assumptions made and their qualitative and quantitative consequences.

Further, the EPA should demonstrate a peer-review process that reflects transparency and commitment to representing all data, not just data that supports its policy goals. The non-profit organization, Toxicological Excellence for Risk Assessment (TERA) provides a superb

[9] Pope CA III, RT Burnett, MJ Thun, EE Calle, D Krewski, K Ito, and GD Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association. 287:1132-1141.

[10] Laden F, J Schwartz, FE Speizer and DW Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. American Journal of Respiratory and Critical Care medicine. 173:667-672.

description of peer review: *A peer review is an in-depth assessment of the assumptions, calculations, alternate interpretations, methodology, and conclusions of the document under review…peer reviewers and consultants should be selected for both independence and scientific/technical expertise…[and]include a range of perspectives on each panel, including diverse professional affiliations (e.g., academic, consulting, environmental, government, and industry). The evaluation of real or perceived bias or conflict of interest is an important consideration and for both peer review and consultation panels and every effort is made to avoid conflicts of interest and biases that would prevent a panel member from giving an independent opinion on the subject…an objective evaluation by independent experts with a variety of different viewpoints and perspectives is critical to the credibility of any peer consultation or peer review.*[11]

TCEQ agrees with this description and strives to implement these principles. In fact, when the TCEQ revises its Guidelines for Risk Assessment, a peer review is conducted by a disinterested third party. No person on the peer review panel works for or receives funds from TCEQ. It is a conflict of interest for such individuals to participate in the peer review process, although stakeholders are welcomed to participate in the public comment process. Following the public comment period, each and every comment is addressed and changes are made to the document (when justified) as a result of this process. When TCEQ disagrees with a comment, justification is provided in the response to comment document, which is made publically available along with the modified Guidelines document. The EPA should utilize an equally transparent process, free as possible of conflict of interest.

To this end, the following recommendations for improvement are suggested:

- EPA risk assessments should include all of the steps listed above with results clearly communicated in resulting policy assessments, regulatory impact analyses, and final rulemakings. Emphasis should be placed on the limitations of the available scientific literature in order to provide policy makers with accurate information. This is particularly important to avoid the appearance that proposed rules are based on policy objectives, and merely "backed up" by applicable scientific evidence.
- Members of the Science Advisory Board and Clean Air Science Advisory Committee should not include the authors of studies utilized in that specific assessment, nor should they be current recipients of EPA funding, as this represents a significant conflict of interest (see figures 1 and 2). If such individuals are to be consulted, equal weight should be given to scientists representing local and state governments as well as industry experts.
- Prior to developing a rule, EPA should solicit stakeholder input during the development phase. TCEQ has found that engagement of affected regulated entities can help avoid the need for drastic changes from proposal to final adoption of a rule.
- When soliciting public comment, EPA should respond to each comment in a substantial manner and revise technical and policy documents accordingly. A response to comments document which lists each comment (acknowledging that there may be multiple comments with very similar objectives that can reasonably be combined for this purpose) along with the agency responses should be provided with every rule.
- Place the risks associated with ambient air pollution into the context of other risks people face. This is a crucial function of public health officials, who have an obligation to present scientific data in an unbiased manner and avoid inaccurate or inflammatory language.

[11] http://www.tera.org/Peer/Description.html

7. You stated near the end of your testimony that these regulations could have negative unintended consequences. Could you explain what you mean by that?

When regulations are routinely based on extrapolated, highly uncertain risk analysis, stakeholders must distinguish between real risk and these often alarmist conclusions. The result is that other necessary tasks are postponed. Indeed, University of Texas law professor Frank Cross observes, *"The truly fatal flaw of the precautionary principle, ignored by almost all the commentators, is the unsupported presumption that an action aimed at public health protection cannot possibly have negative effects on public health."*[12]

In the experience of TCEQ, time and resources spent analyzing and responding to unnecessary regulations based of perceived (rather than real) risks detracts from other, far more urgent needs of our citizens. Activities such as the development of safe screening levels are often delayed while staff respond to policy issues that have significant consequences for the State.

The solution for an adverse health effect associated with an environmental risk factor can itself become a risk factor for other health effects[13]. Public health officials must be aware of such potential consequences when advising citizens. Take, for example the following quote from EPA Administrator Lisa Jackson: *"We are actually at the point in many areas of this country where on a hot summer day, the best advice you can give is don't go outside. Don't breathe the air. It may kill you."*[14] Not only is this hyperbole unscientific, inaccurate, and inflammatory, but this type of statement gives the public the impression that staying indoors and avoiding physical activity is preferable. In fact, indoor air quality is clearly worse than outdoor air quality (Burke *et al.* 2001 and references therein[15]) and this type of advice can lead to unintended negative consequences. For example, suggesting that citizens avoid outdoor exercise in order to prevent exacerbation of asthma symptoms can result in unintended negative consequences such as contributing to obesity.

