The Energy & Environment Legal Institute

Submits:

Comments on
Proposed Rule: National Ambient Air Quality Standards for Ozone
78 Fed. Reg. 75,234 (December 17, 2014)

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U.S. Environmental Protection Agency

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INTRODUCTION

The Energy and Environment Legal Institute (E&E Legal) is a 501(c)(3) organization engaged in strategic litigation, policy research, and public education on important energy and environmental issues. E&E Legal seeks to address and correct onerous federal and state governmental actions that negatively impact energy and the environment. E&E Legal advocates responsible resource development, sound science, respect for property rights, and a commitment to markets as it holds accountable those who seek excessive and destructive government regulation that's based on agenda-driven policy making, junk science, and hysteria.

SUMMARY OF COMMENTS

EPA's proposed ozone rule will cause more harm than it will prevent. The Agency has long held that "those who bear a regulation's compliance costs may also suffer a decline in their health status, and if the costs are large enough, these increased risks might be greater than the direct risk-reduction benefits of the regulation." That is the case for the ozone proposal. None of the three proposals has a benefit to society, considering exclusively the health effects of the proposed alternatives. Specifically, the regulatory alternatives will cause more premature death than they will prevent. Because the estimated premature death avoided is more than 98 % of the estimated benefit of the proposals, and because the loss of disposable income will cause from 17 to 31 times more premature death than the rules will prevent, the rules have no benefit to society and do not protect human health. For this and other reasons discussed below, EPA should withdraw its proposal.

COMMENTS

I. EPA has conflated $PM_{2.5}$ controls with a rule on Ozone.

Based on EPA's own data, and taking the most generous estimate of ozone benefits, the costs exceed the benefits, ranging anywhere from half a billion dollars for a 70 parts per billion standard to $19 billion for a 60 parts per billion standard.

The benefits only outweigh costs if taking into account $PM_{2.5}$ co-benefits. $PM_{2.5}$ co-benefits, assuming the highest levels of benefits, account for anywhere between 70–75% of the total benefits, depending on the standard. In other words, for the proposed ozone standard, ozone benefits are only about a quarter of the benefits.

While the EPA has constantly used $PM_{2.5}$ co-benefits to improperly justify a seemingly endless amount of regulations that have nothing to do with the reason for the regulations, using $PM_{2.5}$ co-benefits to make the case for an ozone standard is particularly egregious.

The EPA has a very clear and direct means to address $PM_{2.5}$ through the National Ambient Air Quality Standards process, and that, not surprisingly, is through the $PM_{2.5}$ standard. The ozone standard is a distinct standard that is supposed to be focused on ozone.

To sell a more stringent ozone standard, the EPA lists a series of alleged facts in its "By the Numbers" document that will scare the public into thinking a more stringent standard is necessary. For example, according to the EPA, setting the ozone standard to 70 parts per billion or 65 parts per billions would avoid:

- 65,000–180,000 missed work days; and
- 790 to 2,300 cases of acute bronchitis among children

Neither of these alleged facts, however, are based on reductions in ozone. They are based exclusively on assumed $PM_{2.5}$.

The public is being led to believe that reducing ozone achieves these health benefits. In reality, much of these alleged benefits have nothing to do with an actual reduction in ozone. These numbers, assuming the facts listed are reasonable estimates, should also be put in perspective. For example, the EPA claims making the standard more stringent would avoid 1,400 to 4,300 asthma-related emergency room visits. Given that there are about 1.8 million such visits in a year, the reduction amounts to eight-hundredths of one percent to two-tenths of one percent (0.08% to 0.2%) a minuscule impact at best. Further, these numbers are based on both reductions in $PM_{2.5}$ and ozone, not on ozone alone. Taking ozone alone, the evidence EPA has cited (and discussed below) could even be used to suggest that small ozone exposures improve asthma conditions. And, as discussed below, ozone alone is not well associated with asthma.

EPA controls $PM_{2.5}$ through a NAAQS. If EPA believes it necessary to further reduce $PM_{2.5}$, it should do so through revision of the $PM_{2.5}$ NAAQS. But, EPA had the opportunity to further reduce the $PM_{2.5}$ standard and did not. In part, this is because $PM_{2.5}$ has a threshold of effect. EPA's use of the ozone rule to address PM2.5 if inappropriate. EPA cannot consider additional reductions in $PM_{2.5}$ as co-benefits as EPA has already determined that the residual $PM_{2.5}$ concentrations in the ambient air, after implementation of the $PM_{2.5}$ NAAQS, do not constitute a significant threat to human health and the environment. If EPA did believe these exposures constituted as significant threat to human health, EPA should have promulgated a significantly lower $PM_{2.5}$ NAAQS. Because EPA had the opportunity to do so, but chose not to do so, EPA is not now able to claim additional threat that should be avoided as it does in the ozone proposa.

