COMMENTS BY THE TEXAS COMMISSION ON ENVIRONMENTAL QUALITY REGARDING THE PROPOSED NATIONAL AMBIENT AIR QUALITY STANDARDS FOR OZONE

DOCKET ID NO. EPA-HQ-OAR-2008-0699

Table of Contents

I. Summary

On December 17, 2014, the United States (U.S.) Environmental Protection Agency (EPA) published in the *Federal Register* a notice of proposed rulemaking regarding the National Ambient Air Quality Standards (NAAQS) for ozone (79 FR 75234). The Texas Commission on Environmental Quality (TCEQ) provides the following comments on this proposed rule (PR).

II. Comments

A. The Primary Standard

The reference list for the following comments regarding the primary standard can be found in Appendix A.

Section 1. Overview of Comments on the Primary Standard

Pertaining to the primary standard, the EPA and the EPA Administrator proposed retaining the indicator, averaging time and form of the current ozone NAAQS, and they proposed to lower the level within the range of 0.065 – 0.07 parts per million (ppm). The Administrator also requested comments on:

- Alternative standard levels below 0.065 ppm, and as low as 0.06 ppm;
- Both the Administrator's proposed decision to revise the current primary ozone standard and the option of retaining that standard;
- On her proposed decision to revise the current primary ozone NAAQS, including consideration and proposed conclusions based on the scientific evidence, exposure/risk information, and advice from the Clean Air Scientific Advisory Committee (CASAC); and
- On the potential approaches to viewing the scientific evidence and exposure/risk information that could support a conclusion that the current standard is requisite to protect public health with an adequate margin of safety.

In the following comments, the TCEQ will address the new proposed level of 0.065-0.07 ppm, as well as the four areas described above. In brief, the TCEQ recommends that the current ozone NAAQS level of 0.075 ppm be maintained, and so levels lower than 0.075 ppm should not be considered because the scientific evidence does not support it. This recommendation is based on our assessment of the scientific evidence and risk assessment, which demonstrates multiple flaws in the underlying studies being relied upon by the EPA to support lowering the ozone NAAQS, as described in detail below. These flaws are significant, and therefore it is irrational for the EPA to give these studies and evidence significant weight in evaluating the extent to which human health and public welfare effects are likely at lower ozone exposures.

In the following comments on the EPA's ozone proposed rule, the TCEQ will address the three sources of ozone health effects data, which are human epidemiology studies, human clinical studies (including adverse effects), and animal toxicology studies. These comments go on to consider the data supporting the designations of "at-risk" populations and the risk and exposure assessment, and finally address the cross-data stream concepts of threshold of response and evidence integration. The conclusions are as follows.

- The EPA failed to consider a critical flaw in the ozone epidemiology studies, which is that personal exposure concentrations are much lower than, and do not necessarily correlate with, ambient concentrations. The epidemiology conclusions are not robust to confounders and the effect estimates have unexplained characteristics such as regional heterogeneity and a lack of dose-response between mortality effect estimates. In addition, the epidemiology studies used in this review all suffer from the same personal exposure errors and are subject to a known publication bias (the selective publication of papers showing positive effects).

- The EPA did not consider an appropriate threshold (or in some cases, any threshold) of ozone concentration for health effects. Mechanistic toxicology data and human clinical data support a threshold of effects for ozone exposure. Therefore, an appropriate threshold should have been considered when modeling forced expiratory volume in 1 second (FEV_1) decrements and for modeling health effects based on the epidemiology data. For example, the TCEQ notes that when the best-fit threshold (56 ppb) is considered, the EPA's long-term mortality estimates decrease by 98% (US EPA 2014b, Figure 7-9).
- Contrary to the conclusions drawn by the EPA, asthmatics and children have similar spirometric responses as healthy adults to ozone, and elderly adults have a lesser response. As the elderly spend almost as much time outdoors as non-elderly adults, they do not qualify as being extrinsically at-risk from ozone exposure. There is also inadequate evidence and conflicting evidence that people carrying certain genetic variants are more at-risk for adverse effects of ozone than those without these variants.
- The TCEQ is very concerned that the EPA's modeled changes in ozone, caused by reducing nitrogen oxides (NOx), show that the greater benefit of ozone decreases will be in suburban and rural areas, whereas the greater costs would be expected to occur in the urban areas. This will cause disparate socioeconomic impacts. These differential effects on ozone in urban areas also lead to the EPA's modeled increases in mortality in Houston and Los Angeles with decreasing ozone standards. The TCEQ strongly encourages the EPA to reconsider a decision that the EPA believes will cause an increase in mortality in Houston (US EPA 2014b, Table 7B-2).
- The EPA's modeling of FEV_1 decrements and total mortality are completely inconsistent with the underlying data. Illogically, the EPA's risk estimates show that there are more people that are experiencing lung function effects from ozone than are being exposed to ozone. Similarly, because of a linear no-threshold model, the EPA is predicting an increase in deaths with decreases in ozone in several cities, which demonstrates a flaw with this model. For all of the exposures of concern, FEV_1 decrements and mortality estimates, no confidence intervals are presented, which misrepresents the uncertainties in the data.
- The EPA did not properly integrate all of the ozone health effects evidence, as is endorsed by the National Research Council (NRC). The animal toxicology and human clinical data do not support the epidemiological conclusion that ozone causes mortality at ambient concentrations and exposures.
- The TCEQ agrees with the EPA that the human clinical data is the best to use to set the NAAQS ozone standard. However, the human data analysis failed to include the filtered air responses in the dose-response curve and the entire data set of exposed individuals instead of a subset. This full dataset could have been used to inform the choice of doses that would not cause adverse effects in the population.
- When using the animal toxicology studies to inform health effects in humans, the EPA did not consider dose. Most of the animal studies cited in the integrated science assessment (ISA; US EPA 2013) use inappropriately high doses and therefore are of limited utility in providing causal information to inform human health effects at ambient ozone concentrations. Therefore, it is irrational for the EPA to give these high dose studies more than minimal weight in the EPA's consideration of the potential risks to human health and public welfare.
- The EPA did not clearly choose and justify a level of adversity for each endpoint of interest, and in particular did not explain the choice of a 10% FEV_1 decrement as being adverse for populations with preexisting lung disease. The presence of adaptive biological responses to ozone should also have been considered and the EPA should not have just assumed that any response is an adverse, clinically meaningful response.

- Included with these comments are three papers authored or co-authored by TCEQ scientists or sponsored by the TCEQ that were recently accepted for publication in the journals Environmental Management (Appendix B), Regulatory Toxicology and Pharmacology (Appendix C), and Critical Reviews in Toxicology (Appendix D). These articles discuss problems with the data used to set the ozone NAAQS, how to correctly consider different types of data specifically in the context of the four elements of the NAAQS, as well as a weight-of-evidence analysis of ozone exposure and cardiovascular biomarkers.

Section 2. Epidemiology Studies

The TCEQ agrees with the EPA Administrator that less weight should be put on the epidemiology studies because of uncertainties in the data (US EPA 2014a, pp. 75276-77). This includes uncertainties in mortality and morbidity risk estimates, the heterogeneity in effect estimates between epidemiologic study areas, the potential for epidemiology-based exposure measurement error, and the shape of the concentration-response functions at lower ozone concentrations. However, the EPA still uses the epidemiology data for making causal conclusions and for estimating benefits of the rule, which is irrational given the severity of the aforementioned uncertainties.

2.1 Lack of personal exposure data is a critical flaw in ozone epidemiology data.
The crucial flaw in the epidemiology data is that personal exposure is not considered in these studies. The EPA is aware of this flaw but chooses to ignore it when interpreting the epidemiology study results. Ecological epidemiology studies often suffer from this exposure measurement error because they assume that people are continuously exposed (24 hours a day, seven days a week) to the pollutant concentrations measured at the ambient monitors. Sometimes these studies even assume that a person is exposed to the highest pollutant concentration measured in the entire area. In the case of ozone this error is even more egregious because of the nature of ozone as a pollutant. Ozone is primarily an outdoor pollutant with ventilation and indoor structures scavenging it and removing it from indoor air. The average American adult, senior citizen, and child will spend only 5.3%, 5.8%, and 7.9% of their time outdoors, respectively (US EPA 2013, page 4-31), and therefore they will often not be exposed to ozone. Those studies that have investigated ozone personal exposure and compared it to ambient concentrations have found that personal exposure is much lower than ambient exposure (about 10% of the measured ambient level; Lee 2004), and that there may not even be a correlation between personal and ambient concentrations (Sarnat 2001, Sarnat 2005). Because of this, not only will an assumption of ambient concentrations not necessarily accurately represent the individuals in the study, it also *grossly overestimates their exposure*. This is particularly true of the short-term mortality data, where the subjects of the study are within days of death when the ambient concentrations are measured, and so are even less likely to be outdoors. In the proposal (US EPA 2014a), the EPA actually acknowledges the challenges of estimating personal ozone exposures: on page 75269, the EPA discusses the variation in ozone concentrations among microenvironments and how it means that the amount of time spent in each location and activity level will influence an individual's exposure to ambient ozone, and on page 75279 it is noted that there is uncertainty with applying area monitoring data because 1) space factors modify effects of ozone on health; and 2) spatial mobility is a key driver of individual-level exposures. However, the EPA does not explain why it does not translate these concerns into the interpretation of epidemiology studies that contain this fundamental bias. Because there may be no correlation between ambient and personal exposure, there is no way of knowing how much ozone anyone in the study was exposed to, so the EPA should not derive any concentration-response functions, nor should it draw any conclusions from these associations.

2.2 The epidemiology data associating ozone with mortality is not robust. The mechanism by which ozone causes mortality is not clear and is not consistent with epidemiology results. Many conclusions about the effects of ozone on the population, and the benefits of reducing ozone, are based on the conclusion that ozone at ambient concentrations causes premature mortality. However, the EPA is not clear on how exactly ozone could cause total non-accidental mortality at ambient concentrations. It is slightly more plausible that ozone is affecting respiratory mortality than total mortality, but those epidemiological associations are not strong. In the studies cited in this ozone review (US EPA 2013, Table 6-42 for a list of references), eight effect estimates were presented for all-year respiratory mortality, and only one was statistically significant; for summer-only data, five of seven were significant but were sensitive to particulate matter (PM) confounding. It is true that a number of epidemiology studies show an association between ozone and mortality, particularly between short-term ozone exposure and total mortality. However, given the small nature of the changes in mortality, many other factors could be affecting the association. PM confounding is an excellent example of this. Of the short-term total mortality studies used in this ozone review that investigated confounding by PM, four of the seven all-year associations and two of the five summer associations became statistically non-significant when confounding for PM was considered (US EPA 2013, Table 6-42). In the proposal, the EPA states that the mortality estimates that include PM are not necessarily reliable because there is only 1/6th the amount of data available when PM is considered (because PM data is typically only collected once every six days; US EPA 2014a, pg. 75258). However, there are often millions of deaths being investigated in these studies (Zanobetti 2008 reported 6.9 million all year deaths and 2.7 million summer deaths from the National Center for Health Statistics databank), so even with only 1/6th the data, there are still hundreds of thousands if not *millions* of data points. This is an enormous sample size, and if it is too small to see an association between ozone and mortality, then it suggests that one may not exist. In addition to PM, there are other potential confounders that could be responsible for the measured association between ozone and mortality such as temperature, other pollutants, pollen, and acid aerosols. Studies inconsistently apply confounding to their data sets, and so even if many studies show a similar association, it is difficult to compare them because of the different confounding data that is used. And measurement error applies to confounders in a similar manner as it applies to ozone (ie. personal exposure), particularly those confounders that are considered at the population-level (eg. other pollutants, demographic information). Taken together, confounding (both calculated and residual) is a problem with the interpretation of the ozone epidemiology studies, and the EPA failed to consistently address this major epidemiological concern to provide convincing evidence that ozone is indeed responsible for the measured associations with mortality and morbidity.

2.3 Regional heterogeneity of ozone epidemiology associations is unexplained. Regional heterogeneity is a characteristic of the ozone epidemiology data that is not consistent with a causal association between ozone and mortality/morbidity. It is well-documented that different cities can show different associations with ozone (in fact, the EPA states in the PR – US EPA 2014a pg. 75278 – that different effect estimates just within the New York City core-based statistical area can vary by up to *10-fold*). However, the EPA neglects to explain this. The EPA remarks on regional heterogeneity in the PR (US EPA 2014a pg. 75279), but it fails to explain how regional heterogeneity affects the conclusions that can be drawn from these studies. The presence of an association in a particular city in one study does not predict that that city will also show a positive association in another study (compare cities with significant effects between Smith 2009a, Zanobetti 2008 and Bell 2005), and to date there has been no successful explanation as to why one city shows an association between ozone and mortality and another does not (Smith 2009a). In the Smith 2009a paper, of the 98 cities investigated, only seven showed positive associations with eight-hour ozone. In fact, this rate of 7% positive associations is close to the cut-off for the number that would be expected due to chance (5% based on a 95%

confidence interval) from making that many comparisons. Because there is no toxicological explanation as to why ozone would cause mortality in one city and not another, this data supports a conclusion that ambient ozone is **not** a causative agent in premature mortality.

2.4 There is no dose-response association between ozone epidemiology studies. When considering the regional heterogeneity of ozone associations, it would be reasonable to expect that the higher the ozone levels in the city, the more likely that city would be to have a positive association between mortality and ozone concentration. However, the association of ozone with short-term mortality in a particular city does not correlate with its ozone levels (Figure 1). Similarly, the EPA notes that there is an epidemiology study that demonstrates an association between short-term ambient ozone concentrations and asthma hospital admissions (HA) in children and the elderly in Los Angeles (US EPA 2014a, pg. 75270). This association is only statistically significant when ozone alert days (high ozone days) are excluded. The EPA and the study author's explanation is that ozone-averting behavior decreased the exposure of the people in the study, which suggests that personal exposure could be important for interpreting epidemiology studies. However, ozone-averting behavior wasn't measured in this study; it was only assumed (Neidell 2010). By excluding the alert days, the authors also excluded the highest ozone days, which are the days using the concepts of dose-response where ozone is most likely to have an effect. These data are not consistent with the Hill criteria for determining causation from epidemiology studies (Hill 1965), which includes the importance of a dose-response effect.