The Policy Assessment (PA)[16] document serves to "bridge the gap" between relevant scientific information and assessments and the judgments required of the EPA administrator in determining whether and how to revise the NAAQS. In reviewing the PA that supports the newly proposed PM NAAQS, the following comment from CASAC member Dr. Robert Phalen came to our attention: *"I am struck by the limitations placed on the Staff in framing the P.A., and concerned that readers may believe that several potentially adverse secondary health consequences have been evaluated along with the direct health effects, when they have not. Thus, I recommend adding an explicit informative statement to the P.A., or the cover letter, such as: 'Due to statute, case-law, and policy decisions, it should be noted that this Policy Assessment addresses only the direct adverse health effects of PM mass fractions. Thus, secondary public health effects, such as (1) the potential health effects of compliance actions on jobs, and the availability of goods and services;(2) the potential health effects at locations that have positive (rather than negative) health associations with PM mass; and (3) the potential health effects of changes in PM mass on other air contaminants (e.g. UFP counts, and airborne acidity), are not considered. In short, the*

[12] Frank B. Cross, "Paradoxical Perils of the Precautionary Principle," Washington and Lee Law Review 53, no. 3 (1996): 860.
[13] Steve Packham, Phd, D.A.B.T. presentation titled: "Utah's Recess Guidance: Based on Air Quality." March 7, 2011.
[14] On HBO's "Real Time with Bill Maher," October 7, 2011.
[15] Burke et al. 2001. A population exposure model for particulate matter: case study results for PM2.5 in Philadelphia, PA. Journal of Exposure Analysis and Environmental Epidemiology 11:470-489.
[16] http://www.epa.gov/ttn/naaqs/standards/pm/data/20110419pmpafinal.pdf

range of potential unintended secondary adverse consequences have not been evaluated in this document. Thus the recommendations herein may, or may not, improve overall public health.'" It is disturbing that such salient and reasonable advice from a member of the CASAC panel has been disregarded by EPA in the Policy Assessment presented to the Administrator.

Looking at the issue in the most practical way, if an unnecessary regulation raises the cost of electricity such that a low-income elderly person feels they can't afford to use air conditioning during periods of intense heat they are more likely to suffer heat stroke.

8. I understand the EPA recently conducted some experiments where they exposed people to high levels of PM. What are the implications of these experiments?

A case study published in February 2012[17] describes exposure of a volunteer to Concentrated Air Particles (CAPs). This individual had a personal and family history of heart disease as well as numerous other medical issues. During the exposure, the volunteer experienced an irregular heart beat and was transported to the hospital. A Freedom of Information Act request initiated by Steve Milloy located data spanning 2010 and 2011 for 40 additional individuals exposed to CAPs[18]. Of these, 39 experienced no clinical effects and 1 experienced an elevated heart rate.

Significant concerns are raised by this information: (1) If the EPA believes $PM_{2.5}$ is lethal, is it ethical and/or legal to expose human volunteers to such high levels of $PM_{2.5}$? Indeed, Administrator Jackson testified to Congress[19] that, "*[Airborne] particulate matter causes premature death. It doesn't make you sick. It's directly causal to dying sooner than you should."* (2) The alternative interpretation is that these results invalidate the EPA's assertion that $PM_{2.5}$ causes premature mortality. In fact, EPA has not been able to articulate a mechanism whereby $PM_{2.5}$ causes mortality[20]. Moreover, Green and Armstrong conclude, *"it remains the case that no form of ambient PM—other than viruses, bacteria, and biochemical antigens—has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to U.S. ambient levels…hundreds of researchers, in the U.S. and elsewhere, have for years been experimenting with various forms of pollution-derived PM, and none has found clear evidence of significant disease or death at relevant airborne concentrations."*[21]

[17] Ghio *et al.* 2012. Supraventricular Arrhythmia after Exposure to Concentrated Ambient Air Pollution Particles. Environmental Health Perspectives. 120:275-277.
[18] http://junkscience.com/2012/04/18/epa-human-experiments-debunk-notion-of-killer-air-pollution-agency-hides-exculpatory-results/
[19] September 22, 2011. House Energy and Commerce Committee Meeting on Air Regulations.
[20] U.S. EPA. Integrated Science Assessment for Particulate Matter (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-08/139F, 2009. From Chapter 5, Possible Pathways/Modes of Action : "Additional studies will be required to clarify the biological mechanisms underlying the health effects of PM."
[21] Laura Green and Sarah Armstrong, "Particulate matter in ambient air and mortality: toxicologic perspectives," Regulatory Toxicology and Pharmacology 38 (2003) 326-335.