II. The Ozone Regulatory Alternatives' Benefits are Negative and will cause more death that they would prevent.

Reductions in premature deaths attributed to coincidental reductions in ozone and fine particulate ($PM_{2.5}$) pollution account for more than 98% of the estimated health benefits of the proposed rule.[1] The bases for these estimates are fatally in error. A proper analysis shows the regulatory alternatives cause more premature death than they prevent.

[1] See, RIA ES-22.

A. Benefits are Over-Estimated

EPA claims that $PM_{2.5}$ pollution currently kills thousands of Americans annually, deaths that would be avoided by the proposed rule, but these estimates are based on cherry-picked studies and extrapolation of health effects below the lowest $PM_{2.5}$ concentrations associated with mortality in epidemiological studies.[2] Such claims also conflict with toxicological studies,[3] which indicate that current $PM_{2.5}$ concentrations in U.S. cities are too low to cause significant disease or death.[4]

The rule's purported health benefits for ozone reduction are even less plausible, since asthma prevalence – especially childhood asthma rates — increased since 1980[5] while, according to EPA, ozone concentrations declined by 25%.[6] The link between asthma and ozone is simply not credible based on this single fact, a fact EPA does not and cannot dispute and has never been able to explain away.

B. EPA Fails to Count Premature Deaths the Rule will <u>CAUSE</u>

EPA's analysis callously ignores the health-wealth relationship of the proposed ozone regulatory alternatives. EPA helped lead research into the relationship between health and disposable income, commissioned studies on how to apply this knowledge and like other agencies has used this analytical method routinely. That it has not done so in this rule is a fatal flaw of its analysis of the health consequences of the regulatory alternatives. Failure to examine this aspect of the health effects of this rule constitutes arbitrary and capricious behavior that requires EPA to conduct the analysis and reoffer the rule for public comment, or more appropriately, withdraw the regulatory proposal.

Amongst the 121.1 million families in the United States (under-counting families that are not legally in residence), over nine million (9,200,000) families have pretax annual incomes of less than $10,000, averaging about $5,000 a year.[7] These families are typically minorities or the

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[2] *See* Attachment C, Goodman, J. "EPA's Assessment of Health Benefits Associated with $PM_{2.5}$ Reductions for the Final Mercury and Air Toxics Standards" available at file:///D:/1%20E&E%20Legal/111d%20Project/111d%20comments/HHRG-112-IF03-WState-JGoodman-20120208.pdf.

[3] *See* Attachment D, Schwartz, J. "Where the Bodies are Buried", available at http://johnlocke.org/site-docs/research/schwartz-tva.pdf.

[4] *See* Attachment E, Green, L.C. & Armstrong, S.R. "Particulate matter in ambient air and mortality: toxicologic perspectives" Regul Toxicol Pharmacol. 2003 Dec;38(3):326-35, abstract available at http://www.ncbi.nlm.nih.gov/pubmed/14623483.

[5] *See* Attachment F, Akinbami, L.J., et al, "Asthma Prevalence, Health Care Use, and Mortality: United States, 2005–2009" National Health Statistics Reports No. 32, available at http://www.cdc.gov/nchs/data/nhsr/nhsr032.pdf.

[6] *See* Attachment G, EPA, "National Trends in Ozone Levels" available at http://www.epa.gov/airtrends/ozone.html.

[7] U.S. Census Bureau 2012 data.

elderly on fixed income. To ignore the consequences of the rule on these families is tantamount to complete failure to examine the environmental equity of the rule. Based on the analysis below and the likelihood that premature deaths will arise within these subpopulations, the rule is both racist and ageist. As discussed below, the 60 ppb rule would consume 60 percent of that average income, a massive amount that these families cannot avoid. The health consequences of losing that amount of disposable income is significant.

The implications of cost increases on the poor are well known. The challenges these very poor families face is legend. [8]

- 28 percent did not make their full mortgage or rent payment.
- 4 percent were evicted from their home or apartment.
- 4 percent had a foreclosure on their mortgage.
- 11 percent moved in with friends or family.
- 3 percent moved into a shelter or were homeless.

They faced other significant financial problems as well.

- 15 percent got a payday loan in the past five years.
- 3 percent were forced into bankruptcy in the past year

Faced with decreased disposable income, low income persons go without food, medical care and prescription drugs. They become sick more often than those who can absorb the increases in energy bills.[9]

- 32 percent went without food for at least one day.
- 42 percent went without medical or dental care.
- 38 percent did not fill a prescription or took less than the full dose of a prescribed medication.
- 24 percent had someone in the home become sick

And, of those that become sick, some die prematurely simply because they no longer had the disposable income to pay for health care. This is the health-wealth relationship at its core.

