Good morning, Mr. Chairman and members of the committee. I am Dr. Michael Honeycutt, director of the Toxicology Division at the Texas Commission on Environmental Quality. I have submitted more detailed written comments on the science behind the EPA's cost benefit analyses, but I'll touch on a couple of highlights now.

The EPA's cost benefit analysis is detailed in the Regulatory Impact Analysis (RIA) for each significant rule[1]. A number of Executive Orders address the requirement for and goals of cost benefit analysis. Under the Reagan administration[2] the benefits for a proposed rule had to outweigh the costs for that rule. However, with the Clinton administration[3] this language was changed substantially such that the benefits must simply justify the costs. That position is maintained in the current administration[4] along with consideration for additional factors such as equity, fairness, promotion of economic growth, and job creation.

The vast majority of the benefits that EPA calculates[5] come from the so-called "co-benefits" of reducing fine particulate matter[6], or PM, even on rules that do not directly target PM. Those estimated benefits rely heavily on two key assumptions: 1) that PM causes mortality and 2) that there is no safe level of exposure to PM. The most recent analysis[7] of the costs and benefits of the Clean Air Act concludes that for every $1 society spends complying with these regulations, $30 in benefits is obtained. However, more objective assessments of the human health benefits from cleaner air do not necessarily support the conclusion that benefits outweigh costs[8].

Keeping that in mind, I want to briefly talk about the ecological epidemiology studies that EPA is using as the primary basis for the PM benefits. These studies are exploratory studies designed to look for correlations. They are supposed to be followed up by more rigorous epidemiology and clinical studies to determine whether the correlations are real. These studies are not supposed to be used quantitatively and they certainly are not rigorous enough to set environmental policy. The assumption is that breathing PM made individuals die earlier than they would have otherwise. This type of study is notorious for unresolved issues: Were the individuals actually outside in the days prior to their death? Did they take their medications that day? Do they have other risk factors with stronger influence on life expectancy (like smoking, cholesterol, or weight)? There are a whole host of common sense questions that go unanswered in these studies. Simply put, these studies cannot tell us if PM caused these deaths or even if these people died prematurely, much less tell us what level of PM might have caused their death.

Since 2009, the EPA has assumed that there is linear relationship between PM exposure and mortality. You can see here (figure 1) data from a typical study showing that the relationship

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[1] OMB Circular A-4: A regulatory action is economically significant if it is anticipated (1) to "[h]ave an annual effect on the economy of $100 million or more" or (2) to "adversely affect in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or State, local, or tribal governments or communities."
[2] EO 12291, 1981
[3] EO 12866, 1993
[4] EO 13563, 2011
[5] EPA, March 2011. "The Benefits and Costs of the Clean Air Act from 1990 to 2020"
[6] $PM_{2.5}$
[7] EPA, March 2011. "The Benefits and Costs of the Clean Air Act from 1990 to 2020"
[8] Tony Cox. 2012. Reassessing the human health benefits from cleaner air. Risk Analysis. 32(5):816-29.

between mortality risk and PM levels is not obvious. In fact, one would be hard pressed to detect a linear association. Nevertheless, statisticians can run data through elegant models to try to find statistically significant correlations, but the output of those models is only as good as the input and, as any scientist will tell you, statistical correlation alone does not imply causation.

EPA also assumes that any exposure to PM, no matter how low, directly causes premature death. This method extrapolates risk far below the NAAQS, extending to background levels. This "no-threshold" approach is not entirely accurate, nor is it conclusively supported by the data[9,10,11,12,13,14,15,16,17]. In fact, ecological epidemiology studies are not designed to detect thresholds. Furthermore, this assumption doesn't take into account the fact that the body can handle small doses of PM. Indeed, this concept is the cornerstone of toxicology.

When the scientific data addressing the association between PM and premature death is examined in detail, it becomes obvious that these statistical associations may have very little biological significance. The increased chance of dying that is reportedly due to PM exposure is extremely small. This chance is communicated as relative risk, with a relative risk of 1.0 being non-significant. Scientific as well as legal guidance indicates that relative risks below 2.0 should not be considered to support a hypothesized relationship[18]. The relative risks for PM 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[19]) and 1.16 (Laden *et al.* 2006[20]). Many of these studies do <u>not</u> show a statistical relationship between PM and premature death[21].

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[9] McDonnell WF, N Nishino-Ishikawa, FF Petersen, LH Chen, DE Abbey. 2002. Relationships of mortality with the fine and coarse fractions of long-term ambient PM_{10} concentrations in nonsmokers. Journal of Exposure Analysis and Environmental Epidemiology. 10(5):427-36.

[10] Koop GM and LA Tole. 2004. An investigation of thresholds in air pollution-mortality effects. Environmental Modeling and Software. 21(12):1662-1673.

