May 25, 2016 3:32 PM

Ms Anderson,
Docket No. 58-0101-1601 - Negotiated Rulemaking.
This comment letter is the second of two and is being submitted prior to the end of the public comment period. It should be noted that DEQ failed to provide a link to the Fann et al and the Hou et al studies that would enable me to analyze DEQ's stance that PM 2.5 is more harmful to human health than ozone. I received the Fann and Hou studies from Dr. Dietrich upon request on May 24, one day prior to the comment deadline. I have not reviewed the Hou et al study as yet.

Having read the Fann et al study, I feel that this study has zero relevance to the state of Idaho and completely fails to support DEQ's desire to increase the NAAQS percentage of ozone and decreas the NAAQS percentage of PM 2.5.. Having read other studies, it appears that the science behind PM 2.5 health risks is far from settled.

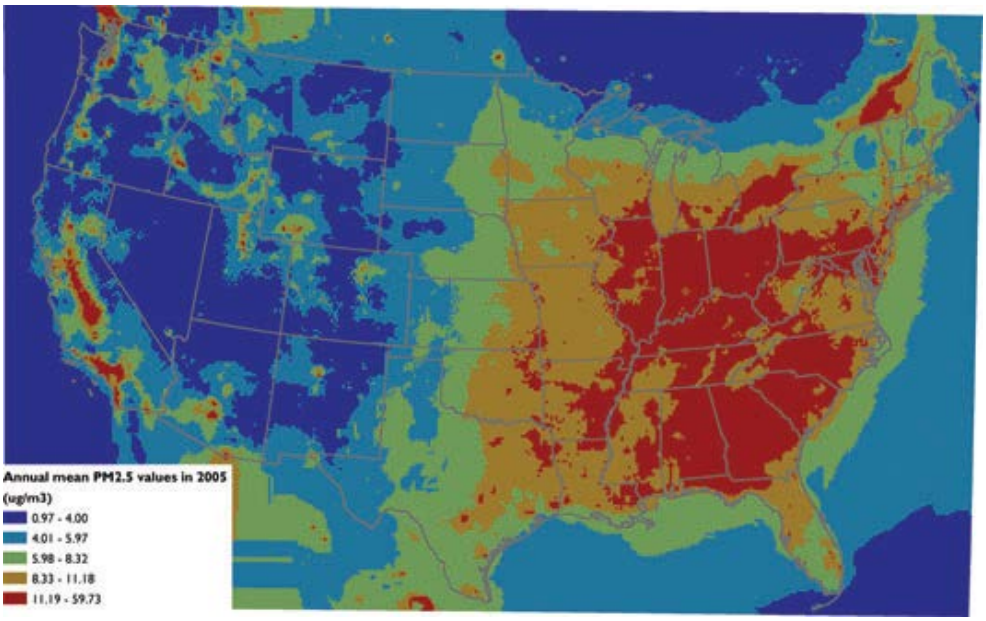

Fig. 2. Annual mean $PM_{2.5}$ levels (fused surface of 2005 modeled and 2004–2006 monitored data).

Fann's Figure2 shows clearly Idaho does not have a PM 2.5 problem. Nearly the entire state fell within about 70% of today's 12.0 µg/m3 NAAQS, and 55% of the previous 15.0 µg/m3 NAAQS (2004-2006). Looking at the 5 ranges of µg/m3, most of Idaho lies within the least 2 ranges, or below 5.98 **µg/m3.** Most of PM 2.5 exceedances are seen in the eastern half of the U.S. **Finding:** With no PM 2.5 issues in Idaho, there is no need to reduce the percentage of NAAQS for PM 2.5.