EPA has examined this "health-wealth" relationship. Lutter and Morrall explain that

> [r]egulations to promote health and safety that are exceptionally costly relative to the expected health benefits may actually worsen health and safety, since compliance reduces other spending, including private spending on health and safety. Past studies relating income and mortality give estimates of the income loss that induces one death--a value that

[8] NEADA 2008 NEAS, http://neada.org/wp-content/uploads/2013/05/2008-NEA-Survey-Executive-Summary.pdf.

[9] *Id*.

we call willingness-to-spend (WTS)--to be around $8.5 million ($US 2006).[10]

The U.S. Environmental Protection Agency has applied this principle to economic analyses, stating: "people's wealth and health status, as measured by mortality, morbidity, and other metrics, are positively correlated. Hence, ***those who bear a regulation's compliance costs may also suffer a decline in their health status, and if the costs are large enough, these increased risks might be greater than the direct risk-reduction benefits of the regulation.***"[11] This, of course, is exactly what the NEADA 2008 Survey found and which the analysis herein documents as well.

EPA failed to estimate the number of premature deaths associated with the loss of disposable income due to its proposal. EPA should have updated and used the U.S. Office of Management and Budget (OMB) federal estimate of one premature death for every $85.5 million

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[10] Lutter, R. and Morrall, J.F., "Health-Health Analysis: A New Way to Evaluate Health and Safety Regulation", Journal of Risk and Uncertainty Vol. 8-1 pp. 43-66 (1994). There is an extensive academic literature regarding the effect of loss of wealth on health. *See*, *e.g.*, Ralph L. Keeney, "Mortality Risks Induced by Economic Expenditures", *Risk Analysis* 10(1), 147-159 (1990); Krister Hjalte et al. (2003). "Health–health analysis—an alternative method for economic appraisal of health policy and safety regulation: Some empirical Swedish estimates," *Accident Analysis & Prevention* 35(1), 37-46; W. Kip Viscusi "Risk-Risk Analysis," *Journal of Risk and Uncertainty* 8(1), 5-17 (1994); Viscusi and Richard J. Zeckhauser, "The Fatality and Injury Costs of Expenditures", *Journal of Risk and Uncertainty* 8(1), 19-41 (1994); U.S.EPA, Economic Analysis and Innovations Division, "On the relevance of risk-risk analysis to policy evaluation," August 16, 1995, http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0311-1.pdf/$file/EE-0311-1.pdf (accessed January 23, 2011); Arnold, F.S. (1995), Economic Analysis of Environmental Policy and Regulation, (John Wiley and Sons, Inc.: New York); Chapman, K.S., and G. Harihan (1994) "Controlling for Causality in the Link from Income to Mortality", Journal of Risk and Uncertainty, 8(1), 85-93; Graham, J., B. Hung-Chang, and J.S. Evans (1992) "Poorer Is Riskier", Risk Analysis, 12(3), 333-337; Keeney, R.L. (1994) "Mortality Risks Induced by the Costs of Regulations", Journal of Risk and Uncertainty, 8(1), 95-110; Lave, L.B. (1981). The Strategy of Social Regulation: Decision Frameworks for Policy, (The Brookings Institution: Washington, DC); Peltzman, S. (1975) "The Effects of Automobile Safety Regulation", Journal of Political Economy, 83(4), 677-725; Portney, P.R., and R.N. Stavins (1994) "Regulatory Review of Environmental Policy: The Potential Role for Health-Health Analysis", Journal of Risk and Uncertainty, 8(1), 111-122; Smith, V.K., D.E. Epp, and K.A. Schwabe (1994) "Cross-Country Analyses Don't Estimate Health-Health Responses", Journal of Risk and Uncertainty, 8(1), 67-84; Wildavsky, A. (1980). "Richer is Safer", The Public Interest, 60, 23-39.

[11] U.S.EPA, Economic Analysis and Innovations Division, "On the relevance of risk-risk analysis to policy evaluation," August 16, 1995, http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0311-1.pdf/$file/EE-0311-1.pdf (accessed January 23, 2011).

($US 2006) in reduction of disposable income.[12] In addition to OMB, the EPA, the Food and Drug Administration (FDA), and the Occupational Safety and Health Administration (OSHA) use this methodology to understand the degree to which their regulations induce premature death amongst those who bear the costs of federal mandates.[13]

The reduction in premature fatalities that EPA claims the rule will produce accounts for over 98% of total monetized benefits in EPA's benefits analysis. Asthma exacerbations accounts for <<1% of the total.[14] As discussed below, EPA did not balance this benefit with the negative benefit of premature deaths caused by loss of disposable income resulting from implementation of the rule. Table 1 summarizes the net premature deaths caused by the three regulatory alternatives.