[11] Enstrom JE. 2005. Fine particle air pollution and total mortality among elderly Californians, 1973-2002. Inhalation Toxicology. 17(14):803-16.

[12] Lipfert FW, JD Baty, JP Miller, RE Wyzga. 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.

[13] Franklin M, A Zeka, J Schwartz. 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*

[14] Zeger SL, F Dominici, A McDermott, JM Samet. 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.*

[15] Krewski D, M Jerrett, RT Burnett, R Ma, E Hughes, Y Shi, MC Turner, CA Pope 3rd, G Thurston, EE Calle, MJ Thun, B Beckerman, P DeLuca, N Finkelstein, K Ito, DK Moore, KB Newbold, T Ramsay, Z Ross, H Shin, B Tempalski. 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*

[16] Klemm RJ, EL Thomas, RE Wyzga. 2011. The impact of frequency and duration of air quality monitoring: Atlanta, GA, data modeling of air pollution and mortality. 61:1281-1291.

[17] Tony Cox. 2011. Hormesis for fine particulate matter ($PM_{2.5}$). Dose-Response. Pre-Press Article.

[18] Federal Judicial Center Reference Manual on Scientific Evidence Second Edition (2000) p384 & fn.140.

[19] 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.

[20] 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.

[21] See references 9-17 above.

Some studies even suggest PM makes you live longer[22,23]. EPA could have chosen a number of studies just as well conducted as the Pope and Laden studies and would have determined there is no health benefit from further regulating PM.

These issues illustrate EPA's *modus operandi*. The concept of "weight of evidence" is misused to discount contradictory data. They use worst-case, often unrealistic assumptions, fail to put risks into proper perspective, and fail to disclose how uncertain the data and therefore the conclusions are. They extrapolate their risk assumptions to generate numbers of lives "saved" which unnecessarily alarms the public, and backs policy makers into a corner so that questioning the basis for EPA actions creates the illusion that you don't care about public health. Indeed these regulations can have negative unintended consequences. Our agency believes regulations to be an integral and necessary tool to protect public health and our natural resources. Likewise, our expectation is that those regulations be based on sound science, be justifiable, and that they realize true benefits.

Thank you for the opportunity to give this testimony.

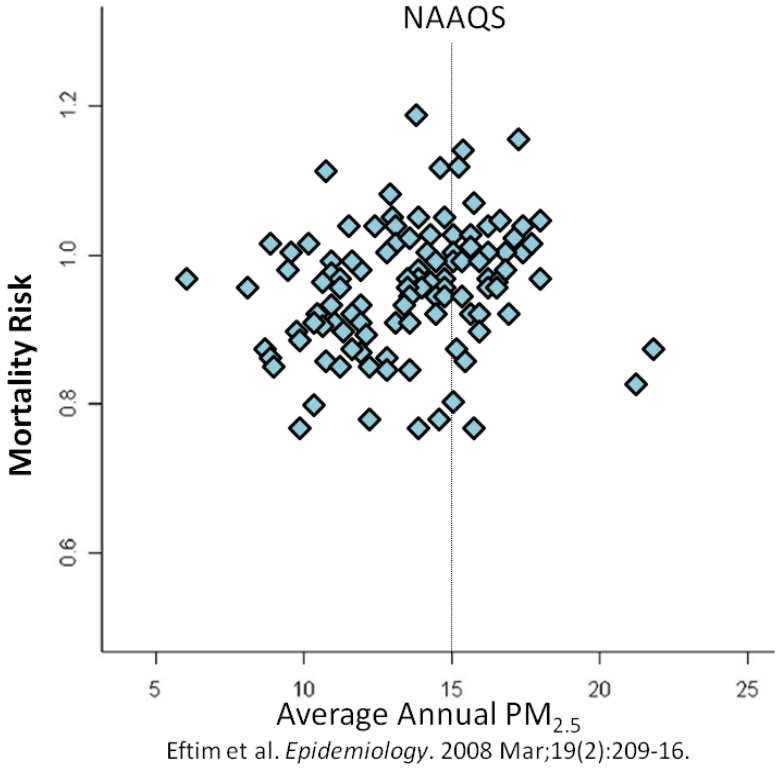

Figure 1. Reported correlation between mortality risk and average annual $PM_{2.5}$ in Eftim *et al.* 2008. Adjusted mortality relative risk estimates for Medicare enrollees are plotted against average $PM_{2.5}$ for the 110 American Cancer Society Study counties.

[22] See Franklin *et al*. 2007 data for Birmingham, Cincinnati, Dallas, Houston, Las Vegas, Los Angeles, and Riverside.
[23] Tony Cox. 2011. Hormesis for fine particulate matter ($PM_{2.5}$). Dose-Response. Pre-Press Article.