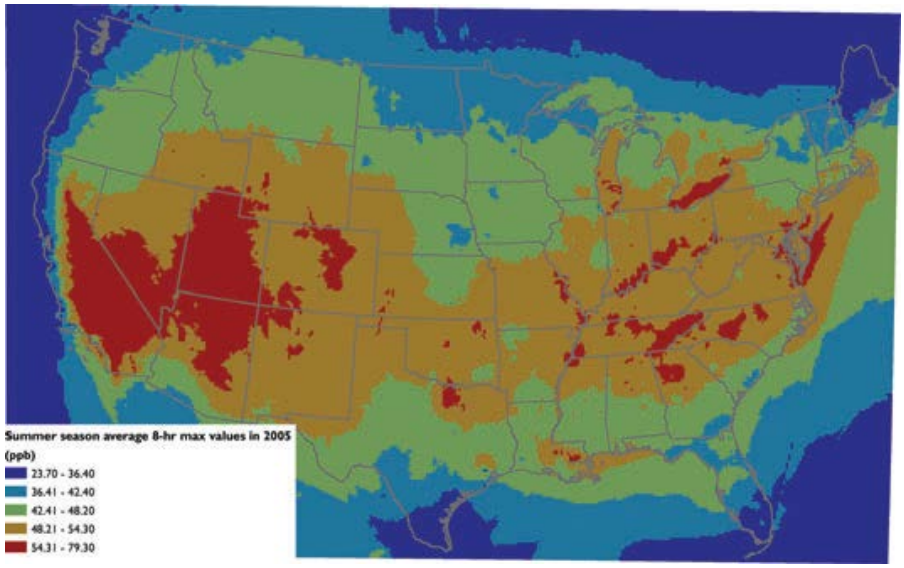

Fig. 1. Average daily 8-hour maximum summer-season ozone levels (fused surface of 2005 modeled and 2004–2006 monitored data).

Fann's Figure 1 shows that Idaho, similarly does not seem to have an ozone problem; however, it must be noted that in contrast to PM 2.5, the ozone levels in Idaho are overall much higher. The majority of the state falls within the 2nd & 3rd highest ranges. The PM 2.5 falls within the lowest two ranges. **Finding:** There is more of an ozone problem in Idaho than a PM 2.5, and perhaps may need to be more protective of human health than PM2.5.

Fann draws the PM 2.5 mortality risks from the classic, outdated Harvard Six-Cities Study (H6C) and the American Cancer Society Study (ACS) & others who "validated" these studies. There are many studies more recent than this that do not present these two studies in a good light. Fann states, "To the extent that PM2.5-related mortality is strongly influenced by particle composition, applying a *H6C-based risk coefficient nationwide may result in biased estimates of PM2.5 mortality in the west*. Conversely, applying an *ACS-based risk estimate nationwide may not characterize well the PM2.5 mortality impacts in the eastern United States*." He also states, "This study (Bell et al) is also an NMMAPS-based analysis, which applies a common methodology to all cities, suggesting that it is not subject to the same risk of publication bias as the meta-analyses, which include the results of single-city studies" with the footnote of, "Anderson and colleagues cite three reasons that *single-city studies may be more prone to publication bias*: (1) such studies frequently rely on easily available daily mortality counts, and researchers may be *less inclined to publish negative findings*; (2) the statistical modeling for time series entail a level of *subjectivity* and (3) each study can produce a large number of estimates and the *researchers may select estimates based on the direction of their effect*." To me, the 6 (single) Cities study falls into this criticism.