Table 1
Premature Deaths Avoided and Caused by the Regulatory Alternatives
For the period through 2025 (Total U.S.)†

	60 ppb	**65 ppb**	**70 ppb**
Premature Death **Avoided**	10,000 (3,300 to 18,000)	5,000 (1,800 to 12,000)	1,800 (1,100 to 4,100)
Premature Death **Caused**			
EPA Estimate	46,000	18,000	4600
NERA Estimate	310,000	120,000*	31,000*
Ave. Net Premature Deaths Caused by the Rule	**178,000 (36,000 to 300,000)**	**64,000 (13,000 to 115,000)**	**17,800 (2,400†† to 29,200)**

†California premature deaths avoided are prorated to 2025.
* Based on the ratio of EPA to NERA estimates for 60 ppb.
†† This net prevention of premature death is an extreme value as it is based only on EPA's estimated direct costs. Use of an integrated macroeconomic model to project related losses in disposable income would likely raise this number significantly. Each of the EPA estimates reflect this gross underestimation.

NERA Economic Consulting, a firm identical in nature to those used by EPA to conduct the Agency's regulatory impact assessment, estimates significantly larger adverse impacts than does the Agency for a 0.06 ppm (60 ppb) standard.[15] The NERA-estimated loss in average annual household consumption (disposable income), applicable to the 121.1 million U.S. households is $2,175 (7% discount rate) for the period through 2025. Reflecting the integrated macroeconomic effects of this rule, the reduction in disposable income through 2025 would equal $2.7 Trillion for

[12] The dollar value of expenditures that induce one premature death was inflated to 2006 dollars using the Bureau of Labor Statistics CPI Inflation Calculator.

[13] *See* notes 10 & 11, *supra* and associated text.

[14] EPA Regulatory Impact Analysis Table 5-22, note (b).

[15] NERA Economic Consulting, "Economic Impacts of a 65 ppb National Ambient Air Quality Standard for Ozone" available at: http://www.nam.org/Issues/Energy-and-Environment/Ozone/Economic-Impacts-of-a-65-ppb-NAAQS-for-Ozone-(NERA).pdf.

a standard set at 60 ppb. This loss in disposable income will result in 310,000 premature deaths, applying a conservative estimate of one premature death for every $8.5 Million (in $2006) in lost disposable income. See Table 1.

EPA did not apply an integrated macroeconomic analysis within its regulatory analysis, did not calculate the cost per household of the regulatory alternatives, and did not calculate the premature deaths caused by the loss of disposable income. Rather, it estimated the present value (in $2006) of direct costs. Although this would be a gross underestimate of actual lost disposable income, Table 1 uses that direct costs as an extreme lower-bound estimator of premature deaths costs by the regulatory alternatives. Using that estimator, the 60 ppb alternative would result in 46,000 premature deaths. Similar extreme lower bound estimates for the other regulatory alternatives are also gross underestimates, likely to be roughly 4 to 8 times too low, because the direct cost does not capture the multiplier effects within the economy that significantly reduces disposable income.

The number of premature deaths caused by loss of disposable income is well in excess of the number of premature deaths EPA claims will be prevented by the rule. A gross assumption that the same ratio of effects at the 60 ppb level also applies to the 65 and 70 ppb alternatives again results in more death caused than death prevented and, on average, by a very wide margin.

Former EPA economic analysts describe a rule with these kinds of disproportionate impacts as a "rule with blood on its hands."[16]

EPA's failure to fully examine the adverse effects on human health associated with the proposed rule requires EPA to withdraw the rule and more properly analyze the actual harm its proposal will cause.

III. FEV_1 Studies, including the Kim and Adams Studies Do Not Support a Reduction in the Standard.

EPA relies changes in FEV_1 as the basis for concluding that very low ozone levels (<75ppb) impose a significant risk to health in children, citing to Kim, et. al, "Lung Function and Inflammatory Responses in Healthy Young Adults Exposed to 0.06 ppm Ozone for 6.6 Hours", American Journal of Respiratory and Critical Care Medicine Vol 183, 1215 (2011) and Adams "Comparison of Chamber 6.6-h Exposures to 0.04–0.08 PPM Ozone via Square-wave and Triangular Profiles on Pulmonary Responses," Inhalation Toxicology, Vol. 18, No. 2 , pp 127-136 (2006). Reliance on these studies is inappropriate.