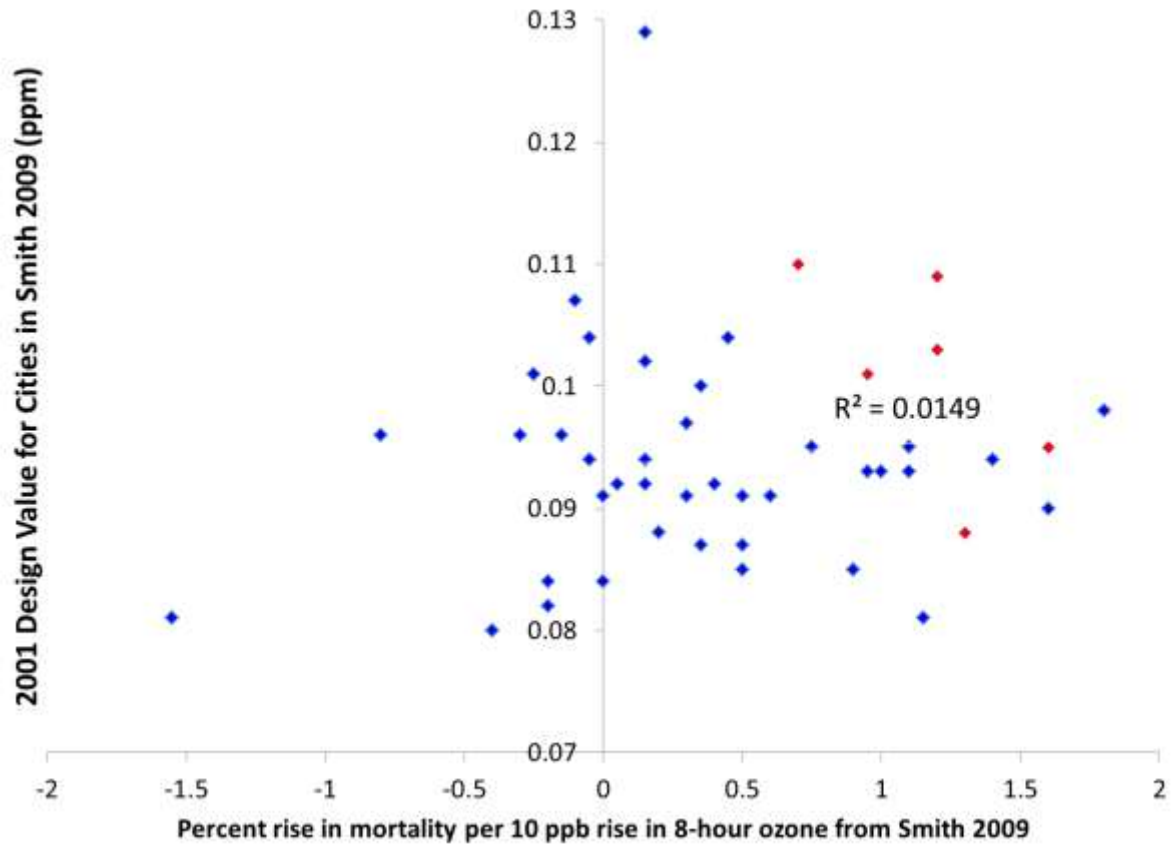


Figure 1. Association between 2001 ozone design values and eight-hour effect estimates of cities

Figure 1 Notes: Approximate mortality effect estimates (in percent rise per 10 parts per billion (ppb) increase in eight-hour ozone) from different cities in Smith et al 2009a are plotted against the available 2001 ozone design values (the 4th highest ambient ozone concentration, averaged over the years 1999-2001). Blue points represent cities whose effect estimates are not statistically significantly different from zero and red points represent cities whose effect estimates are statistically significantly different from zero. The correlation coefficient for the relationship between the mortality effect estimates and the ozone design values (R^2) is given. From Smith 2009a.

2.5 Bias and error in the epidemiology studies increases uncertainty despite the presence of many studies showing positive associations. The EPA concludes that there is a likely causal association between ozone and short-term mortality, partially because there are many studies showing positive effect estimates, some of which come from new studies. The problem with this is that, while it is true that the confidence in a causal connection increases with an increasing number of studies showing a positive association, **this does not apply if all of the studies suffer from the same error or bias** (Hill 1965). All of the epidemiology studies showing an association between ozone and mortality/morbidity have the same measurement error (ie. they do not take into account personal exposure). Therefore, an increase in the number of studies does not increase the confidence in the causal conclusion because they all have the same error. The same is true of the new studies that the EPA cites in this review – they had the same exposure measurement error, and in most cases even use the *same data sets* as previous analyses (for example, Bell 2004 and Smith 2009a use exactly the same data, but the EPA cites Smith 2009a as being a "new" study). Another important consideration when drawing conclusions about consistency of results is the presence of publication bias. Several recent publications have shown evidence for this type of bias (Bell 2005, Ito 2005, Bell 2014), which is caused by the selective publication of results showing significant associations. The EPA failed to address these discrepancies to provide convincing evidence that ozone is causing the endpoints that are attributed to it by the epidemiology studies.

2.6 A single positive epidemiology study is not enough to draw causal conclusions. The EPA concludes that there is a likely causal relationship between ozone and long-term respiratory mortality based on a single epidemiology study. The relationship between long-term ozone exposure and mortality has been investigated in at least 12 epidemiology studies (Dockery 1993, Abbey 1999, Lipfert 2000, Pope 2002, Chen 2005, Jerrett 2005, Jerrett 2009, Lipfert 2006a, Lipfert 2006b, Krewski 2009, Smith et al, 2009b, Wang 2009). When considering other potential causes of mortality, such as other air pollutants, *only one* of those studies (Jerrett 2009) showed a statistically significant (but very small) effect of ozone on respiratory mortality. The effect only occurred at temperatures above 82°F. It is well known that very warm or very cold temperatures are associated with increases in mortality (Ye et al. 2012). Paradoxically, the increased mortality was not observed in US regions with the highest ozone concentrations (southern California) nor in areas with the highest number of respiratory deaths (the northeastern US and the industrial Midwest). Therefore, similar to the short-term epidemiology studies, this long-term mortality study also demonstrated unexplained regional heterogeneity and a lack of ozone dose-response. In the face of so much contradicting evidence, the EPA should not draw conclusions based on a single study showing a positive association.

In conclusion, the EPA committed a critical error failing to consider personal exposure when interpreting the ozone epidemiology studies. Personal exposure concentrations are much lower than ambient concentrations (by up to 10-fold), and they do not necessarily even correlate with ambient concentrations. Even assuming that the exposure of the people in the epidemiology studies is appropriate, the conclusions from these studies are not robust, either to confounders or to close analysis of their assumptions. There are several characteristics of the epidemiology data that make it not scientifically-supportable for the EPA to use for drawing a causal conclusion, including the unexplained regional heterogeneity of the effect estimates and the lack

of dose-response between mortality effect estimates. Although there are a number of studies showing positive associations, particularly between ozone and premature mortality, these studies are suspect because they all suffer from the same errors and are subject to publication bias. The EPA should have addressed all of these concerns before trusting the epidemiology data to draw causal conclusions and to derive benefits estimates.

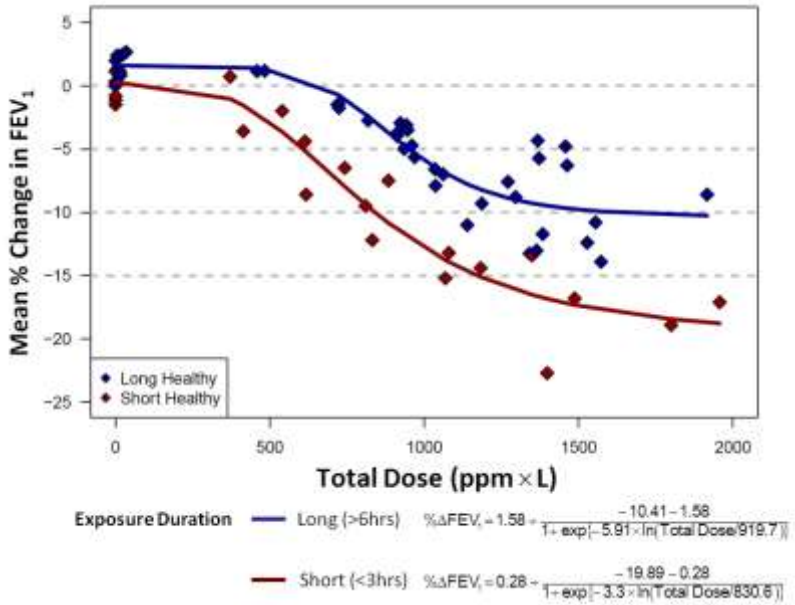
Section 3. Sensitive Populations

In the proposal, the EPA considers a number of sensitive or "at-risk" populations when determining at what level to set the ozone standard. This is entirely appropriate. However, the actual sensitivity of those at-risk populations is not accurately represented by the EPA. For example, the EPA states that clinical studies use healthy, not at-risk populations (US EPA 2014a, pp. 75245, 75273, 75288, 75295), and this needs to be considered when interpreting the results of these studies. However, a number of clinical studies have been carried out on mild asthmatics (Linn 1994, Balmes 1997, Koenig 1985, Koenig 1987, Stenfors 2002, Holz 1999, Nightingale 1999, Basha 1994, Horstman 1995), and studies have also been completed on the elderly and children (Hazucha 1985, McDonnell 1985). This provides data from which the EPA should have drawn conclusions about the sensitivity of these populations to ozone exposure.

3.1 Asthmatics do not have increased spirometric responses to ozone. Asthmatics are an at-risk population that is carefully considered in this PR. The EPA states in several places that asthmatics have a heightened spirometric response to ozone in clinical studies, and a few references are given (US EPA 2014a, pp. 75255, 75265). However, *this is not true*, as an assessment of the weight of evidence shows. Of nine studies that have investigated the spirometric responses of asthmatics compared to healthy people, eight showed no difference between the two populations (Linn 1994, Balmes 1997, Koenig 1985, Koenig 1987, Stenfors 2002, Holz 1999, Nightingale 1999, Basha 1994), and in the proposal, the EPA cites only the single study (Horstman 1995) that showed a difference (US EPA 2014a pg. 75265). In addition, when we compare asthmatics to a dose-response curve that uses data from young healthy adults, we see that the asthmatics (and children) fall along the same curve (see Appendix E and Figure 2B, further explained in Section 4.3). Together these data show that, contrary to the EPA's conclusions, asthmatics *do not* experience increased spirometric responses to ozone, and therefore it is appropriate to use healthy people to approximate their responses. One drawback to these studies is that they only use mild asthmatics. However, moderate and severe asthmatic subjects would not be able to sustain the exercise levels required to reach a dose of ozone at which an effect could be seen. For the same reason, they are not able to attain that dose in a real-life exposure situation. The EPA also states in this proposal that there were no at-risk populations in the studies showing ozone effects at concentrations of interest for this review: that is, 40, 60, and 72 ppb (US EPA 2014a, pg. 75273). The EPA therefore concludes that we do not know how at-risk populations will respond to these concentrations. However, if there is no difference in the responses between asthmatics and non-asthmatics at higher doses, logic dictates that they would have similar responses at lower doses as well. The EPA also cites studies showing that asthmatics are sensitive to ozone when considering non-spirometric endpoints, such as bronchial reactivity. However, many of the studies that investigate the effects of ozone on asthmatics do not compare them to healthy controls, thereby making it unclear whether those responses are in fact enhanced in asthmatics, or if it is a standard response at that dose of ozone (e.g. bronchial reactivity and eosinophil infiltration in asthmatics on US EPA 2014a pg. 75266 – most of the cited studies did not compare to healthy controls). This type of data does not prove that asthmatics are an at-risk population, merely that they have physiological responses to ozone. The EPA should have only drawn conclusions about asthma-specific responses if those responses had been proven to actually be asthmatic-specific. Still other studies show no difference between asthmatics and healthy people for airway hyper-

responsiveness (Linn 1994), and neutrophil recruitment (Stenfors 2002, Holz 1999, Mudway 2001, Nightingale 1999), which is not mentioned in this proposal.

A



B

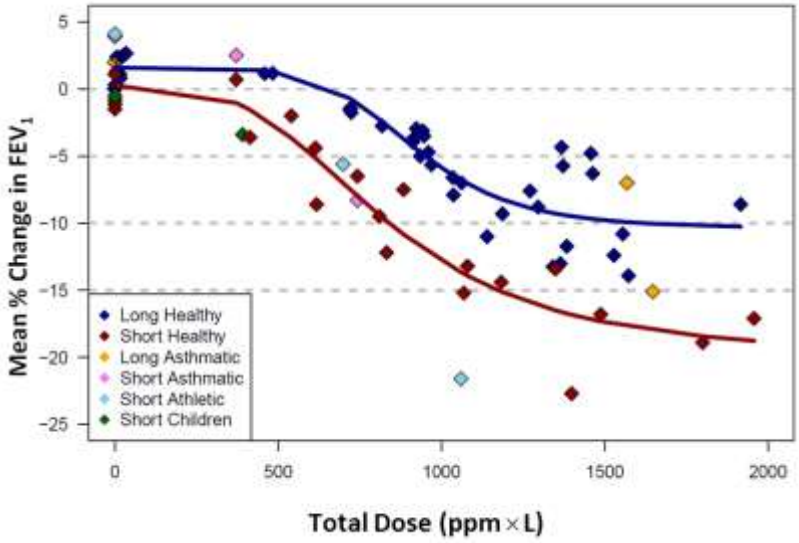


Figure 2. Dose-Response Plots

(A) Graph of total inhaled dose (in ppm×L) versus percent change in mean FEV1; data is derived from mean FEV1 change of healthy young adults exposed for ≤3 hours (short) or > 6 hours (long) to ozone while exercising. Below the plot are the equations associated with each curve.
(B) Plot of total inhaled dose (in ppm×L) versus percent change in mean FEV1 as in (A); also plotted are data from mild asthmatics exposed for < 3 hours (short asthmatic) or > 6 hours (long asthmatic); data from children exposed for < 3 hours (short children); and for elite athletes exposed for 1 hour (short athletic).

References for Figure 2 are in Appendix A.

3.2 CASAC advice about asthmatics is unclear and poorly explained. The EPA cites the advice that CASAC provides to them concerning exposures at ozone concentrations of 60 ppb being a relevant exposure of concern to asthmatics (US EPA 2014a, pp. 75252, 75289). However, as noted above, asthmatics and other sensitive populations have never been exposed to this lower concentration, and they show no difference from healthy adults at higher concentrations, so the EPA has no data to support the assertion that asthmatics are more sensitive than healthy people to 60 ppb ozone. CASAC also states that "asthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting ozone-induced pulmonary function decrements." This is a very misleading phrase that the EPA should not quote because it does not appropriately capture the available data showing that asthmatics are not more sensitive than non-asthmatics at manifesting ozone-induced pulmonary function decrements.