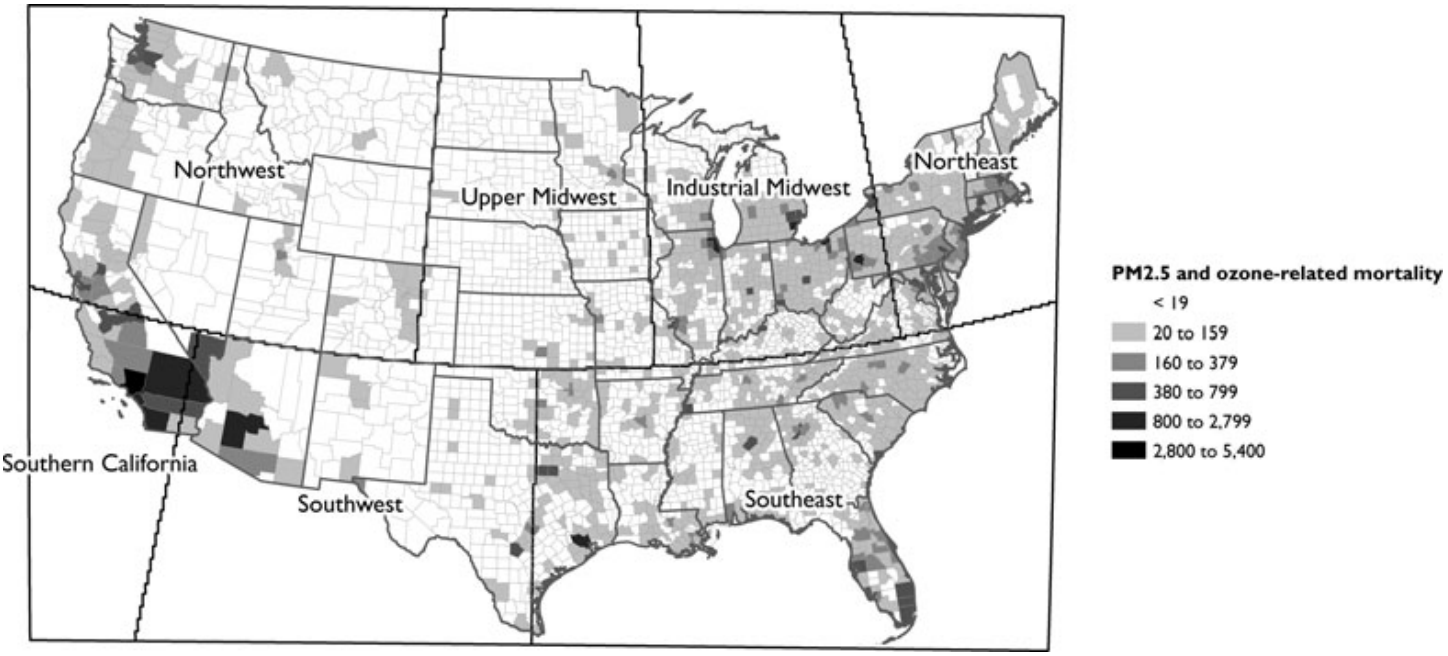

Fig. 3. PM2.5 and ozone-related excess mortalities at the county level by geographic area.

Fann's Figure 3 clearly demonstrates that all but 3-4 counties in Idaho have the lowest level of PM 2.5 and ozone-related mortality, and the 3-4 other counties exhibit the next to the lowest levels. Fann validates this when he states, "The reduction in O3 and PM2.5-related excess mortalities are not distributed evenly across the United States, and *most are concentrated in the Northeast, Southeast, and industrial Midwest*." **Finding:** Idaho has very few deaths of either cause. There is no evidence that the PM 2.5 percentage should be reduced in Idaho. Perhaps there is justification that it should be increased to the same 90% proposed increase for ozone.