EPA conflates a statistically significant difference between exposed and unexposed subject with a significant increase in risk. While Kim and Adams saw very small, if statistically significant, differences between the two test samples (a mere 1.7% and 2.8%, respectively), the magnitude of the actual decrement in FEV_1 values is too small to be meaningful. EPA has ignored the fact that FEV_1 is variable both within groups and in individuals. Only changes greater than

[16] Personal communication with Dr. David W. Schnare, former Chief of the Economic, Legislative and Policy Analysis Branch of the Office of Groundwater and Drinking Water.

5% are considered significant changes in health status. See, e.g., Pellegrino, et al, "Interpretative strategies for lung function tests," Eur. Respir. J. Vol. 26, pp. 948-68 (2005).

In Kim, only 6 of 59 subjects had a greater than 5% reduction in FEV_1 and of these, three had a reduction of greater than 10%, one of which was -17%. Additionally, one subject had a 15% increase in FEV_1. These five subjects are clear outliers and should have been rejected. Had they been rejected, the differences found would not be statistically significant. In nearly 90% of the subjects, the suggested effects of ozone were within normal variation and cannot be characterized as adverse. Indeed, 17 of the 59 subjects (29%) had positive FEV_1 values. Using the presumption of effect applied by Kim, these 17 subjects "benefited" from the ozone exposures. Using a Wilcoxon Sign Rank test and all the data, the effect of the 0.06 ppm (60 ppb) ozone exposures is not statistically significant and EPA can put no reliance on the Kim paper using standard scientific principles and under EPA's own data quality act guidances.

The Balmes study further demonstrates that FEV1 is not correlated with measures of inflammation and thus a presumption of adverse health effect from ozone, on the basis of a decrement in FEV1, is inappropriate. See, J R Balmes, L L Chen, C Scannell, I Tager, D Christian, P Q Hearne, T Kelly, and R M Aris "Ozone-induced decrements in FEV1 and FVC do not correlate with measures of inflammation." American Journal of Respiratory and Critical Care Medicine, Vol. 153, No. 3 (1996), pp. 904-9.

IV. Ozone, alone, does not cause Asthma.

Kim must be weighed against other studies of children and when done is found to be an outlier, probably because the data it used contained outliers. For example, "Despite occasional high levels of central-site O_3 (8-hr maximum O3 90th percentile, 83.9 ppb), O_3 was not associated with percent predicted FEV_1." Delfino, et. al, "FEV_1 in asthmatic children and airborne PM," Environmental Health Perspective Vol. 112 (8), (2004).

In fact, asthma severity is not associated with FEV_1 and "FEV_1 % predicted did not differ by level of asthma severity." Bacharier, et.al, "Classifying Asthma Severity in Children, mismatch between symptoms, medication use, and lung function," Am J Respir Crit Care Med Vol 170. pp 426–432 (2004),

A one hour exposure to 120 ppb ozone does not cause any significant respiratory effects in healthy or asthmatic adolescents. See, Koenig, et al, "Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents," Am. Rev. Respir. Dis. Vol. 132(3):648-51 (1985). Other studies have found similar results. See, Holz, et al, "Ozone-induced Airway Inflammatory Changes Differ between Individuals and Are Reproducible," American Journal of Respiratory and Critical Care Medicine, Vol. 159, No. 3, pp. 776-784 (1999) and Chen, et al, "Effect of ozone exposure on airway responses to inhaled allergen in asthmatic subjects," Chest. Vol. 125(6):2328-35 (2004).

There is mixed evidence that high ozone days increase the number of hospitalizations for asthma, and several multi-city studies show no relationship: Schildcrout et al, "Ambient Air

Pollution and Asthma Exacerbations in Children: An Eight-City Analysis," Am. J. Epidemiol. 164 (6): 505-517 (2006); and, O'Connor et al, "Acute respiratory health effects of air pollution on children with asthma in US inner cities" Journal of Allergy and Clinical Immunology , Volume 121 , Issue 5 , 1133 – 1139 (2008).

Over the last ten years, the incidence of asthma has increased[17], whereas, and according to EPA itself, the ambient concentrations of ozone have decreased.[18] If asthma incidence was associated with ozone concentrations, then the incidence should be going down, not up.

Altogether, there is very little evidence that people with asthma are more sensitive to ozone. However, there are many other known triggers for asthma, including cold dry air, allergens, tobacco smoke, dust mites and mold. The Centers for Disease Control have information about these triggers: http://www.cdc.gov/asthma/triggers.html. All but cold dry air are controlled under the NAAQS for $PM_{2.5}$. Thus, even if ozone exacerbates effects from PM 2.5, the way to control any asthma hazards is through control of $PM_{2.5}$, especially in light of EPA's arguments that $PM_{2.5}$ is lethal at any dose.