3.3 Asthma morbidity effect estimates are neither consistent nor robust. The EPA investigated the epidemiology studies that show effects of ambient ozone concentrations on asthma health outcomes. Keeping in mind that these studies suffer from the same exposure measurement errors as the mortality studies, the EPA showed that 21 of the 33 reported associations between ozone and asthma symptoms were *not statistically significant*, and those that were significant were not consistent with one another. This result is quantified in the regulatory impact analysis where the EPA shows that there is *no statistically significant decrease* in asthma exacerbations with a decreasing level of the ozone standard (US EPA 2014c, Table 5-19). The EPA fails to explain this important result in the proposal. The EPA also states in the PR that hospital admissions (HA) and emergency department (ED) visits are robust to co-pollutant confounders (US EPA 2014a, pg. 75258) but does not mention investigation of confounding by pollen, which is a known inducer of asthma symptoms (Lierl 2003, Delfino 1997, May 2011). Also, proper confounding by race, ethnicity, and household poverty are important considerations, as was shown in a recent study demonstrating that asthma incidence and morbidity is not more associated with urban (more polluted) areas but rather with ethnicity and poverty (Keet 2015). Therefore, the EPA should not have drawn the conclusion that ozone enhances asthma morbidity at ambient concentrations based on this data.

3.4 Children and young adults have equivalent spirometric responses to ozone, and the elderly have lesser spirometric responses. The EPA labels children as an at-risk population in the PR. The TCEQ agrees that children may be more likely to be exposed to ozone because they spend more time outside at higher ventilation rates. However, the EPA is misleading when it addresses the spirometric response of children and adolescents to ozone. It states that children, adolescents, and young adults (<18 years old) have equivalent spirometric responses, which are greater than middle-aged and older adults (US EPA 2014a, pg. 75267). What the EPA does not mention is that the responses of children and adolescents *are equivalent* to those of young adults (18-35 years old; McDonnell 1985) and that this response diminishes in middle-aged and older adults (Hazucha 1985). For readers not familiar with this literature, the EPA's statement makes children and adolescents appear to have higher spirometric responses than healthy young adults, which is not, in fact, the case (McDonnell 1985, Hazucha 1985). One of the reasons that the elderly are considered to be an at-risk population by the EPA is because they spend more time outdoors than younger adults (US EPA 2014a, pg. 75267), thereby getting higher exposures. However, according to the EPA's own assessment (US EPA 2013, pg. 4-31), elderly adults spend an average of 5.75% of their time outdoors, versus 5.26% in younger adults. This is not a big enough difference for the EPA to consider the elderly to have an extrinsic risk factor for ozone exposure.

3.5 There is insufficient information provided to designate people with certain genetic variants as at-risk. A new at-risk group designated by the EPA in this ozone review

is people carrying genetic variants that may predispose them to increased health effects upon ozone exposure (US EPA 2014a, pg. 75265). This is based largely on papers published about the Children's Health Study (CHS). These analyses connect the lower risks of asthma in lower ozone communities with particular genetic variants (HO-1, ARG1 and GSTP1) (US EPA 2014a, pg. 75265). However, this is a difficult analysis, with potential confounders in communities that may have pockets of particular ethnic groups or people who are related to one another skewing the distribution for reasons unrelated to ambient ozone concentrations or to asthma incidence. It is not clear from these studies or the EPA's discussions about them that these considerations have been properly controlled for. The EPA should have required that there be further investigation with human clinical and animal toxicology data before drawing conclusions about whether people carrying specific genetic variants (including GSTM1, GSTP1, HMOX1 and NQO1 polymorphisms) are actually more at-risk for ozone-related effects. In some cases, this data exists, and the EPA did not use it. For example, several human clinical studies have shown that people with a null allele of GSTM1 have the same inflammatory and spirometric responses to ozone as people with a functional GSTM1 allele (Kim 2011, Frampton 2015), showing that these people are not at an increased risk from ozone exposure. The EPA failed to use these types of studies to assess the biological plausibility of the findings of epidemiology studies and did not explain why.

In conclusion, the EPA's assessment of asthmatics, children, and the elderly as at-risk populations for ozone exposure was not consistent with the scientific data; asthmatics and children have similar spirometric responses as healthy adults to ozone, and elderly adults have a lesser response. As non-elderly adults spend almost as much time outdoors as elderly adults, elderly adults do not qualify as being extrinsically at-risk from ozone exposure. In addition, there is inadequate evidence that people carrying certain genetic variants are more at-risk for adverse effects of ozone than those without these variants because there are shortcomings in the studies and because the evidence is inconsistent.

Section 4. Human Clinical Studies

The TCEQ agrees with the EPA that the human clinical data is the most reliable for use in setting the level of the ozone standard. However, there are flaws in the way that the EPA used this data and modeled it to draw conclusions about the risks of ozone exposure on the general population.

4.1 Filtered air FEV_1 should not be subtracted from ozone FEV_1, and 40 ppb ozone has positive effects on FEV_1. When creating a dose-response curve, it is most appropriate to include a zero-dose point and not to subtract the zero-dose from the other doses. Therefore, an individual's filtered air FEV_1 response should not be subtracted from their ozone FEV_1 response. Because the subjects are exercising during the filtered air exposures, this methodology still accounts for the effects of exercise. In addition, comparing to the baseline measurement taken the day of the exposure controls for individual day-to-day variation. In these clinical studies the ozone and filtered air exposures can be weeks if not months apart, and because, according to the American Thoracic Society, an individual's responses can vary over time (Pellegrino 2005, Hruby 1975), comparing to filtered air exposures conducted on different days introduces error into the dose-response curve. When ozone response is compared to baseline, it is clear that filtered air actually has a positive effect on FEV_1, particularly for long exposures, and that this positive effect is maintained at low doses of ozone, including 40 ppb exposures for 6.6 hours with intermittent moderate-vigorous exercise (see Adams 2006, Figure 1). The PR states that FEV_1 is decreased (non-significantly) at 40 ppb (US EPA 2014a, pg. 75249), but actually, when compared to the pre-exposure baseline, FEV_1 increases at 40 ppb (Adams 2006, Adams 2002), and it is not until 60 ppb that any decreases are measured. Even for exposures at 60 ppb for 6.6 hours with intermittent moderate-vigorous exercise, the decreases in FEV_1 are slight, mostly not statistically significant, are not associated with symptoms, and are well within the range of

normal daily variation in FEV_1, which the American Thoracic Society (ATS) considers to be ± 5% (Pellegrino 2005). The EPA incorrectly placed too much weight on the FEV_1 decrements at 60 ppb, which were actually very small and not adverse.

4.2 It is inappropriate to only use the most responsive people in clinical trial study groups. The EPA should not have used only the most responsive people in the study groups to model population responses to ozone. Their models inappropriately use only those with FEV_1 decrements >10%, >15%, or >20% and discard the rest of the data. This is particularly concerning because people may not be reproducibly responsive to ozone exposure (LeFohn 2010), which means that using only the most responsive people in clinical trial study groups does not ensure that the EPA is meeting its statutory mandate to protect sensitive groups. Instead, EPA's reliance on data from clinical trials that use only the most responsive people irrationally ignores large portions of relevant data. The most scientifically appropriate decision is to use all of the data, which means looking at mean ± standard deviation, not only the small number of people outside of one standard deviation. The "MSS" and exposure-response(E-R) models incorrectly used only the most responsive people, so even though 120 people have been exposed to 60 ppb ozone for 6.6 hours with moderate-vigorous exercise (and 30 of them were exposed twice), only nine to 11 people experienced FEV_1 decrements of >10% (five experienced >15% and one experienced >20%), so *the entirety of the conclusion and the model of the response to 60 ppb, which is then applied to the entire population, is based on* ***nine to 11 people*** (US EPA 2014b, Table 6-3). This is not an adequate sample size upon which to base a standard that is expected to apply to >300 million people. It is irrational of the EPA to rely on such small sample sizes to make determinations regarding likely human health and public welfare impacts. In addition, the proposal states that:

> The legislative history of section 109 indicates that a primary standard is to be set at "the maximum permissible ambient air level…which will protect the health of any [sensitive] group of the population," and that, for this purpose, "reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group." S. Rep. No. 91-1196, 91st Cong., 2d Sess.10 (1970) (US EPA 2014a pg. 75237).

Therefore, the EPA should have drawn conclusions about sensitive people in the population by using studies that investigated those sensitive populations (such as when asthmatics or children are included in the clinical studies) rather than singling out potentially more sensitive individuals in a group.

4.3 Ozone dose, which is exposure time, concentration and ventilation, must be considered when interpreting responses and when extrapolating to the entire population. The EPA did not properly consider ozone dose when interpreting the human clinical data. Ozone total dose includes three factors: time of exposure, concentration, and ventilation rate. The EPA emphasizes only the concentration without properly considering and communicating the necessity of the other two factors and their combined effects on human health and public welfare. For example, the risk assessment outcomes that are presented in the PR draw from the data for the E-R model that the EPA presents on page 6-18 of the final draft of the Health Risk and Exposure Assessment (HREA; US EPA 2014b). This model shows responses at different concentrations of ozone but does not consider the dose of these effects. The text suggests that exercise and time of exposure are equal, so only concentration needs to be discussed. However, the actual total ozone inhaled doses at 40 ppb ranged from 458-483 ppm·L; those exposed to 60 ppb ranged from 719 – 818 ppm·L; 72 ppb was at a dose of 935 ppm·L (a single study, with only 6 people with responses higher than 10%, used to draw conclusions about the effects of 72 ppb ozone on the whole population); and at 80 ppb the doses ranged from 912 - 1062 ppm·L. Because these people are not exposed to the same dose, they cannot be judged to have the same exposure and would therefore not be expected to respond consistently. This makes the EPA's dose-response curve incorrect, so any risk estimates (and

conclusions) derived from that would also be incorrect. Therefore, the EPA should have analyzed *dose*, not concentration, in *dose*-response curves, and then information should have been extrapolated out of that. We have done this, and have attached a copy of a poster (presented at the Society for Risk Analysis in Dec. 2014; see Appendix E) explaining how EPA should have used population means and FEV_1 to model dose-response. The EPA failed to conduct this data analysis that includes all of the data, using real-world ventilation rates, to inform the Administrator in making her decision about ozone concentrations that will protect public health with an adequate margin of safety. The TCEQ's properly conducted alternative analysis demonstrates that a person would have to be exercising at 44.9 L/min (equivalent of high intensity exercise in a child) for *eight hours continuously* at 75 ppb to achieve an FEV_1 decrement of 10% (Appendix E, Table 3). Given the unlikely nature of this exposure scenario, a 75 ppb standard is adequately protective of public health.

In conclusion, the TCEQ agrees with the EPA that the human clinical data is the best to use to set the ozone NAAQS. However, the EPA incorrectly analyzed the data. The EPA failed to include the filtered air responses in the dose-response curve (it should not have subtracted them from ozone responses), and should have included the *entire* data set of exposed individuals instead of a subset. This more scientifically appropriate analysis should have been used to inform the choice of doses that will not cause adverse effects in the population. Figure 2a (a dose-response curve) and Table 1 make use of this information and provide a tool for the EPA Administrator to best choose an ozone standard to protect public health.

Table 1. Ozone Response Matrix

	FEV_1 Decrement = 10%		Ozone Concentration (ppb)									
			Time (hrs)									
Source	Population & Exercise	V_E (L/min)	1	2	3	4	5	6	7	8	12	24
EPA	Sedentary Child	**5**	3219	1609	1073	805	899	899	770	674	449	225
EPA	Sedentary Adult	**5**	2915	1458	972	729	814	814	698	610	407	203
EPA	Light Int Child	**11**	1405	702	468	351	392	392	336	294	196	98
EPA	Light Int Adult	**12**	1288	644	429	322	359	359	308	270	180	90
TCEQ	General Pop (24 hr)	**14**	1104	552	368	276	308	308	264	231	154	77
Samet	Child Outdoor Play	**16**	966	483	322	241	270	270	231	202	135	67
EPA	Med Int Child	**22**	702	351	234	176	196	196	168	147	98	49
TCEQ	Adult Worker (8 hr)	**22**	702	351	234	176	196	196	168	147	98	49
Zuurbier	Adult Bicycle Commute	**24**	657	329	219	164	184	184	157	138	92	46
EPA	Med Int Adult	**26**	594	297	198	149	166	166	142	124	83	41
Samet	Child Bicycling	**27**	572	286	191	143	160	160	137	120	80	40
EPA	High Int Child	**42**	368	184	123	92	103	103	88	77	51	26
EPA	High Int Adult	**50**	309	155	103	77	86	86	74	65	43	22
Samet	Adult Male Bicycling	**65**	238	119	79	59	66	66	57	50	33	17

Notes: Concentrations of ozone at which a population would be expected to experience an FEV1 decrement of 10%, given different exposure times and ventilation rates (VE - ie. exercise levels). The highlighted eight-hour time point is the averaging time of the ozone NAAQS; grey numbers indicate times and ventilation rate combinations that are unlikely to occur based on physiological limitations. For times ≤ 4 hours, the short dose-response curve was used, and for times > 4 hours, the long dose-response curve was used.

Section 5. Adversity of Effects

The definition of an adverse effect is important when delineating those effects that people need to be protected from, as opposed to those that they do not need to be protected from. For example, a biological effect to a stimulus such as ozone may be adaptive, and adaptive responses are not defined as adverse (Goodman 2014a). The EPA should have defined adverse and

adaptive effects *a priori*, and then applied this definition consistently to clinical health effects. As it is written, the application of the term "adverse" in the PR appears to be arbitrary.