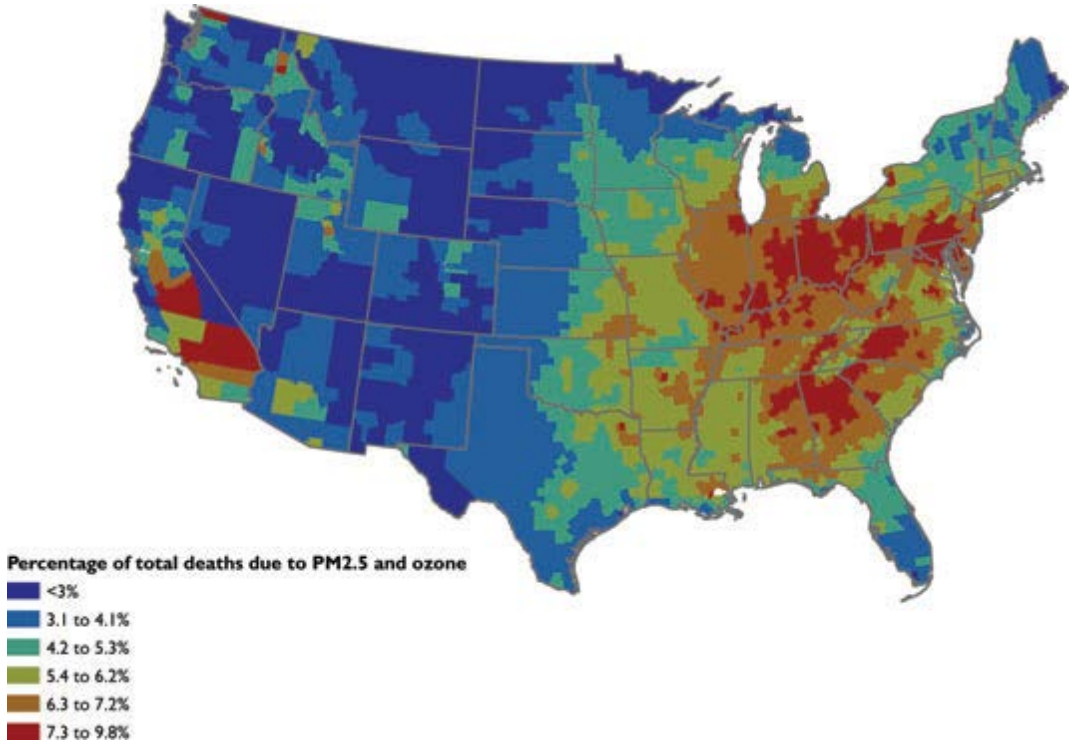

Fig. 6. Percentage of premature deaths attributable to 2005 PM2.5 and ozone air quality by U.S. county.

Fann's Figure 6 shows again that most of his paper is relative to the eastern U.S., and certainly not Idaho. Although there is county representation at most percentage ranges, approximately half of Idaho sees less than 4.1% deaths attibutable to PM 2.5 or ozone and the vast majority of the states sees less than 5.3%. **Finding:** Idaho has far less PM 2.5 and ozone premature deaths than much of the nation. There is no evidence to substantiating the reduction in the PM 2.5 percentage.

Fann estimates that PM 2.5 and ozone-related mortalies between those 65-99 accounted for 7% of the total life years lost in 2005. This is based on estimated PM 2.5-related deaths and estimated ozone-related deaths and is subtracted from the U.S. Census all-cause death data for that year. This means that 93% of these aging people died from other causes. It was mentioned that, "For both ozone and PM2.5, the percentage appears to decline modestly for older populations, suggesting that older populations may live in areas with lower modeled concentrations." This implies that Idaho's elderly population is less at risk.

Fann states, "Finally, using a standard life table, we quantify the change in life expectancy at birth and by 5- year age increment resulting from the elimination of PM2.5 and ozone-related mortality risk (Table SVI). Among populations at birth, we *estimate a change in life expectancy of 0.7 years*, a result that comports well with recent analyses of the effect of air pollution on life expectancy." Without being able to view Table SVI in my document, I would infer that the benefit to reducing ozone & PM 2.5 would add less than 1 year to one's life, suggesting the benefit is not remarkable.