Finally, clinical studies using direct exposure to ozone and epidemiological studies that assume ozone exposures equal to those measured at monitors do not replicate actual human exposures. Several national studies have shown that actual personal exposure is much lower than the concentrations of ozone that the EPA is considering for a new, lower standard (Meng et al 2012). This is also true for outdoor workers. For example, a study by O'Neill et al 2003 reported that outdoor workers in Mexico City experienced average personal ozone exposures that were 60% lower than ambient monitor levels. In addition, there is a protective ozone standard already in place for outdoor workers in the United States. Thus, the studies like Kim and Adams and the epidemiological studies that estimated human exposure from levels measured at monitors over-estimate the dose-response relationship and must be discounted. Only those studies using personal exposure data have sufficient scientific utility as the basis for EPA regulations and none of those studies support a standard less than the current standard (and do not actually support the current standard either). In fact, the EPA scientific advisory committee that reviewed the Agency's ozone assessment stated:

> The Ozone Staff Paper should consider the problem of exposure measurement error in ozone mortality time-series studies. It is known that personal exposure to ozone is not reflected adequately, and sometimes not at all, by ozone concentrations measured at central monitoring sites….Therefore, it seems unlikely that the observed associations between short-term ozone concentrations and daily mortality are due solely to ozone itself."

CASAC ozone review panel – June 5, 2006

Applying the weight of the evidence approach EPA states is uses, the Agency must conclude that asthmatics are not necessarily more sensitive to ozone than non-asthmatics.

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[17] CDC, "Asthma Facts, CDC's National Asthma Control Program Grantees" (July 2013), available at: http://www.cdc.gov/asthma/pdfs/asthma_facts_program_grantees.pdf and included in this comment by reference.

[18] EPA, National Trends in Ozone Levels (2014). Available at http://www.epa.gov/airtrends/ozone.html.

V. Ozone concentrations below 75 ppb are not associated with increased Mortality.

The relationship between long-term ozone exposure and mortality has been investigated in at least 12 epidemiology studies. When considering other potential causes of mortality, such as other air pollutants, ***only one*** of those studies showed a statistically significant (but very small) effect of ozone on mortality.

Table 2
Studies examining the relationship between long-term ozone exposure and mortality, while considering other air pollutants

Statistically Significant Effect	NO Statistically Significant Effect
Jerrett et al 2009	Abbey et al 1999 http://www.nejm.org/doi/full/10.1056/NEJM199312093292401
	Lipfert et al 2000 http://informahealthcare.com/doi/abs/10.1080/713856640
	Pope et al 2000 http://jama.jamanetwork.com/article.aspx?articleid=194704
	Chen et al 2005 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1314912/
	Jerrett et al 2005 http://journals.lww.com/epidem/pages/articleviewer.aspx?year=2005&issue=11000&article=00004&type=abstract
	Lipfert et al 2006a http://informahealthcare.com/doi/abs/10.1080/08958370600742946
	Lipfert et al 2006b http://www.sciencedirect.com/science/article/pii/S1352231005008459
	Krewski et al 2009 http://pubs.healtheffects.org/view.php?id=6
	Smith et al 2009 http://informahealthcare.com/doi/abs/10.1080/08958370903161612
	Wang et al 2009 http://www.ncbi.nlm.nih.gov/pubmed/?term=Long-term+exposure+to+gaseous+air+pollutants+and+cardio-respiratory+mortality+in+Brisbane%2C+Australia

Different cities have different associations between short-term exposure to ozone and mortality, and very few of those associations are positive. This has been shown by many studies (Smith et al 2009, Bell et al 2004, Bell et al 2005, Zanobetti & Schwartz 2008). Of those cities that

do show an association with mortality, there is no correlation between a positive association of ozone with mortality, and the ambient concentrations of ozone in that city:

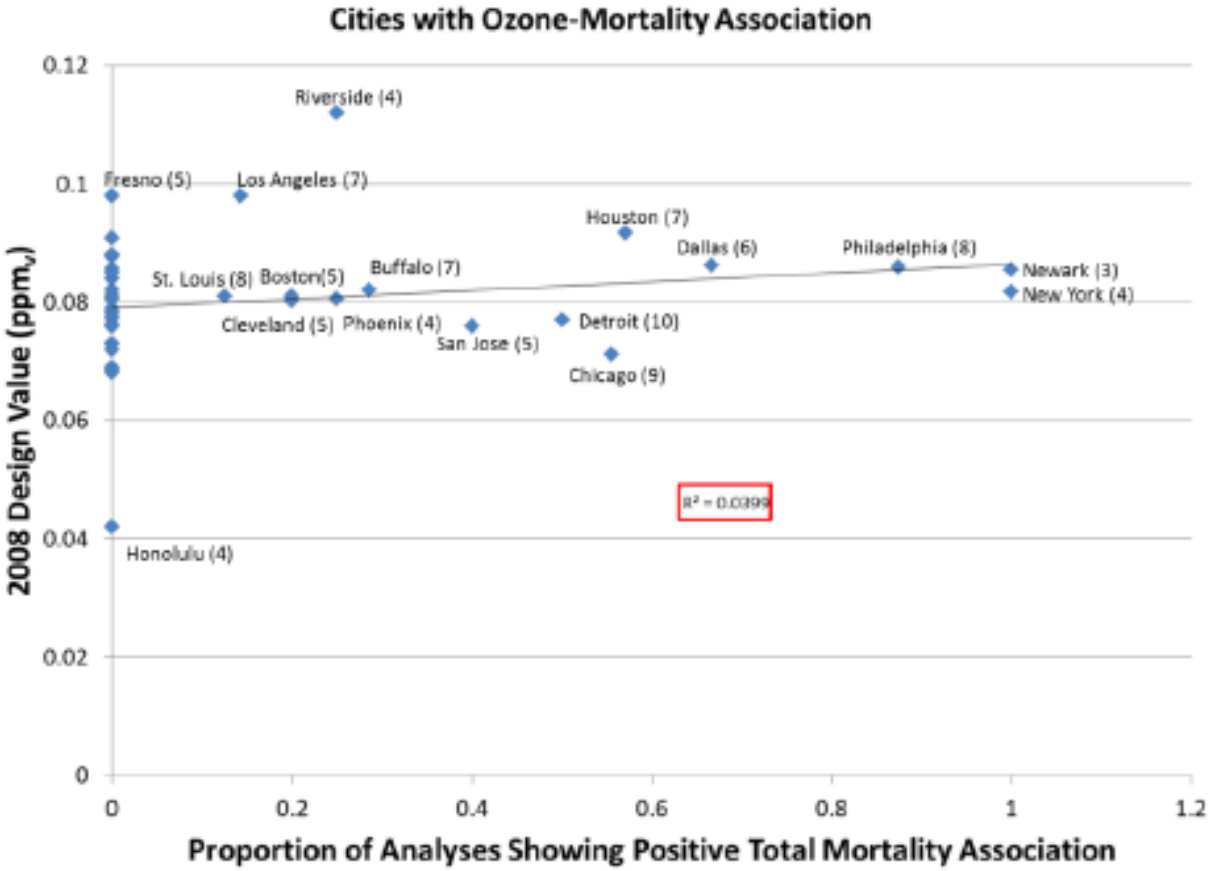

Figure 1. *Graph comparing the proportion of studies that have shown a positive association between ozone and mortality for a particular city (number of studies shown in parentheses next to the city name), compared to the 2008 ambient concentrations of ozone in those cities.*

Even when a positive association is observed between short-term mortality and ozone concentration, that association is very small when considering other factors that affect mortality, such as socioeconomic status, temperature, time of year, and even napping:

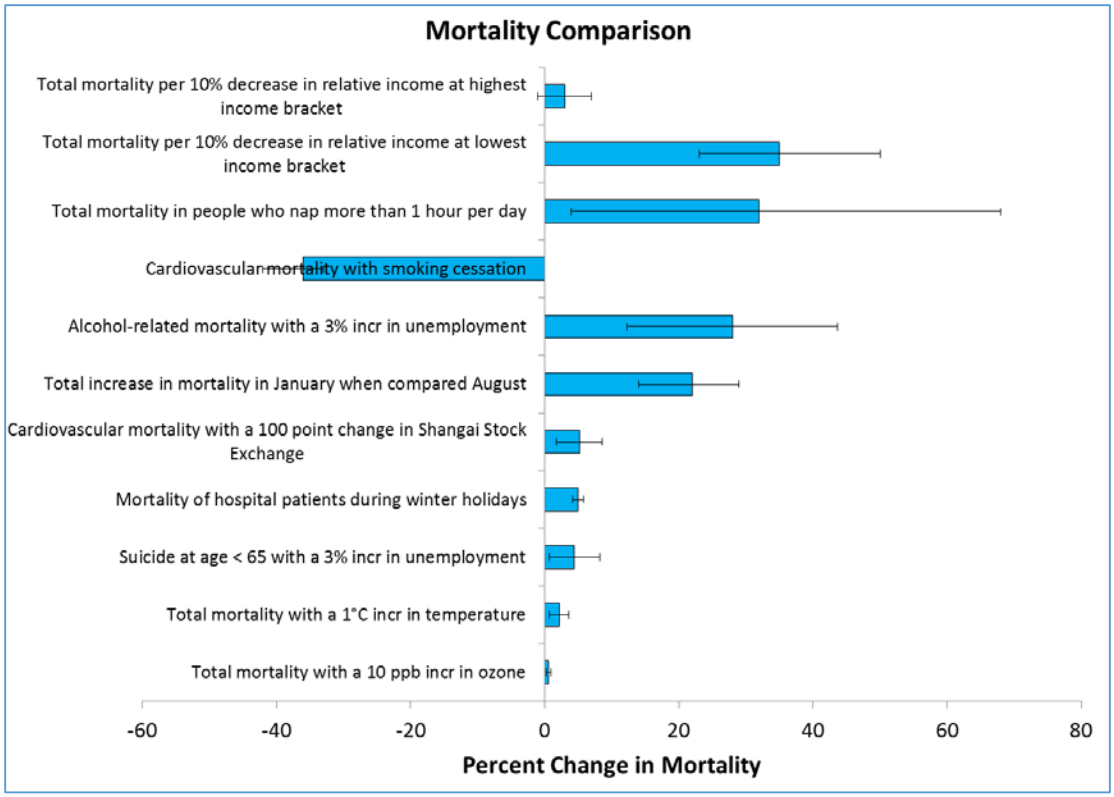

Figure 2: *Graph comparing the percent change in mortality caused by different stimuli. The reference table for this figure is at the end of this document.*

Statistic	Reference (s)	Pubmed ID
Total mortality per 10% decrease in relative income at highest income bracket	Elstad et al 2006. Eur J Public Health. Associations between relative income and mortality in Norway: a register-based study	16476681
Total mortality per 10% decrease in relative income at lowest income bracket	Elstad et al. 2006	16476681
Total mortality in people who nap more than 1 hour per day	Leng et al 2014. Am J Epidemiol. Daytime napping and the risk of all-cause and cause-specific mortality: a 13-year follow-up of a British population	24685532
Cardiovascular mortality with smoking cessation	Critchley & Capewell 2003. JAMA. Mortality risk reduction associating with smoking cessation in patients with coronary heart disease: a systematic review	12837716
Alcohol-related mortality with a 3% incr in unemployment	Stuckler et al 2009. Lancet. The public health effect of economic crises and alternative policy response in Europe: an empirical analysis	19589588
Total increase in mortality in January when compared August	Van Rossum et al 2001 Int J Epidemiol. Seasonal variation in cause-specific mortality: are there high-risk groups? 25-year follow-up of civil servants from the first whitehall study	11689530
Cardiovascular mortality with a 100 point change in Shangai Stock Exchange	Ma et al. 2011 Eur Heart J. Stock volatility as a risk factor for coronary heart disease	21196446
Mortality of hospital patients during winter holidays	Phillips et al 2004. Circulation. Cardiac mortality is higher around Christmas and New Year's than at any other time: the holidays as a risk factor for death	15596560
Suicide at age < 65 with a 3% incr in unemployment	Stuckler et al 2009	19589588
Total mortality with a 1°C incr in temperature	Martiello & Giacchi 2010. Scand J Public Health. High temperatures and health outcomes: a review of the literature	20688791
Total mortality with a 10 ppb incr in ozone	All year studies referenced in Table 6-27 (page 6-222) from the EPA Ozone 2013 Integrated Science Assessment	http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=247492

Based on the EPA's analysis, lowering the ozone standard would ***increase*** overall mortality in certain U.S. cities, including Houston. These numbers were not presented in the main text, but could be found in Appendix 7 of the EPA Ozone HREA (see Final HREA, Appendices 7-9). This result is not discussed in the executive summary for the EPA Ozone Policy Assessment, although it was briefly mentioned on page 3-115 of that document, as well as on pages 7-69 – 7-70 of the HREA.

The idea that mortality increases with decreasing ozone doesn't make logical sense, and shows that the EPA models and assumptions are flawed. Consider Table 2, below, an example of EPA's analytical approach as applied to Houston, Texas.

Table 3
Number of Premature Mortalities Predicted by EPA to Occur in Houston (2009 simulation year, mortality per 100,000 people)

	Presented by EPA in Chapter 7	Based on Full Analysis found in Appendix 7 going from 2009 ozone levels to standard level
Meeting Current Standard (75 ppb) from Present Day Ozone Levels	*Not presented*	47 more deaths
Going from 75 ppb to 70 ppb	1 more death	48 more deaths
Going from 75 ppb to 65 ppb	3 fewer deaths	44 more deaths
Going from 75 ppb to 60 ppb	12 fewer deaths	35 more deaths

Because mortality has little connection to ozone concentration (and doesn't take into account personal exposure), it should not be the basis of a new, lower national standard for ozone.

CONCLUSION

For the reasons offered above, EPA should withdraw its proposed rule.

Respectfully submitted:

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