5.1 More clarity is required in the definitions of "adverse effect." In its analysis of the adversity of spirometric lung effects caused by ozone, the EPA seems to arbitrarily use two different definitions from the ATS (ATS, 2000): i) a significant decrease in FEV_1 plus a significant increase in symptoms; or ii) a shift in the population distribution such that no individual experiences a change but that the whole population could be more susceptible to further respiratory insults. These are used interchangeably throughout the document with no clarity as to which one is being used at any one time. For example, at 60 ppb there is no evidence of a significant decrease in FEV_1 with a significant increase in symptoms, therefore, when the EPA (and CASAC) present this is as being adverse (US EPA 2014a, pg. 75297), they must be referring to the change in population distribution. However, it is unclear how to apply a definition of adversity that does not involve any of the population showing an effect, and because the EPA does not provide any guidance in the use of this difficult-to-define concept of adversity, they should have just used the first ATS definition. The proposal quotes CASAC's recommendation that a 60 ppb benchmark is appropriate to protect against for sensitive populations (US EPA 2014a, pg. 75288), but it is not clear why this is, since the definition of adversity used by CASAC (significant FEV_1 decrement plus symptoms) was not met at this exposure for healthy adults, and other studies have shown that healthy adults accurately represent asthmatic and child sub-populations (evidence described in sections 3.1 and 3.4). The EPA failed to use a clear, consistent definition of adversity to judge the levels at which lung function effects are likely to occur in the population.

5.2 There is no clear justification of the choice of an FEV_1 decrement of 10% as being adverse. In this ozone review, the EPA chose an FEV_1 decrement of 10% as their cut-off for adversity (US EPA 2014b). A study of exercise-induced bronchoconstriction (EIB) is cited as justification (Dryden 2010), but it is not at all clear how appropriate EIB is for establishing an ozone-induced adversity cut-off. The EPA states that the 10% cut-off is an appropriate threshold for those with lung disease such as asthma or COPD, but it applies the 10% cut-off to the entire population for expected FEV_1 decrements (US EPA 2014a, pg. 75275). The FEV_1 decrement of 10% should not be modeled in healthy children, and by applying an inappropriate FEV_1 decrement cut-off, the EPA is misleading the readers of this document, as well as the Administrator.

5.3 All inflammation is not adverse. Another clinical endpoint of interest is inflammatory cell infiltration into respiratory fluid upon ozone exposure in human clinical studies. This has been observed to occur after ozone exposure at levels down to 60 ppb (in sputum samples, Kim 2011). However, the EPA does not provide evidence to support this as an adverse effect. The PR states that "the initiation of inflammation can be considered as evidence that injury has occurred." (US EPA 2014a pg. 75264) However, this does not take into account the difference between adaptive and adverse responses, and the ATS committee cautions that "not all changes in biomarkers related to air pollution should be considered as indicative of injury that represents adverse effect." (ATS 2000) Inflammation can be a physiologically beneficial process and the EPA inappropriately over-simplifies by stating that all inflammation is adverse.

In conclusion, the EPA should have chosen and justified a level of adversity for each endpoint of interest, and this definition of adversity should have been used consistently throughout the documents and the decision-making processes. The choice of a 10% FEV_1 decrement as an adverse effect threshold in at-risk populations should have been thoroughly justified, and because it only applies to at-risk populations, 10% FEV_1 decrements should not have been modeled in healthy populations. In addition, the adaptive versus adverse responses for other

endpoints should have been defined, and the EPA should not have assumed that any response is an adverse, clinically meaningful response.

Section 6. Animal Toxicology Studies/Mode of Action

Animal toxicology studies are a key evidence stream for understanding the biological effects of ozone and for determining the biological plausibility of the health effect associations found in epidemiology studies. When translating animal studies to humans, understanding the dose is very important. Effects may be seen at high doses in animals, which through the toxicological principles of dose-dependent transitions in mechanisms of toxicity (Slikker 2004) may have no relevance to the effects seen at ambient concentrations. Therefore, the highest certainty is obtained only when drawing conclusions using studies where the animal doses are comparable to potential human exposures.

6.1 Human and animal tissue doses of ozone are similar with similar exposures. There are three factors that affect dose: concentration, time of exposure, and ventilation. Translation of doses from animals to humans can also vary. According to toxicology principles of allometry (US EPA 1994), we would expect that rats and humans exposed for the same time and ventilation rate to the same concentration of ozone would have approximately similar tissue doses. However, the parts of the respiratory tract that receive the dose may differ because humans are oro-nasal breathers and rodents are obligate nasal breathers. The idea that rats and humans would expect to get similar ozone doses with similar exposures was confused by Hatch et al in 1994, when they published that humans received five times the dose as rats when exposed at the same concentration and for the same time. However, in that experiment the rats were at rest and the humans were exercising intermittently at a high ventilation rate (65 L/min) for two hours. It is not clear that the toxicology community was explicitly aware of this important difference because of papers (even recent papers) published that cite Hatch 1994, claiming that high doses (>500 ppb) were environmentally relevant because of the reported five-fold difference in tissue dose (Zhao 1998, Vella 2014, Martinez-Lazcano 2013, Theis 2014). In their 1994 paper, Hatch hypothesized that the difference between rat and human tissue doses in their experiment was indeed the exercise (and therefore the ventilation) difference between the two species, and they confirmed this hypothesis in 2013 (Hatch 2013). They showed that exercising humans (exercising intermittently at 65 L/min for two hours) had five times the inhaled dose, and five times the tissue dose as humans who were at rest, and that resting humans and resting rats received similar tissue doses when exposed to the same ozone concentrations at the same time and ventilations. The Hatch 1994 study was also interpreted incorrectly in this proposal (US EPA 2014a, pg. 75255), with the statement that “even very high ozone concentrations in rodents could be equivalent to much lower exposure concentrations in humans.” This must be considered very carefully in terms of the comparability of the entirety of the animal exposure dose (time, concentration and ventilation) to the entirety of the human dose. These concepts can be used to understand animal toxicology data and to apply it to actual human exposures.

6.2 Many animal doses are non-informative for human exposures. The results from many toxicology studies are mentioned in the ozone proposal describing ozone-attributable effects on the respiratory, cardiovascular, nervous, hepatic, and reproductive systems (US EPA 2014a, pg. 75247). However as stated above, information about the doses (ie. concentrations, time of exposure and ventilation) at which these effects occur is required to draw conclusions about their applicability to current human exposures. Most of the cited studies expose animals either at concentrations well above those experienced by humans or with dosing schedules that are similarly inappropriate (US EPA 2013, pg. 6-209, 7-24). This information is therefore not informative as to whether an effect occurs in a human at a much lower dose than the animal. The EPA did not derive a table showing plausible human doses and comparable animal doses to

allow direct comparisons to help the Administrator use this data to make an informed policy decision. A good example is the endpoint of chronic damage to the lungs caused by long-term ozone exposure (US EPA 2014a, pg. 75267). The studies used to draw these conclusions expose infant monkeys to 500 ppb ozone for eight hours per day for five days, followed by five – nine days off, and then the exposure is repeated for up to five months (US EPA 2013, Table 7-1). Assuming that the infant monkeys were at rest during exposure, then to achieve an equivalent dose a human child would have to be outdoors, moderately exercising continuously for 8 hours a day, five days a week at an eight-hour average of ~114 ppb. These ozone concentrations no longer exist in the United States and certainly won't exist when the current standard of 75 ppb is attained. Because many of these studies did not provide vital information about the ventilation of the animals being tested, the EPA is inappropriately drawing conclusions about levels of ozone that cause relevant health effects. As such, the EPA should have used only those animal toxicology studies that are done at equivalent or near equivalent ambient doses (and who are explicit about the dose, which is not only concentration and time, but also ventilation) as proof of the potential for a health effect in current ambient ozone-exposed humans.

In conclusion, the EPA did not correctly consider dose differences between relevant human exposures and the animal toxicology studies before extrapolating from animals to potential human impacts. Because most of the animal studies used by the EPA were conducted at inappropriately high doses, they are of limited utility in providing causal information to inform human health effects and policy decisions, and it is irrational for the EPA to credit them with anything more than minimal weight in assessing the potential risks to human health or public welfare.

Section 7. Risk and Exposure Assessment

7.1 Urban disbenefits are caused by reducing NOx and ozone in urban core areas.
The EPA uses the higher order direct decoupled method (HDDM) to model the reduction of ambient ozone concentrations from known 2007 or 2009 levels to levels that would attain the current standard or the alternative standards. Included in this model is NO_X scavenging, which means that if NO_X decreases at the source, then ozone will increase close to the source, and will decrease farther away. Because many of the NO_X sources are within urban areas (automobile traffic, etc.), decreasing urban NOx will lead to more of a decrease in median ozone levels in suburban and rural areas than in urban areas (US EPA 2014a, pg. 75277). This explains why some of the study areas where ozone was modeled to attain alternative standards, including Houston and Los Angeles, actually showed *increases in mortality*, not decreases (US EPA 2014b Figure 7B-1 and 7B-2). Because of this, suburban and rural areas will gain more benefits of decreasing ozone, while in the urban areas there will be fewer benefits. This is demonstrated by the comparison of urban and non-urban exposures of concern and FEV_1 decrements shown in Appendix 9 of the HREA (US EPA 2014b, Figures 9A-1 to 9A-24) – as the ozone levels are pushed lower, the discrepancy in ozone exposures between urban core and non-urban areas becomes greater (Figure 3). Importantly, the urban areas will bear the brunt of the costs because they will be in nonattainment. This type of discrepancy between those who benefit and those who pay could be an environmental justice issue, and as such, should be carefully considered by the Administrator if she decides to change the level of the ozone NAAQS.

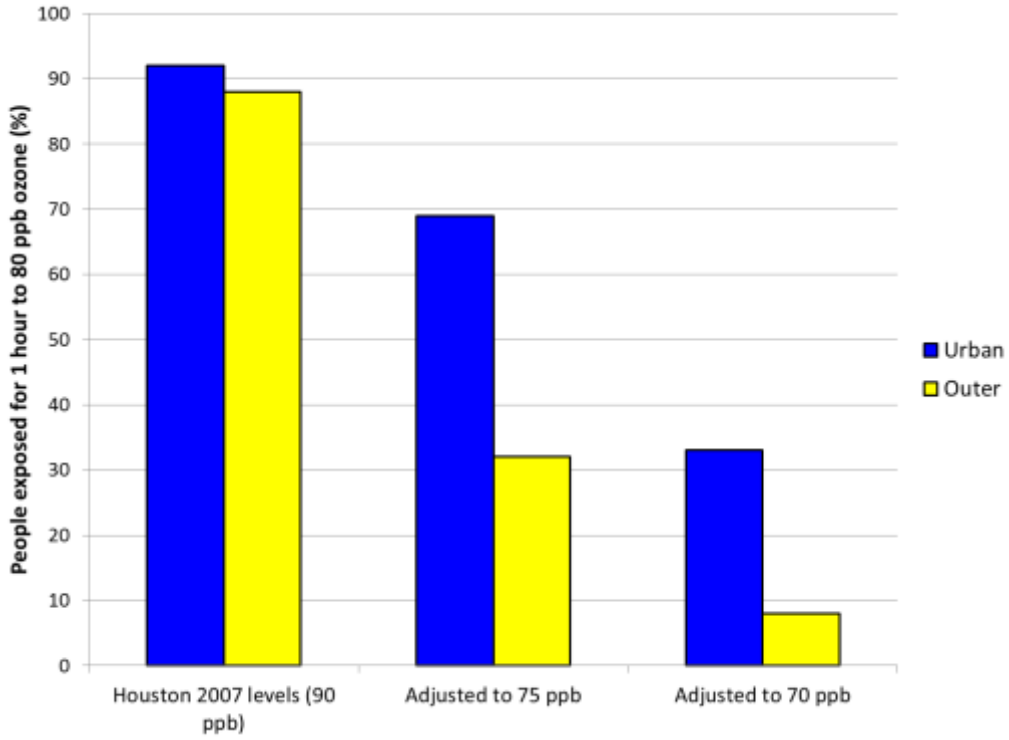


Figure 3. Exposure to 80 ppb ozone for one hour in the Houston urban core vs. outer areas

Estimate of the percent of people exposed to 80 ppb ozone for one hour at 2007 levels, and when air quality is adjusted to 75 ppb and 70 ppb standard levels, in the city's urban core and outer areas. Based on estimates from US EPA 2014b, Appendix 9 Figure 9A-7.

7.2 There are critical flaws in the EPA's linear no-threshold model of ozone and mortality. The EPA's modeling of mortality caused by decreasing ozone concentrations incorrectly used a linear no-threshold model to ascribe premature mortality to ozone levels. This model assumes a linear relationship between ozone levels and mortality, which is summarized by the EPA as"total risk estimates are equally influenced by decreases in high ozone concentrations and increases in low ozone concentrations when the increases and decreases are of equal magnitude" (US EPA 2014a pg. 75278). The EPA is saying that if the ambient ozone distribution decreased all the days spent at 80 ppb to 70 ppb, and at the same time similarly increased 20 ppb days to 30 ppb days, then the changes in mortality would cancel out. Therefore, the EPA is ascribing equal ability of 30 ppb and 80 ppb ozone to cause mortality, and it fails to explain how this could be the case. In so doing, the EPA is ignoring the fundamental concept of dose-response and this demonstrates a crucial flaw in the EPA's choice of models. Using this linear no-threshold model, the EPA estimates the mortality of people in the study areas with decreasing levels of ozone. This modeling results in estimated **increases** in mortality in two to six of the study areas when ozone is decreased from baseline 2007 or 2009 levels to 75 ppb (US EPA 2014b, Tables 7B-1 and 7B-2) and clearly shows that this model is flawed.

7.3 The HREA estimates more FEV_1 decrements than exposures of concern. Using the HDDM model to estimate decreases in ozone levels, the EPA also estimates the number of people (and the PR focuses on children) at exposures of concern of 60, 70, or 80 ppb ozone, as well as the number of people expected to experience FEV_1 decrements of <10%, <15%, or <20%. However, there is a discrepancy between the FEV_1 and the exposure of concern risk estimates (presented on pp. 75272, 73 & 75 of US EPA 2014a). For example, at a modeled standard of 60 ppb, 70,000 children are predicted to be exposed to a benchmark concentration of ≥ 60 ppb one

or more times. Yet, also at a modeled standard of 60 ppb, 1.4 million children are predicted to experience at least one FEV_1 decrement of ≥ 10%. Even more remarkably, at a modeled standard of 60 ppb, 57,000 children are predicted to experience FEV_1 decrements of ≥ 20% - this appears to be equivalent to ~81% of the children exposed to ≥ 60 ppb experiencing FEV_1 decrements ≥ 20%. This is not consistent with the data that the EPA presents, which shows that only ~9% and ~0.8% of people exposed to ozone at 60 ppb would experience FEV1 decrements of ≥ 10% or ≥ 20%, respectively (US EPA 2014b, Table 6-3). It is also not consistent with the EPA's own statement saying that "only a subset of individuals who experience exposures at or above a benchmark concentration can be expected to experience health effects." (US EPA 2014a pg. 75273). If it is true that most people at an exposure of concern won't experience an FEV_1 decrement, then there should be more people at the exposures of concern than those experiencing FEV_1 decrements, not the other way around. The EPA fails to explain this discrepancy. It perhaps could be explained by the use of the MSS model, which includes not only exposures of eight hours at moderate exercise, but also shorter exposures at different exercise levels (as suggested on pg. 75251 of US EPA 2014a). However, the EPA does not make that clear, and no numbers for non-eight hour, moderate exercise exposures are presented so that the Administrator can review the accuracy of the data and the assumptions. A reader who had not dug into the depths of the ozone documents would be egregiously misled by the EPA into thinking that 1.4 million children could be affected by 70,000 exposures to 60 ppb.