In conclusion, Fann maintains that it is the largest metropolitan areas see the most in excess PM 2.5 & ozone-related deaths, Southern California sees the highest percentage of PM 2.5-related mortality, the Midwest sees the largest ozone-related deaths and the largest number of combined PM 2.5 & ozone-related deaths are in the Southeast. Noted is, "While *estimating* the contribution of air pollution to total morbidity impacts is difficult due to *incomplete data on hospitalizations and other health endpoints*, we find that the nonmortality impacts of air pollution are substantial, consistent with previous studies estimating air pollution mortality and morbidity… It is more challenging to evaluate the contribution of air pollution to total morbidity impacts. For example, incomplete information regarding the *total number and spatial distribution of asthma hospitalizations* prevent us from calculating the percentage of *total asthma hospitalizations attributable to PM2.5* ***with confidence***."

In fact, Fann devotes nearly the entire nearly 2-page **Discussions and Conclusions** section to various limitations and potential inaccuracies/deficiencies of the study. Here are just a few:

"The estimates presented here are subject to a *number of important limitations and uncertainties*, only some of which we can quantify. Many of these are *endemic to health impact assessments* (e.g., the transfer of risk estimates from epidemiology studies to other contexts and the selection of epidemiological studies used to quantify impacts) and are described in detail elsewhere.(25,55) However, certain sources of uncertainties are likely to influence the analysis *greatly* and are worth noting here. This health impact analysis ***relies upon modeled air quality estimates that utilize a national emissions inventory***. Previous analyses(46) have found that *even small errors in emission inventory, when compounded with other uncertainties in the analysis, can have a significant impact on the overall size of the estimated health impacts."*

"As another example of how alternative methods would have affected our results, we might have employed an Institute of Occupational Medicine (IOM)-style life table approach, calculating lifetime air pollution risk among a cohort of individuals. A principal advantage of this technique is that it *characterizes changes in risk among a population cohort over time and reduces the chance that the same health impact may be counted twice from one year to the next*. Future research might build upon this analysis by employing the IOM life-table tool in conjunction with both historical and projected air quality. Such a method would yield an *improved characterization of the public health burden over time after the implementation of national air quality regulations*."

"One advantage of this type of comparative risk assessment (GBD) is that by attempting to systematically account for all sources of mortality risk, of which air pollution is one component, it may *reduce the potential for attributing an incorrect fraction of total mortality risk to air pollution*."

"U.S. EPA projections of future air quality indicate that overall ambient levels of PM2.5 and ozone will decline significantly compared to those levels estimated here. We anticipate that these rules will address a large portion of the PM2.5 and O3 public health burden identified in this article."

"*While we estimate PM2.5-related mortality using risk estimates drawn from two long-term cohort studies*, we use both a *short-term time-series study and a long-term cohort study to quantify ozone impacts*. PM2.5 cohort analyses are generally understood to better characterize the total risk of PM2.5 exposure over time because they capture the impacts of both long-term and some portion of short-term exposures. (46,53,56) However, it is *less clear* as to whether there is a separate short- and long-term mortality impact related to ozone exposure—or whether the *longterm study used in this analysis might be capturing these impacts*. For this reason, *there is some uncertainty as to whether the ozone mortality impacts estimated using the Levy et al. (2005) short-term study and the Jerrett et al. (2009) long-term study are additive or overlapping*."

Fann et al also says in a footnote, "This 153-day period generally conforms to the ozone season across most parts of the United States and contains the majority of days with observed high ozone concentrations in 2005. We acknowledge that the ozone season extends beyond these dates in some urban areas (e.g., Houston, L.A.) *and failing to account for the full duration of the season in these areas may introduce a downward bias in our estimate of health impacts*.

The most noteworthy problem with the study is that it relies on the NEI for emissions data. The NEI has been highly criticized by the NARSTO group as well as many scientists for a variety of reasons. NARSTO, in its "Improving Emission Inventories for Effective Air Quality Management Across North America" has cited its weaknesses as being:

*Quality assurance and quality control procedures are not strictly applied in the development of most emission models and inventories.
*Emissions for mobile sources and many important stationary nonpoint source categories are uncertain and inadequately characterized.
*Emission estimates are frequently based on a small number of emission measurements that may not be representative of real world activity.
*Insuffi cient information is available on chemical composition – of both PM and gases – for many sources.