FIGURES

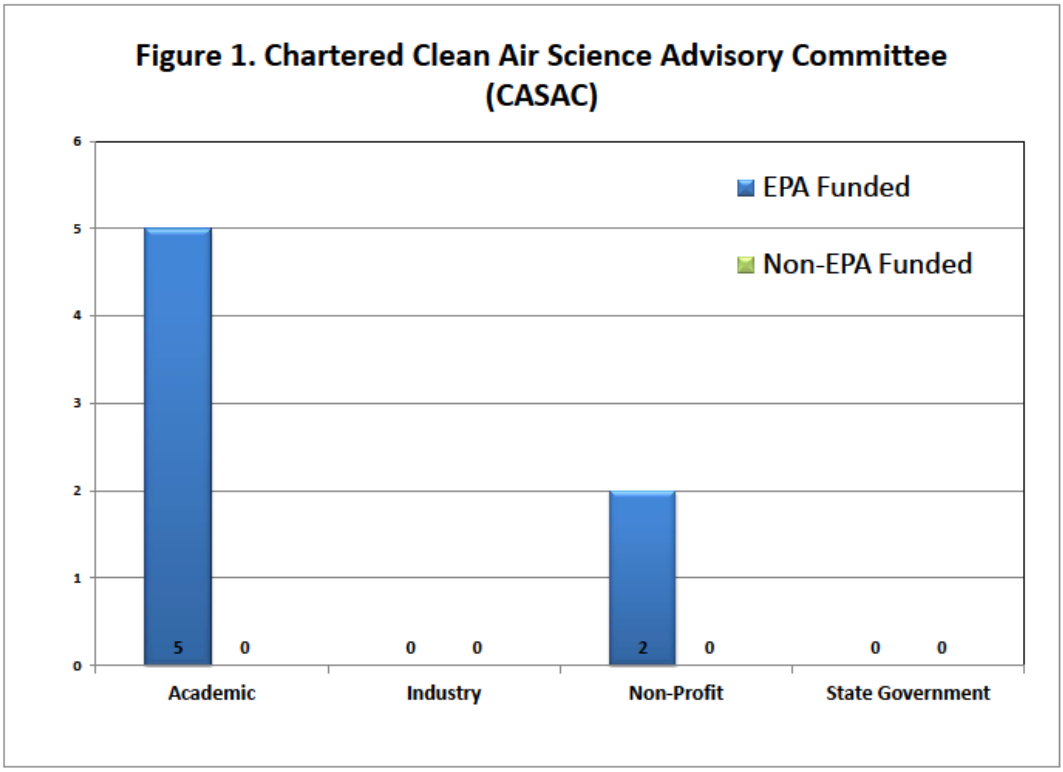
Figure 1. Chartered Clean Air Science Advisory Committee (CASAC)
EPA Funded
Non-EPA Funded
6
5
4
3
2
1
0
5
0
0
0
2
0
0
0
Academic
Industry
Non-Profit
State Government

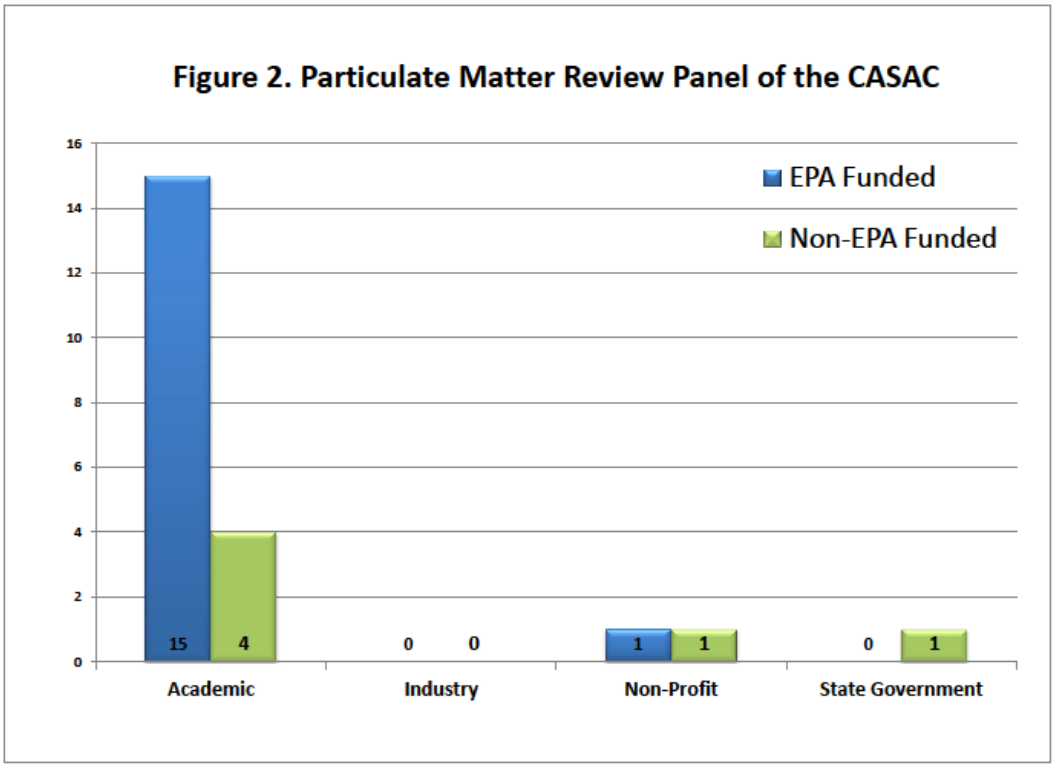
Figure 2. Particulate Matter Review Panel of the CASAC
EPA Funded
Non-EPA Funded
16
14
12
10
8
6
4
2
0
15
4
0
0
1
1
0
1
Academic
Industry
Non-Profit
State Government