7.4 Confidence intervals should be presented with the risk estimate data. For the overall estimates of exposures of concern and FEV_1 decrements, the EPA acknowledges that there is substantial variability in these numbers (US EPA 2014a, pg. 75274), but no confidence intervals are provided in the PR – this is misleading and unscientific. The same is true for the estimates of mortality, and given the EPA's uncertainty in the epidemiology estimates, it is even more important to have confidence intervals presented with that data. This is particularly true because for eight out of 12 of the study area estimates, the confidence intervals included zero when adjusting down to 75 ppb (US EPA 2014b, Tables 7B-1 and 7B-2). Of the four cities with confidence intervals that did not include zero in the 2009 estimates, all four of them had **increases** in mortality when going from the base case to 75 ppb (except Detroit, where no estimate was given for 75 ppb, but Detroit had increases in mortality going down to 70 ppb and 65 ppb in 2009). This is a strong reminder of the uncertainty in the mortality data that the EPA should have discussed and explained in the PR, so that the Administrator could assess the confidence in these estimates. One explanation of the counter-intuitive nature of these risk analysis results is that the epidemiology studies themselves are inappropriately assigning causation of ozone to these premature deaths (as we discuss elsewhere in this document). Because of this, even if the Administrator focuses on the deaths attributed to occur above 40 ppb or 60 ppb, that data is equally flawed and likely also has confidence intervals that approach or include zero.

In conclusion, the TCEQ is confused as to why the EPA is not very concerned that if it decreases the level of the ozone standard, the EPA's own modeling shows that the greater benefit of ozone decreases will be in suburban and rural areas, whereas the greater costs associated with nonattainment would be expected to occur in the urban areas. This may qualify as an environmental justice issue that the EPA failed to address. In addition, the EPA has incorrectly modeled the exposure and lung function data, such that there are more FEV_1 decrements than there are exposures. This should have been clarified and the data explaining how these numbers were derived should have been produced by the EPA. Similarly, because of a linear no-threshold model, the EPA is predicting an increase in deaths with decreases in ozone in several cities, which demonstrates a flaw with their model that should have been addressed before the EPA used this model as the basis for drawing its conclusions about mortality or calculating benefits. For all of the exposures of concern, FEV_1 decrements and mortality estimates, the EPA does not

present any confidence intervals, which misrepresents the uncertainties in the data and omits necessary information for the Administrator to make decisions about whether there are going to be significant health benefits from reducing the level of the ozone NAAQS.

Section 8. Threshold

In its risk analysis, the EPA should have used a threshold of response. Animal and human toxicology studies strongly support the concept of a threshold for ozone-mediated health effects (US EPA 2013, chapter 5). As the EPA itself has thoroughly reviewed, the epithelial lining fluid of the respiratory tract contains antioxidants that can scavenge and inactivate ozone. These antioxidants are replenished, such that the slower the exposure, the lower the respiratory effects (Schelegle 2007).

8.1 Human clinical data supports a threshold of effects with ozone exposure. The presence of a threshold is consistent with the ozone clinical data, which shows that at low exposures there are no health effects that are distinguishable from filtered air exposure (eg. 40 ppb for 6.6 hours with exercise). Despite the fact that exposure to 40 ppb ozone under these conditions does not cause any respiratory effects (in fact, FEV_1 *increases* compared to baseline), the EPA illogically states that FEV_1 decrements result from days with ozone concentrations above about 40 ppb (US EPA 2014a, pg. 75274). This is not consistent with the EPA's own statement that there are no significant health effects at this ozone dose (US EPA 2014a, pg. 75249). The EPA notes that there is a smooth dose-response curve with no threshold between 40 and 120 ppb (US EPA 2014a, pg. 75249), but again, this is contrary to thefact that no effect has been observed with exposure to 40 ppb. If there is no effect at 40 ppb, and some effect at 60 ppb, then included in the model there should be an effect threshold between 40 and 60 ppb. Additionally, the MSS model made by McDonnell et al and used by the EPA modeled a threshold (McDonnell 2012), which appears to have been lost in this analysis. Not only does the spirometric data support a threshold of effect, but so too does the data for ozone-induced infiltration of inflammatory cells into the bronchiolalveolar (BAL) fluid. Mudway et al. (2004) reviewed many studies investigating the presence of neutrophils in BAL fluid, and they found a significant dose-response with a threshold of 645 mg/m^2 for a response at three to six hours after exposure, and a threshold of 810 mg/m^2 for a response at 18 to 24 hours after exposure.

8.2 A no-threshold model for the ozone epidemiology data is inconsistent with the known ozone mechanism of action. As discussed above, the toxicology and human clinical data both support a threshold mechanism of action for ozone, which is consistent with the general principle that non-carcinogenic chemicals have thresholds of effect. Despite this solid, well-acknowledged scientific evidence, the EPA states that there isn't a population level threshold below which it can be confident that ozone-attributable effects don't occur (US EPA 2014a, pg. 75244). This is an irrational conclusion given the other overwhelming evidence streams. One potential explanation for the inability for the epidemiology studies to correctly identify a threshold is given in the PR itself, which is that in multi-city studies, regional heterogeneity can obscure a threshold, if one exists (US EPA 2014a, pg. 75282). Since these are the studies that are often used to investigate the existence of a threshold, it suggests a plausible reason why a population threshold hasn't been found. At least one single-city epidemiology study has found evidence for effect thresholds (Atkinson 2012). In addition, Rhomberg (2011) found that the presence of exposure measurement error can linearize concentration-response functions, which can obscure the presence of a threshold and overestimate the risks at lower exposures. Alternatively, the inability to identify a population-level threshold may demonstrate that, because of the known flaws in the epidemiology studies (exposure measurement error, bias, confounders), the mortality that is being attributed to ozone may in fact be caused by something else. The TCEQ applauds the EPA in their consideration of a threshold of effects in the long-term respiratory mortality data based on Jerret 2009. We note that when the best-fit

threshold (56 ppb) is considered, the long-term mortality estimates decrease by 98% (US EPA 2014b, Figure 7-9). By not modeling a threshold for the epidemiologically-based health effects, the EPA has unscientifically inflated the health effects of lowering the ozone standard, and this inflation is translated into the benefits calculations.

In conclusion, the EPA failed to consider a threshold of effect for ozone despite very strong scientific evidence from both mechanistic toxicology data and human clinical data that a threshold of effects exists for ozone exposure. The TCEQ asserts that an appropriate threshold of effects should be considered when modeling FEV_1 decrements (ie. a threshold at > 60 ppb) and that the EPA incorrectly uses epidemiology studies and C-R functions that show no threshold in mortality and morbidity estimates.

Section 9. Evidence Integration

The EPA states that there is a causal association between short-term ozone exposure and respiratory effects, and a likely causal association between long-term exposure and respiratory effects (the latter is based on animal toxicology studies and epidemiology studies; US EPA 2014a, pg. 75247). To reach these causal conclusions, the EPA assesses the health effects measured by epidemiology studies in the context of the extent and dose at which ozone effects occur in clinical studies and animal toxicology studies (US EPA 2014a, pg. 75245). The TCEQ agrees that it is appropriate to consider all of these health endpoints together, and to draw conclusions based on how all of the evidence streams integrate with one another. However, the EPA did not adequately integrate *all of the evidence* in their assessment of ozone health effects.

9.1 Exhaled nitric oxide (eNO) is not a good biomarker for increased lung oxidant responses. The ozone clinical data can be used to inform the plausibility of the epidemiology endpoints. For example, the EPA states that epidemiology studies have shown that people have increased oxidants in their lungs with increased exposure to ambient ozone by measuring eNO (US EPA 2014a, pg. 75253), but human clinical studies have shown at doses up to 200 ppb that ozone exposure does not cause a change in eNO exhalation (Newson 2000, Nightingale 1999). The EPA did not appropriately consider the human clinical data to inform the epidemiology results.

9.2 The human clinical data does not support ozone-induced mortality at ambient ozone concentrations. The EPA concludes from the ozone epidemiology studies that ambient ozone concentrations can cause respiratory morbidity as evidenced by HA and ED visits, and it can cause total and respiratory mortality. The effect estimates from these studies are small, so it is very important for the results to be biological plausible before drawing conclusions and making policy based on them. From a toxicological point of view, the best way to look at all of this data together is to consider the doses at which it occurs. Beginning with the clinical data, we know that when people are exposed to ozone at ambient concentrations (ie. 40 ppb – 120 ppb) for ~eight hours at moderate to vigorous physical exertion, a range of respiratory effects occur, including FEV_1 decrements, symptoms, pulmonary inflammation, airway hyper-responsiveness, and epithelial permeability. These are mild to moderate health effects, and in the human clinical studies, have always been fully reversible at these doses. In fact, the only way an Institutional Review Board will approve these clinical studies is if the clinicians are certain the participants will not be harmed. When considering dose in the epidemiology data, the EPA should have considered the assumptions made in those studies – that is, that the people in the studies are exposed to ambient concentrations of ozone all the time. This is not consistent with what is known about the micro-environmental dependence of ozone (e.g. very low levels inside, where people spend most of their time), and so should be considered when calculating dose. According to studies that measure personal exposure (Lee 2004, Sarnat 2001, Sarnat 2005), exposure can be 10-20% of the ambient concentrations. Therefore, when ambient concentrations are at 40-120 ppb, then personal exposure of the

people in the study is in the range of 4 – 24 ppb. Assuming that those people are exercising at the same level as in the clinical studies, this still puts the dose at levels far below those that cause health effects in the clinical studies. Even if you consider that the clinical studies group is young and healthy (although also exercising vigorously and therefore getting a higher dose), and the people in the mortality studies are near-death, it does not make defensible, scientific sense that a concentration that only causes a mild, reversible effect, or no effect at all, also causes death. The morbidity epidemiology data ascribes these same ambient concentrations of ozone (< 40 ppb to > 120 ppb) to asthma hospitalizations and ED visits. It is possible that the health effects shown to occur at these concentrations (assuming an outdoor exposure time of ~ eight hours with physical exercise for most of that time) could lead to ED visits, but hospitalization is a more severe effect that is therefore less plausible at these doses. For the short-term mortality data, it is even less likely that in the days before a person's death, they were constantly exposed to ambient levels of ozone while exercising vigorously enough to elicit even one of the mild health responses measured in the clinical studies, let alone death. In fact, there is no known toxicant that causes mild reversible effects at the same doses at which it causes death. Integrating these streams of evidence – human clinical studies, epidemiology studie, and personal exposure studies – shows that the EPA's likely causal association of ozone with mortality is incorrect and scientifically unsound, and the causal association with morbidity is implausible at the measured doses. This is supported by the comments made by CASAC in 2006 (EPA-CASAC-06-007, June 5 2006):

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.

9.3 The toxicology data does not support the ozone-induced health effects shown by epidemiology studies. Another important stream of evidence that the EPA failed to appropriately integrate is the toxicology data, which is mostly provided by animal studies. A number of places in the PR (US EPA 2014a, pp. 75267, 75288, etc.) cite the animal toxicology data as being proof that a plausible mechanism of action exists for a given endpoint. These studies provide mode of action information for ozone and are important for understanding how ozone causes these health effects. Many respiratory and systemic effects have been caused by ozone in animal toxicology studies (US EPA 2014a, pg. 75247), and these are used as evidence that ozone can cause systemic effects, including mortality, in humans at ambient levels. However, again, the dose is important here and was not considered by the EPA. As is discussed in section 6.2 of these comments, the doses of ozone applied to animals are often much higher than those that are environmentally relevant. According to the concept of dose-dependent transitions in mechanisms of toxicity, higher doses of a toxicant can cause effects that are not necessarily relevant to the effects that occur at lower doses. As it pertains to informing the epidemiology data, the doses of ozone required to kill animals is orders of magnitude higher than ambient ozone (the National Institutes for Occupational Safety and Health (NIOSH) Immediately Dangerous to Life of Health value for ozone is 5 *ppm*; NIOSH 2005), so again does not support the EPA's assertion that there is a mechanism for ambient ozone to contribute to mortality. Given the plethora of data in animal and human studies, the EPA should have produced a plausible, dose-driven step-by-step mechanism of ozone effects from low dose to high dose and then should have used this to inform the doses at which the moderate and severe responses measured in the epi studies are most likely to occur. The EPA's ozone documents fail to specify the mechanism by which ozone at ambient levels can cause premature mortality leaving the Administrator to speculate about the way in which this important endpoint could occur. The EPA does state that because ozone affects multiple pathways, this is a plausible

mechanism that could lead to mortality (US EPA 2014a, pg. 75258). However, this argument leaves out the important details about how this could *actually happen*. The need to assess epidemiology studies in the context of animal studies also goes the other way: it is important for the EPA to consider the results from the animal studies in the context of what is known to affect humans. For example, the endpoint of chronic damage to the lungs caused by long-term ozone exposure (US EPA 2014a, pg. 75267) is supported by studies in non-human primates (US EPA 2013, Table 7-1). However, those studies were undertaken using doses that are not applicable to humans, as explained in section 6.2. In addition, there is little evidence that long-term exposure to ambient ozone is associated with significant deficits in the growth rate and lung function in children, from studies done by Gauderman et al (2004) using the Children's Health Study cohort. This study was very recently updated and shows the same result: that ozone does not affect lung development in children (Gauderman 2015).

9.4 A systematic review is the best method for carrying out evidence integration. The EPA should have, but failed to, use a more appropriate method for appraising the coherence of the ozone epidemiology data by using a formal systematic review framework. This method is encouraged by the National Research Council (NRC 2011, NRC 2014), and a number of good frameworks have been published (Adami 2011, Suter 2011, Rhomberg 2013). This systematic review process has been applied to the cardiovascular endpoint, both after short-term or long-term ozone exposure (Goodman 2014b, Prueitt 2014). In both cases, the conclusion drawn for the causal relationship was "below equipoise," which means that the evidence is insufficient to conclude that a causal relationship exists. This type of framework is used by the EPA in the NOx ISA (US EPA 2015, Table 5-1), and it should have been used for ozone. This analysis involves a step-by-step, unbiased, transparent review of the available data, including appraising the quality of the study and drawing conclusion based on study quality and consistency of results, not based on which studies show a particular result.