Finding: This study has a host of accuracy issues, which could potentially negate part or all of its findings. It should not be used as supporting evidence by DEQ for its proposed dual actions in this proposed negotiated rule making.

PM 2.5 Analysis. There are other studies that seem to articulate the need for the EPA to establish a causal relationship between particulate matter and deaths not caused from accidents (all-cause). It would seem logical that if PM 2.5 causes negative health effects and premature death, then to reduce PM 2.5 would reduce these incidences. It would seem the higher the PM 2.5 concentrations, the more ER visits and deaths and the lower the PM 2.5 concentrations, the less ER visits and deaths. This has not to date been shown to be unequivocably true. Cox et al in a massive study "**Temperature, Not Fine Particulate Matter (PM2.5), is Causally Associated with Short-Term Acute Daily Mortality Rates: Results from One Hundred United States Cities"**, used Bayesian

model-averaging, rather than a single model. The study showed that there is a higher incidence of ER visits and deaths in warmer months, than when PM 2.5 is elevated.

The study suggests, "It is worth revisiting this causal interpretation of the statistical evidence. *Do reductions in recent ambient levels of PM2.5* cause *reductions in mortality rates* (e.g., by reducing cardiovascular disease (CVD) and other inflammatory diseases of the lung and heart that can be exacerbated by high levels of pollutants), or might the historical associations between PM2.5 levels and mortality rates reported in multiple cities and countries reflect coincident trends, modeling artifacts, incomplete control of confounders, or other non-causal explanations? The role of causation in reported associations has often been questioned and discussed, but without an unequivocal resolution (Clyde 2000; NRC 2002; Green and Armstrong 2003; GAO 2006; Koop *et al.* 2007; Schwartz 2007." Clyde recommended applying *Bayesian model-averaging* (BMA) (i.e., using the data to identify an ensemble of plausible models, all of which are consistent with the data, based on their relative likelihoods; and examining the fraction of them that indicate a significant PM-mortality association) as a more objective way to interpret the data than selecting any single model. Cox maintains, To avoid or reduce potential model selection biases, our main analysis emphasizes simple nonparametric *comparisons of death rates on high- vs. matched low-pollution day.* The result of applying this non-parametric test to the Hundred Cities data was that 0.51 (95% CI [0.47, 0.55]) of the city-month-temperature combinations had a greater mortality rate for the high PM2.5 exposure days than for the matched low-exposure days. This evidence does not allow the null hypothesis of no difference to be rejected. The mean difference between mortality rates on high-exposure vs. low-exposure days matched for city, month, and mean daily temperature also has a 95% confidence interval that includes zero, and hence leads to the same conclusion. If these nonparametric results are accepted at face value, it appears that, in this data set, the highly statistically significant association between PM2.5 and all-cause mortality is largely or completely explained by the confounding effects of temperature and month in model-free analysis. (The converse is not true: differences in PM2.5 levels do not explain away the effect of Month, as shown in classification tree analysis.) The same conclusion also holds for cardiovascular and other cause-specific mortality risks and for lagged as well as unlagged PM2.5 levels: In this large data set, in the nonparametric analyses performed, it does not appear that current or lagged PM2.5 values have statistically significant positive associations with all-cause or cause-specific mortality rates, after conditioning on month and temperature-related variables. Thus, the significant statistical C-R associations between PM2.5 and mortality rates do not appear to reflect a causal relation, based on these conditional independence tests."