In conclusion, for the EPA to adequately draw conclusions about ozone-induced health effects, it was imperative that it integrate all of the evidence streams, including animal toxicology, human clinical, and epidemiology data. The preferred method for this type of evidence integration is the systematic review, which is endorsed by the NRC. If the EPA had considered all of the evidence streams together, it would have concluded that the animal toxicology and human clinical data do not support the epidemiological conclusion that ozone causes mortality at ambient concentrations and exposures.

Section 10. Dose Response Analysis

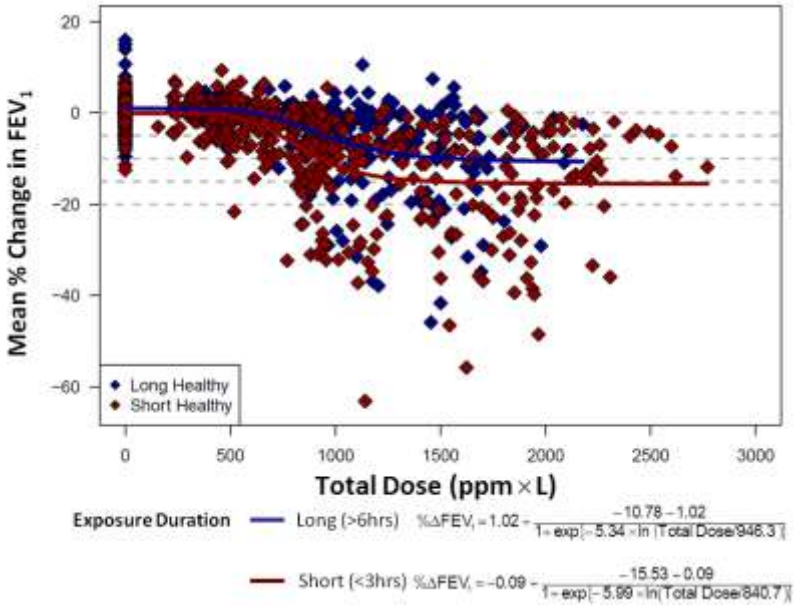
10.1 The EPA should have analyzed the thresholds at which there is no FEV1 response, and thresholds below which there is no adverse FEV1 response. The EPA should have used these thresholds, coupled with real-world exposure times and ventilation rates, to present ozone concentrations at or below which no adverse FEV1 response would be expected to occur. In this section TCEQ describes its' own analysis of the ozone clinical data (referred to in sections 3 and 4 of these comments). This analysis presents a dose-response model that fits both healthy young adults and other populations, such as children and asthmatics. The TCEQ used these thresholds to determine at what concentrations of ozone (assuming different exposure times and ventilation or exercise rates) a certain FEV1 response would be expected to occur. This type of simple but powerful analysis should have been done by the EPA to transparently discuss how the human clinical data can inform the level of the ozone standard.

In order to understand the dose-response relationship between ozone and FEV1, we plotted total inhaled dose of ozone (which is calculated from ozone concentration, time of exposure and ventilation rate) versus FEV1 decrement. The main analysis was done with the EPA dataset of 541 individuals (McDonnell 2007).

The data were divided into two exposure categories: < 3 hours and 6 - 8 hours, to determine if the time it takes to achieve the dose affects the dose-response curve. The zero ozone dose (called filtered air in the experiments) was also plotted on the graphs, and was not subtracted from the ozone exposure data.

There were two distinct sigmoidal-shaped curves for the short and long exposure times (Figure 4A, B). These two curves were statistically significantly different from one another ($p < 0.0001$).

A



B

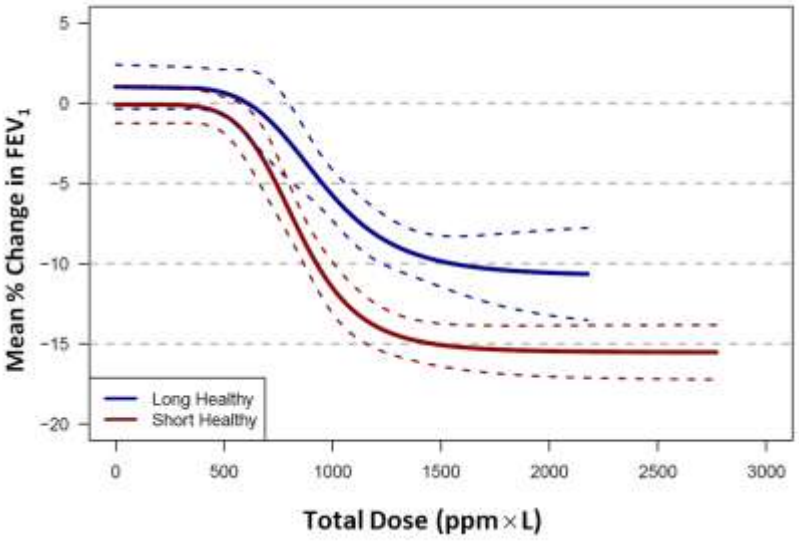


Figure 4. Ozone-FEV_1 dose-response curves

(**A**) Plot of total inhaled dose (in ppm x L) versus mean percent change in forced expiratory volume in 1 second (FEV_1) of healthy young adults from the US EPA individual dataset exposed for ≤ 3 hours (short exposure, red diamonds and trend line) or 6 – 8 hours (long exposure, blue diamonds and trend line) to ozone while exercising, with the equations associated with each curve below the graph; (**B**) The trend lines from A with 95% confidence intervals (dashed lines).

Using the equations of the dose-response curves, we could calculate the dose at which a certain mean FEV1 response would be expected to occur, and the results are summarized in Table 2. No doses were derived for a 15% FEV1 decrement from the long exposure curve, because that curve never crossed 15%. Similarly, no 0% FEV1 decrement dose was derived for the individual short exposure, because that curve was never at or above 0.

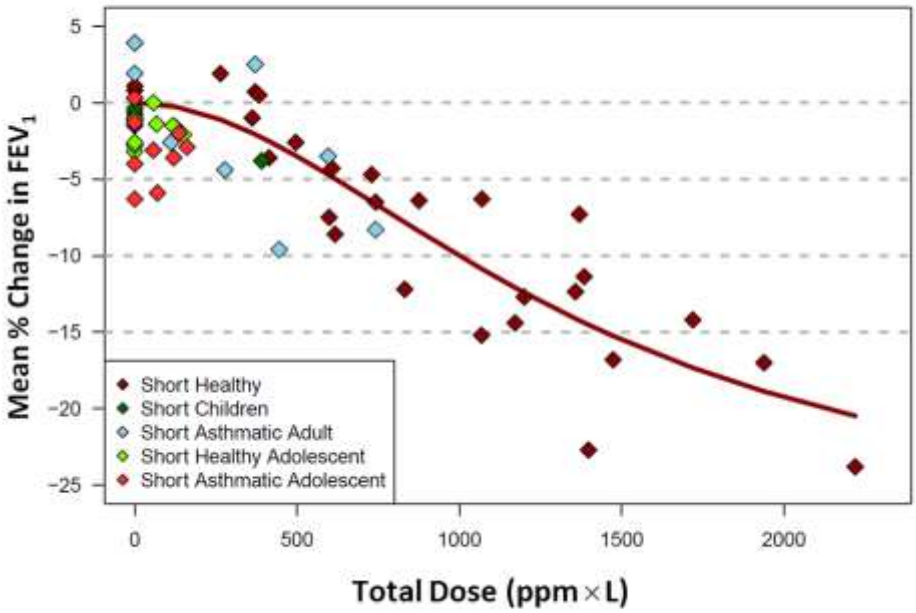
Table 2: Total doses to produce a mean FEV_1 response

Mean % ΔFEV1	Long Exposure Dose (ppm x L)	Short Exposure Dose (ppm x L)
0	608.5	N/A
- 5	953.5	740.2
- 10	1553.8	926.7
- 15	N/A	1467.4

Our initial ozone-FEV1 dose-response analyses were done using data from healthy young adults (18-35 years old). We did an additional analysis using group mean data (see reference list for studies used for group mean dose-response), so that we could compare responses of young healthy adults to the responses of children and asthmatics, who are potential at-risk populations for ozone exposure.

We plotted the adult asthmatic group mean FEV1 response on the short and long healthy ozone exposure curves and found that for both exposures, the asthmatic data points showed both increased and decreased responses, compared to healthy young adults (Figure 5 A,B). Overall, this suggests that asthmatics do not demonstrate increased spirometric responses to ozone, which is consistent with the conclusions reported from many studies (Linn 1994, Balmes 1997, Koenig 1985, Koenig 1987, Stenfors 2002, Nightingale 1999, Basha 1994). There is also data investigating the effects of short-term ozone exposure on healthy and asthmatics adolescents (aged 11 to 18 years old). These studies were done at quite low doses, making it difficult to derive a dose-response relationship. Generally however, the responses between the healthy and asthmatic adolescents were similar (Figure 5B). The group mean response for children was consistent with the dose-response plotted for the adult group mean (Figure 5B). This data all shows that children and asthmatics have similar FEV1 responses to ozone as healthy young adults.

A



B

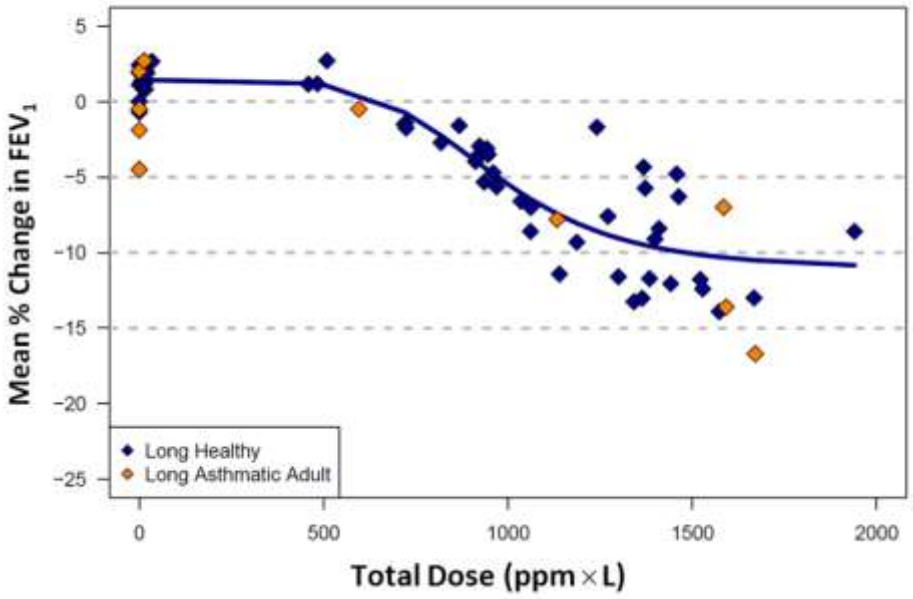


Figure 5. Ozone-FEV_1 dose-response curves that include subpopulations.
(**A**) Plot of group mean data for total inhaled dose versus mean percent change in FEV_1 of healthy young adults (dark red diamonds and trend line) exposed for ≤ 3 hours to ozone with 95% confidence intervals, and also plotted is the group mean exposure data points from children aged 8-11 exposed to ozone (dark green diamonds), from adult asthmatics (blue diamonds), from healthy adolescents (light green diamonds) and from asthmatic adolescents (bright red diamonds); (**B**) Plot of group mean data for total inhaled dose versus mean percent change in FEV_1 of healthy young adults (blue diamonds and trend lines) exposed for 6-8 hours to ozone with 95% confidence intervals, and also plotted is the group mean exposure data from adult asthmatics (yellow diamonds);

The threshold doses in Table 2 are a combination of time, ventilation and ozone concentration, whereas the ozone standard level is just a concentration. Therefore, we have to consider different times and ventilations at which a particular FEV1 response occurs, and then make assumptions about reasonable, real world exposure times and ventilations in order to calculate a protective ozone concentration.

For the time variable, the current standard is an 8 hour maximum average, and previous standards have been 1 hour maximums. So these are some appropriate exposure times for dose calculations.

The EPA has two standard numbers they use for ventilation in risk calculations, which is 20 m3 per day (24 hours) for the general public (this assumes that 8 hours are spent at a worker ventilation), or 10 m3 per 8 hours for an outdoor worker (US EPA 1994). Ventilation rates associated with particular exercises have also been measured experimentally (Zuurbier 2003, Samet 1993).

Using a time scale of exposure from 1 to 24 hours, we used the short and long dose-response thresholds and various relevant ventilations to calculate at what dose of ozone (for that time and ventilation) a given FEV1 mean response would be predicted to occur. For times less than or equal to 4 hours, we used the short dose-response thresholds, and for times greater than 4 hours, we used the long dose-response thresholds. We produced a matrix that demonstrates the ozone concentrations at which a mean FEV1 decrement of 10% would be expected to occur, given these different ventilations and exposure times (Table 1). From this data, one can see that, for the general public 24 hour outdoor exposure at 20 m3/day (14 L/min), an average ozone concentration of 77 ppb would be required to cause an FEV1 decrement of 10%.

These matrices can be narrowed down to only include the 8 hour exposure time, since that is averaging time of the standard. This is shown in Table 3. For the manual labor ventilation (22 L/min for 8 hours), an ozone concentration of 147 ppb would cause an FEV1 decrement of 10%. This can be compared to current ozone air concentrations to see if these concentrations are currently occurring. For example, in Texas the monitor that had the highest 8 hour maximum concentration in 2014 was the Manvel Croix Park monitor in the Houston-Galveston-Brazoria area. This monitors' highest 8 hour max concentration was 95 ppb, its highest 1 hour ozone concentration was 135 ppb and its highest 24 hour average was 44 ppb (http://www.tceq.state.tx.us/cgi-bin/compliance/monops/ozone_summary.pl).

Table 3. Ozone Concentrations Resulting in a 10% FEV1 Decrement

Population and Exercise	Ventilation (L/min)	O3 Concentration for a -10% FEV1 (ppb)
Child, Sedentary	5	674
Adult, Sedentary	5	610
Child, Light Intensity	11	294
Adult, Light Intensity	12	270
General Population (24 hr)	14	231
Child, Outdoor Play	16	202
Child, Medium Intensity	22	147
Adult Worker (8 hr)	22	147
Adult, Bicycle Commute	24	138
Adult, Medium Intensity	26	124
Child, Bicycling	27	120
Child, High intensity	42	77
Adult, High Intensity	50	65
Adult male, vigorously bicycling	65	50

Notes: Concentration of ozone at which a population would be expected to experience an FEV_1 decrement of 10%, given an 8 hour exposure time and different ventilation rates

The presence of threshold doses at which no FEV1 response would be expected to occur (the 0% FEV1 threshold) is very consistent with the known ozone MOA, where antioxidants scavenge ozone in the epithelial lining fluid and prevent it from reacting and causing damage in the respiratory tract. Using the short exposure times, the dose for a 0% FEV1 response could not be calculated because the dose response curve was always below zero (even at a dose of zero). However, the shapes of the individual response curve shows that the curve does not deviate significantly from zero until a dose of almost 500 ppm x L. This means that the short exposure responses are also consistent with a threshold of response to ozone exposure. Other groups who have investigated dose-response curves for ozone exposure have also shown evidence of thresholds or doses of onset (McDonnell 2012, Schelegle 2012).

As mentioned above, there is published information regarding the adverse effect levels of FEV1. For FEV1, the American Thoracic Society (ATS) suggests that a significant decrease in FEV1, combined with significant symptoms, should be considered as adverse (ATS 2000). In the studies used for our analysis, the lowest dose at which a long-term exposure met this criteria was 912 ppm x L (from Adams 2002), and for a short-term exposure the lowest dose was 608 ppm x L (from McDonnell 1983). However, because an individuals' FEV1 response and their symptoms correlate poorly (Schelegle 2009, McDonnell 1999, Frampton 1997), this threshold for adversity is difficult to apply to ozone response. Alternatively, the ATS has suggested that two-point changes in FEV1 of >12% may be clinically significant (Pellegrino 2005). In their most recent ozone standard review, the EPA uses two FEV1 thresholds of adverse effects: a 10% decrement for populations with respiratory disease, and a 15% decrement for healthy

populations (US EPA 2014b). In our analysis we use the 10% FEV1 decrement threshold, in order to be more protective.

Several other papers have been published that describe the relationship between ozone dose and different health endpoints, particularly the FEV1 response (e.g. McDonnell 2007, Schelegle 2009, McDonnell 2012). The McDonnell 2012 analysis was used to derive the MSS model that the EPA relies on to model FEV1 decrements in the HREA (US EPA 2014b). Our work differs from these papers and from the MSS model in several important ways. One way is that we do not subtract the individuals' filtered air response, but rather include it as a zero dose in the dose-response curve. Including a zero-dose is a common practice in modeling dose-response and allows the inclusion of the inter-individual variability that occurs just in response to the study protocol. Another difference is that we use the entire dataset, either by including data from all of the individuals, or from the group mean response. By including the entire dataset, we are provided with a model that is more likely to represent a general population response. In addition, when other models use only the "sensitive" responders in a population, it ignores the fact that they may not consistently be responders. As noted above, we also consider the data from different subpopulations that have been experimentally exposed to ozone, to determine whether or not the healthy young adult dose-response also adequately represents these populations. Finally, unlike the other papers that have published dose-response models, we use real-world ventilations and exposure times to allow this information to be applied to the general population, which aids policy makers in making decisions.

In conclusion, we report a unique analysis that incorporates ozone exposures with group mean and individual FEV1 responses, and considers other subpopulations, in order to produce dose-response curves. Different threshold doses are then used to make a matrix of time, ventilation and ozone concentrations at which a given FEV1 decrement would be expected to occur. This provides a tool that translates ozone human clinical data into a format that can be used by policy makers to decide on a protective level for the ozone NAAQS. The results shown here demonstrate that the current ozone NAAQS level of 75 ppb is adequate to protect the population against FEV1 decrements of 10% or above.

B. Ozone Precursors

The TCEQ does not agree with the EPA's statement that methane and carbon monoxide are ozone precursors for purposes of ozone formation.

On page 79 FR 75241 of the proposal, the EPA states:

> "Ozone is formed near the Earth's surface due to chemical interactions involving solar radiation and precursor pollutants including volatile organic compounds (VOCs), nitrogen oxides (NO_X), methane (CH_4) and carbon monoxide (CO)."

Although ozone formation from methane and carbon monoxide is not zero, these two compounds are exceedingly weak ozone precursors. According to the latest calculations by Carter (2015), the maximum incremental reactivity (MIR) of methane is 0.014 grams of ozone formed from each gram of methane oxidized.[1] To put this in perspective, the MIR of ethane, one of the weakest ozone precursors among the aliphatic hydrocarbons, is 20 times higher (0.28 g O3/g ethane). For carbon monoxide, the MIR is only 0.06 g O3/g CO. The highly reactive volatile organic compounds (HRVOCs) defined in the TCEQ's Houston state implementation plan (SIP) range in MIR reactivity from 9.00 to 15.16 g O3/g VOC—factors of 643 to 1083 times higher than the reactivity of methane. Consequently, the amount of ozone formed in a city from

[1] Carter (2015), Development of an Improved Chemical Speciation Database for Processing Emissions of Volatile Organic Compounds for Air Quality Models, http://www.cert.ucr.edu/~carter/emitdb/ , College of Engineering, Center for Environmental Research and Technology, University of California, Riverside, CA; latest update January 21, 2015.

either methane or carbon monoxide emitted in or near that city is extremely small, even though the emissions can be fairly large. There simply isn't enough time for substantial amounts of ozone to form from these compounds. These facts do not support methane and carbon monoxide being listed as an important part of ozone formation, nor does the EPA's own definition of VOC, found in 40 Code of Federal Regulations §51.100(s), which specifically excludes methane and carbon monoxide as they were determined by the EPA to have negligible photochemical reactivity.

C. The Secondary Standard

The TCEQ reviewed the evidence presented in the proposal regarding revising the secondary ozone standard and concludes that it is insufficient to support lowering the secondary standard below the level set in 2008 (75 ppb). The TCEQ urges the EPA to leave the secondary standard at the 2008 level established until a more definitive relationships between ozone concentration and welfare effects is established.

The EPA is proposing revising the eight-hour secondary ozone standard and defining the necessary protection in terms of a "W126 index" in a range of 13 to 17 parts per million-hours (ppm-hours), averaged over three years. To achieve a level of protection equivalent to 13 to 17 ppm-hours based on the W126 metric, the EPA is proposing to set an eight-hour secondary standard at a level within the range of 65 to 70 ppb. In addition, the EPA is taking comment on retaining the existing eight-hour secondary standard of 75 ppb.

The Administrator found that the "…type of information most useful in informing the selection of an appropriate range of protective levels is appropriately focused on information regarding exposures and responses of sensitive trees and other native species known or anticipated to occur in protected areas such as Class I areas…," which naturally are located in rural, often mountainous areas. But, as noted in the Proposal, "a*pproximately 80 percent of the O_3 monitoring network is urban focused."* This means that the vast areas of the United States, particularly in the Mountain West, are very sparsely monitored, especially in the most remote areas.

The EPA's analysis of the various levels of the secondary standard was based on results of using an ozone model originally designed for urban applications. The model was extrapolated to remote areas using a technique (enhanced Voronoi Neighbor Averaging, or eVNA) that first interpolates observed ozone concentrations to unmonitored areas, then estimates ozone concentrations using a "gradient adjustment." Thus, the ozone concentrations used in the assessments made for the proposal are heavily influenced by observations, the vast majority of which are collected in or near urban areas. The EPA states in the proposal that *"[t]herefore, the W126 index values estimated in the rural areas in the West, Northwest, Southwest, and West North Central with few or no monitors … are more uncertain than those estimated for areas with denser monitoring"*. The EPA makes the unsupported assertion that "*… this interpolation method generally underpredicts higher 12-hour W126 exposures*." Even if true for monitors near urban areas, this assertion can hardly be supported in the remote areas considered because of the paucity of monitoring data. Consequently, the EPA's estimates of biomass loss and foliar injury resulting from adherence to various W126 thresholds are subject to large uncertainties and do not provide sufficient justification to tighten an already burdensome standard. As there is limited monitoring data available for Class 1 areas, the EPA should conduct further analysis of its model's effectiveness before using it as the basis for a revised standard. It is not appropriate to extrapolate conclusions from heavily monitored urban areas to rural areas with very limited monitor coverage, particularly since those areas may have very different characteristics related to air quality.

The TCEQ supports the EPA's conclusion that defining a separate form for the secondary standard (W126 or similar cumulative index) is unnecessary.

During the review of the proposal that established the 2008 ozone standard, the TCEQ provided comments regarding the adequacy of using the same form for the primary and secondary standard. The TCEQ continues to oppose a separate form for the secondary standard and reiterates those comments (attached as Appendix F, with additional references provided in Appendix G) for EPA's consideration in this docket. The TCEQ appreciates that the EPA appears to have taken those comments into consideration in choosing to propose the secondary standard with the same eight-hour format as the primary standard.

If a distinct W126-based secondary ozone standard is promulgated, the EPA should involve states in the development of implementation guidance, which should be a priority.

The EPA is soliciting comment on revising the secondary standard to a W126-based form, averaged over three years, at a level within the range of 13 ppm-hrs to 17 ppm-hrs. If the EPA does promulgate a distinct W126-based secondary ozone standard, the TCEQ urges the EPA to include states in developing tools to assist with implementing the secondary standard, including implementation guidance. A collaborative approach to developing implementation guidance will provide for guidance that appropriately considers the issues that states may face in implementing a distinct secondary standard.

D. Ambient Monitoring Requirements and Costs

The EPA's proposal of a range of standards, as well as ambient air monitoring network changes with an inadequate level of detail, precludes meaningful review and evaluation of potential impacts.

Ambient monitoring requirements are fully dependent on an area's designation status. Without a singular standard to evaluate available air quality data, states are unable to fully evaluate the potential for monitoring network changes, both additions and decommissions. To ensure meaningful public participation, the EPA should provide the states and the public with an opportunity to comment on a singular level rather than a range of potential standards.

Furthermore, the EPA's expectations for the design of the monitoring network in nonattainment areas under the proposed new standard are inadequately outlined. The EPA's proposed rule language rewrites 40 Code of Federal Regulations (CFR) §58, Appendix D, Section 5 to require a National Core (NCore) multi-pollutant site in existing nonattainment areas and an Enhanced Monitoring Plan (EMP) for areas designated nonattainment under the new standard. However, the EPA failed to provide even basic guidance on the requirements for an approvable EMP in nonattainment areas. Without this discussion, there is no information for states to evaluate and provide comment for EPA consideration. A regulatory requirement that does not specify the criteria for approval and content requirements does not meet the requirements of the Federal Administrative Procedures Act for public notice and comment, nor does it provide for consistency in application and review. For example, it is unclear if the full suite of pollutant monitoring required at existing NCore sites would be required in each new nonattainment area or how many monitoring sites within the new area are sufficient to adequately understand ozone formation.

Finally, it is unclear how the different monitoring requirements would fit together under the proposed rule. The EPA proposes to maintain existing NCore sites with the addition of photochemical assessment monitoring station (PAMS) pollutant monitoring. The EPA also solicited comment on whether ozone network design requirements should consider both attainment status and population. Although the TCEQ agrees that attainment status along with population should determine applicable monitoring requirements for an area, the preamble did

not discuss the potential population thresholds that would apply. Without discussion of the population thresholds under consideration, there is no way for states to provide meaningful comment. Also, it is ambiguous whether the EPA intends to require a number of ozone monitors based on population in addition to NCore/PAMS monitoring and monitoring under the EMP. Given this uncertainty, it is unreasonable to require states to have all ozone and PAMS monitors in operation by the 2017 deadlines provided in the proposed rule.

The EPA should provide or further articulate the flexibility states have in moving or decommissioning monitors placed in nonattainment areas under the EMP.

The EPA's preamble language suggests that greater flexibility would be provided to states for the design and maintenance of the monitoring network under the EMP. However, it is unclear if these monitors would be as immoveable as monitors that have historically determined compliance with the current ozone NAAQS. If the EPA intends for the EMP to be flexible enough to fully evaluate ozone formation chemistry, the monitors should be considered special purpose monitors (SPM), the EPA's Air Quality System (AQS) needs to be updated to allow EMP classification of monitors, and states need to be provided explicit ability to move or decommission monitors, including those that may have historically been used for compliance purposes.

The proposed changes to the PAMS requirements will not necessarily save money or resources, as the EPA suggests in the proposed rule.

The EPA proposes to collapse current PAMS monitoring requirements and, instead, only require PAMS measurements at existing NCore sites that are in ozone nonattainment areas. The EPA's proposed plan oversimplifies the monitoring required to understand ozone formation chemistry in large metropolitan areas. Under existing federal rule, states are only required to place between one and three NCore sites within a state in areas not impacted by large emission sources. As shown in historical monitoring data, ozone and ozone precursor concentrations can vary widely across a metropolitan area within a single day. Discontinuing PAMS measurements currently being collected across a large nonattainment area and relying on only one monitoring site to determine concentrations, compliance, and trends in the area disregards the natural heterogeneity of ozone concentrations within a region and over time. Furthermore, the siting criteria for the original NCore sites does not take into account population exposure, ozone formation, and ozone transport within a region, which would be important to siting monitors intended to determine compliance with the ozone NAAQS.

Because a network of monitors is required to understand the particulars of ozone formation chemistry and regional transportation, states would likely need to keep the existing network of PAMS monitors and may even need to increase its monitoring. In Texas, the current placement of 22 PAMS monitoring stations in two ozone nonattainment and two ozone maintenance areas has proven invaluable to understanding ozone formation and trends, as well as assessing air quality strategies for achieving compliance with the NAAQS. Depending on the level of the standard, as many as 8 additional areas in Texas alone are predicted to be in nonattainment. Each new ozone nonattainment area would require significant resources to set up a network of monitors to study and track ambient ozone levels. The evaluation of this potential increase in needed monitors is, again, complicated by the lack of guidance on an approvable EMP.

Uncertainty in the evaluation of the impact on monitoring resources is further obscured by lack of information about federal funding. On December 15, 2014, an EPA representative presenting on the proposed ozone rule stated that federal grant money allocations would likely be recalculated based on monitoring network changes in the final rule. To date, no calculation mechanism has been established. The lack of federal funding for ozone and ozone precursor

monitoring would create a heavy burden on state finances, the extent of which can only be speculated on at this point.