"Whatever the explanations for differences across studies, the implications for risk-based policy and communication seem clear. Suggesting that each unit of increase in PM2.5 corresponds to (or produces) a certain proportional increase in daily mortality rate, as is now commonly done in reporting study results (e.g., Ostro *et al.* 2006; Katsouyanni *et al.* 2009; Balakrishnan *et al.* 2011; **EPA 2011; Fann *et al.* 2012**), is overly simple and misleading. Rather, such correspondences appear to exist in some cities but not in others, and for some models and analyses but not for others (Clyde 2000; Koop and Tole. 2004; Franklin *et al.* 2007; Schwartz 2007). Policy makers should not be led to expect that further reducing exposure PM2.5 concentrations will necessarily further reduce daily mortality rates, as the actual effects caused by such a change are neither as known nor as consistent as such a statement suggests. Even where positive associations do exist, they may not be *causal*. The results of our BMA, classification tree (CART), conditional independence, and Granger-Sims analyses suggest that, at least in this study of many U.S. cities, the *causal impact of PM2.5 on daily mortality rates is far less clear than might previously have been expected*." (Emphasis added)

The EPA & the CASAB have stated that the weight of epidemiological evidence supports a determination of causality. According to Tony Cox's comments to OMB-2014-0002, "…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 PM2.5 and human health effects, this *causal fraction* has never been determined. 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… 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 must be noted here that the Energy & Environment Legal Institute (EELI) is curently suing EPA on behalf of the Western States Trucking Association and Dr. James Enstrom, a retired University of California-Los Angeles epidemiologist who was blacklisted for challenging EPA claims about particulate matter. "2. This Action arises out of the appointment by EPA Of members of the Clean Air Scientific Advisory Committee Particulate Matter Review Panel (CASAC PM Panel) on November 17, 2015. EPA Violated its obligation to appoint an " Independent scientific review committee " Pursuant to the Clean Air Act, 42U.S.C.§7409(d)(2)(A)." Apparently, according to the the EPA's Research Grants data base, 22 of 26 members of the latest CASAC PM Panel have been or are "principal investigators" for EPA research and collectively have received over $190 million in discretionary grants from the EPA. This brings me back to my first comment letter's reference to the House of Representatives' Subcommittee on Energy and the Environment Committee on Science, Space, and Technology's 10/4/2011 hearing on "Quality Science for Quality Air" and the testimony of Dr. Anne E. Smith, Ph.D. who said in part, "Thus, overnight in 2009, in the course of preparing RIAs that are not subject to public peer review, EPA dramatically escalated its estimates of benefits for all of its RIAs." The resultant affect of EPA's "overnight" actions, was the motivation by HR 1030, the Secret Science Reform Act of 2015 which has overwhelming passed in the House. Plainly stated, the EPA's science is often quoted in other studies, but it is unclear if EPA's science is true science or politically-motivated precautionary pseudo science.

Another study, "Reassessing the human health benefits from cleaner air" says in part, "Recent proposals to further reduce permitted levels of air pollution emissions are supported by high projected values of resulting public health benefits. For example, the Environmental Protection Agency recently estimated that the 1990 Clean Air Act Amendment (CAAA) will produce human health benefits in 2020, from reduced mortality rates, valued at nearly $2 trillion per year, compared to compliance costs of $65 billion ($0.065 trillion). However, while compliance costs can be measured, ***health benefits are unproved**: **they depend on a series of uncertain assumptions***."

Summarily, DEQ has failed through its scientific documentation to demonstrate that the air pollutant ozone is less danger than the other NAAQS air pollutants. DEQ has failed to demonstrate through its scientific documentation that PM 2.5 is more dangerous than the other NAAQS air pollutants, including ozone. DEQ's scientific documentation, if accepted as accurate, demonstrates that neither ozone or PM 2.5 pose a great health risk in Idaho, when compared to most of the nation. It is seemingly ludicrous for DEQ to accept EPA's decision that there are 6 pollutants of concern to human health, set limits more restrictive than EPA's then attempt to justify through a few studies, that one is better or worse than another.

Logical actions could include DEQ either drop the negotiated proposed rule making to let the 70% stand for all 6 NAAQS air pollutants, or raise all 6 NAAQS air pollutants to 90%, which is still below the NAAQS. Apparently that option is available.

Jann Higdem