For these reasons the proposed changes to monitoring requirements will likely not save states money and resources, as the EPA's preamble language suggests. Instead, the proposed requirements may increase required funding, particularly state funding, to adequately measure area air quality. The EPA should provide opportunity for public comment when greater detail on monitoring network design and funding can be published.

The EPA needs to further evaluate the feasibility of the mixing height requirement before including it as a network requirement.

The proposed rule requires collection of mixing height data at required PAMS sites. Though the TCEQ supports the consideration of less expensive technologies to fulfill this data need, the EPA should provide states with a later deadline for meeting this need so that the technology and data handling procedures can be better evaluated.

The proposed rule gives states only nine months from the date the EPA Administrator promulgates final designations to submit an annual monitoring network plan that incorporates PAMS requirements in new ozone nonattainment areas. States then have 11 months to deploy any required monitors. It is unreasonable for the EPA to expect states to purchase and deploy their own equipment in this time given the significant cost of radar profilers, uncertainty with the level of federal funding, and state budget and procurement processes. Although the EPA suggests the use of ceilometers, which would provide a much more cost-effective method, this technology, by EPA's own admission, is not currently appropriate for this measurement and it is unclear if they can be upgraded, particularly in time to meet the implementation deadline provided in the proposed rule.

Furthermore, the proposed rule considers states' use of mixing height data collected by other monitoring entities but does not provide adequate data handling information for states to fully evaluate the implications of using secondary data. The EPA needs to clarify which monitoring organization will be the primary quality assurance organization (PQAO) and certifying agency for mixing height data collected at National Oceanic and Atmospheric Administration stations. The EPA also needs to clarify whether mixing height data would be required to be reported in AQS. If the data will be reported, the EPA's AQS database needs to be updated to both allow the ingestion of the data and allow mixing height data to be collected from a site that is separate from the PAMS. Currently in AQS, the location of the meteorological data is indicated at the site, which implies that all the meteorological data is either collected at that particular site or another designated location.

The EPA should clarify which meteorological parameters would be required at PAMS.

The preamble states that precipitation, solar radiation, and UV radiation are proposed to be required at PAMS. However, the proposed revisions to 40 CFR §58 Appendix D, Section 5 do not include these parameters in the PAMS requirements.

The EPA should clarify the data selection criteria for NAAQS comparison.

The proposed Section 2 in Appendix U of 40 CFR §50 outlines criteria for monitoring method, placement, quality assurance, and data submittal that must be adhered to before measurement data can be used for NAAQS comparison. The section also states that data "otherwise available to the EPA shall be used in design value calculations." The EPA needs to clarify that any data used to calculate a design value should also meet the same monitoring method, placement, and quality assurance requirements as data submitted to AQS, and that any other data "otherwise available to the EPA" only be used in design value calculations if the EPA provides that data to

states by a specific date that allows for adequate time for states to refute its use or appropriately consider it in design value calculations. Without these restrictions, the EPA's discretion would be wholly unfettered and leave states without administrative or legal recourse to rebut the use of "other data" in design value calculations.

E. Ambient Monitoring and Exceptional Events Demonstrations

The EPA should provide states with either more time for exceptional event analysis and documentation or more detailed approval criteria and guidance on approvable exceptional event demonstrations.

The TCEQ appreciates the EPA's commitment to evaluate exceptional events and establish schedules for states to flag data and submit related documentation that will be used in the initial designations for a revised ozone NAAQS, if promulgated. Under its proposal, the EPA appears to rely on the fact that excluding air quality monitoring data influenced by exceptional events provides a mechanism to prevent areas from being designated nonattainment based on certain air quality issues beyond an area's control. The TCEQ considers the analysis of exceptional events to be critical in determining accurate design values and establishing nonattainment boundaries, but current guidance and proposed timelines make the use of the exceptional events process impractical for adequately addressing background ozone concentrations for designation purposes, especially given that the EPA has no prescribed review and approval deadlines for submittals.

The proposal of an ozone standard that is at or near background concentrations places increased importance on the exclusion of measured concentrations that are influenced by exceptional events, yet the proposal does not provide an adequate mechanism for approval of these demonstrations in a time frame that allows the data to be excluded for designation. The proposed ozone rule gives states 12 months from the time of promulgation to provide any exceptional event demonstration documents to the EPA for events occurring in 2013, 2014, and 2015 if the data are to be excluded for purposes of designations. Assuming promulgation of the revised standard occurs as the EPA plans in October 2015, states would have less than a year to complete the research required to understand the potential contributing natural and anthropogenic sources and historical fluctuations and establish clear, causal relationships between the air quality measurement and the event to the detailed degree required by the EPA for 2015 data. This process presents a significant challenge for Texas and other states, especially as the guidance for exceptional event demonstrations is not definitive since the Exceptional Events Rule has not been finalized.

The EPA should propose revisions to the Exceptional Events Rule in a timely manner.

At this time, states lack the specific approval criteria in current rule and guidance necessary to make an exceptional event package worth the expense of development. The EPA indicates in the proposed rule that revisions to the Exceptional Events Rule will be proposed in a future notice and comment rulemaking effort and that the EPA will solicit public comment at that time. Considering that the analysis of exceptional events is critical for developing state designation recommendations, the TCEQ requests that revisions to the Exceptional Events Rule be proposed and finalized as soon as possible. Ideally, that rulemaking would have been on the same timeline as promulgation of the NAAQS in order to implement the exceptional event statutory objective. In order to be useful in focusing limited state resources, the Exceptional Events Rule must be adopted at least several months prior to the proposed deadline for the submission of exceptional event demonstrations, or the EPA should allow for initial exceptional events submissions for the revised ozone NAAQS to be supplemented as necessary to meet the Exceptional Events Rule requirements.

The final rule for the revised ozone standard, if promulgated, should establish deadlines for completion of the EPA's review of exceptional event documentation, and should establish an expedited appeal process for states.

The 12-month exceptional event submission deadline coincides with the deadline for states to make designation recommendations to the EPA, which is also a resource-intensive effort. The EPA Administrator would then have 12 months to make final designations while concurrently reviewing exceptional event packages.

The TCEQ recommends that the final ozone rule require the EPA complete the review of exceptional events submissions in time for its findings to be used in making initial designations. States will face competing demands for limited resources based on the regulatory deadlines for multiple aspects of the standard implementation process under this proposal. To justify the expenditure of resources involved in making an exceptional event demonstration, states need certainty that the EPA's review of their demonstration will be completed in a timely manner so that it can be taken into consideration for the initial designations under the proposed standard, if promulgated. Additionally, the rule should establish an appeal process for states to receive review of EPA denials of exceptional event submissions.

F. Implementation Considerations Regarding Background Ozone Levels

An ozone standard of 70 ppb or lower will make background levels critically important for many areas. The EPA must clearly define and describe methods by which these areas can account for the effects of background ozone, especially from foreign emission sources.

Concerns about how background ozone influences peak ozone are borne out by studies described in the literature. Langford et al. (2009), in a study of Houston background ozone, found that 8% of the variation in peak ozone could be explained by background ozone.[2] Berlin et al. (2013) confirmed the large impact of background ozone upon peak Houston ozone, showing that most of the interannual variation in mean peak ozone is due to background ozone variation, and that, even on high ozone days, background ozone is usually at least 50 percent of the peak value.[3] In Houston, the local contributions to high ozone are dropping more rapidly than background ozone, so that the relative background contribution is increasing. In fact, the EPA notes that

> "while the majority of modeled O3 exceedances have local and regional emissions as their primary cause, there can be events where O3 levels approach or exceed the concentration levels being proposed in this notice (i.e., 60-70 ppb) in large part due to background sources" which "typically result from stratospheric intrusions of O3, wildfire O3 plumes, or long-range transport of O3 from sources outside the U.S."[4]

The EPA offers scant remedies for urban areas influenced by sources beyond their control. The "exceptional event exclusion" may be useful in rare instances, but demonstrating even a single instance is extremely burdensome and, as previously discussed, the states face uncertainty regarding what is required for an acceptable exceptional events demonstration. The other potential remedy relies on federal Clean Air Act (FCAA) §179B and requires a demonstration

---

[2] Langford, A. O., C. J. Senff, R. M. Banta, R. M. Hardesty, R. J. Alvarez II, S. P. Sandberg, and L. S. Darby (2009), Regional and local background ozone in Houston during Texas Air Quality Study 2006, J. Geophys. Res., 114, D00F15, doi:10.1029/2008JD011687.

[3] Berlin, S.R., A.O. Langford, M. Estes, M. Dong, D.D. Parrish (2013), Magnitude, decadal changes, and impact of regional background ozone transported into the greater Houston, Texas area, Environ. Sci. Technol., 47(24): 13985-13992, doi: 10.1021/es4037644.

[4] While it is the TCEQ's convention to spell out ozone, when quoting from an EPA document, O3 is used if that is what was used in the original text.

that an area would attain the standard by its attainment date "but for" emissions emanating from outside the United States. However, the EPA has only approved such demonstrations for two areas adjacent to the Mexican border. The EPA does note that areas distant from international borders may be affected by emissions from foreign sources, offering some hope of relief for large sections of the country but offers little guidance on how such a demonstration should be made or what would be acceptable. For example, would modeling that excluded emissions from foreign areas within the modeling domain and using adjusted boundary conditions constitute an acceptable demonstration?

The EPA should clarify how states are to address the significant implementation challenges associated with attaining an ozone standard at or near "background" levels.

In the preamble to the proposed standard revision, the EPA explicitly states that it does not believe that a prospective standard of 70 ppb "would create significant implementation-related challenges." However, the same claim is not posited for other proposed standards, thereby implying that a standard lower than 70 ppb would result in significant implementation challenges. While the TCEQ agrees with this implied rejection of a standard lower than 70 ppb, it disagrees with the assertion that a standard between 70 ppb and the current level of 75 ppb would be free of significant implementation challenges.

Part of the challenge associated with background ozone is stratospheric ozone intrusion. There is a growing body of scientific research that stratospheric ozone contributes to background ozone concentrations entering eastern Texas.[5] While the research may not provide evidence to show that deep stratospheric intrusions routinely cause exceedances of the current ozone standard there would be concern with this background impact with lower levels of the ozone standard. The influence of deep tropopause folding cannot be ruled out.

The EPA notes that the Policy Assessment provides three specific definitions of "background" after noting that using the term generically "can lead to confusion as to what sources of O3 are being considered." With a standard in the 65-70 ppb range, background can amount to well over half of the standard in some cities regardless of which definition of "background" is chosen. The closer the standard is set to background, the more limited the opportunity for further reductions with the significant controls already in place and the few remaining options for control become increasingly expensive and lifestyle-changing for citizens in the nonattainment area.

G. Implementation Timing

The EPA should complete redesignation of areas that have demonstrated attainment with the 2008 and 1997 ozone standards and then revoke them prior to adopting a new standard.

The TCEQ notes that the EPA has not yet completely revoked the 1997 ozone standards, as was indicated in prior proposals relating to the 2008 ozone NAAQS. If a new ozone standard is adopted as proposed without action to revoke the prior standards, there could be three different ozone standards in effect. The TCEQ strongly recommends that the EPA completely revoke the 1997 and 2008 standards, as the continuing applicability of legacy ozone standards causes unnecessary confusion and a needless expenditure of resources. However, the TCEQ continues

[5] Thompson et al. (2008), Tropospheric ozone sources and wave activity over Mexico City and Houston during MILAGRO/Intercontinental Transport Experiment (INTEX-B) Ozone Sonde Network Study, 2006 (IONS-06), Atmos. Chem. Phys. 8: 5113-5125.
Lin et al. (2012), Springtime high surface ozone events over the western United States: Quantifying the role of stratospheric intrusions, JGR (accepted).
Lefohn, A.S., Wernli, H., Shadwick, D., Oltmans, S.J., Shapiro, M., (2012), Quantifying the Importance of Stratospheric-Tropospheric Transport on Surface Ozone Concentrations at High- and Low-Elevation Monitoring Sites in the United States, Atmospheric Environment, doi: 10.1016/j.atmosenv.2012.09.004.

to support the redesignation of areas that demonstrate attainment of the relevant ozone NAAQS. Without such a redesignation, it is possible that nonattainment consequences, including the assessment of FCAA, §185 fees, may continue to be imposed on an area that is no longer monitoring nonattainment for the prior standards. The EPA must provide for such an opportunity for redesignation or an area that has, through extensive control requirements, managed to meet previous standards will continue to unfairly experience penalties and requirements related to those standards.

Should the EPA move forward with adoption of a revised ozone standard as a result of this proposal, the 2008 ozone NAAQS should also be completely revoked as part of that standard's adoption to simplify implementation by focusing limited state resources on only the most recent standard.

The EPA should commit to firm deadlines for the proposed and final implementation rule for addressing the revised ozone NAAQS in order to provide timely guidance for state implementation plan (SIP) development. The EPA should also provide specific, timely guidance for the transport requirement, which is a part of the infrastructure requirement.

The proposed rule provides general timelines for the proposal and finalization of an implementation rule to address any new implementation requirements resulting from revisions to the ozone NAAQS, including nonattainment area classification methodologies, SIP due dates, attainment dates, transport assessments, and required implementation programs such as nonattainment new source review (NNSR) and conformity. The TCEQ agrees with the EPA's general intent to propose this implementation rule within one year after the revised ozone NAAQS are promulgated and finalize the implementation rule by no later than the time the area designations process is finalized (approximately one year later). However, the TCEQ requests that the EPA commit to firm deadlines for the completion of such implementation guidance documents rather than relying on "target dates."

As a result of the EPA's lack of a timely implementation rule for the 2008 standard, states have been forced to expend effort and resources to develop SIP revisions without EPA guidance, and therefore may ultimately be wasting resources by developing submittals that will not be approvable. Texas has been in the process of developing nonattainment area SIP revisions for the 2008 ozone standard for years in order to meet statutory deadlines. These nonattainment area SIP revisions are due to the EPA in July 2015 and an implementation rule was just issued. In order to meet statutory deadlines, states do not have the option of waiting for the EPA to provide guidance before proceeding with SIP development, review, and submittal.

Overall, the EPA has routinely failed to issue timely implementation guidance for SIP revisions and to even meet statutory deadlines in the FCAA. As a result, the EPA has disrupted the SIP development process nationwide, undermining the states' ability to submit sufficient SIP revisions meeting the EPA's expectations and requirements. The EPA should commit to finalizing the implementation rule for the revised ozone NAAQS at the same time as finalizing the area designations process.

The TCEQ supports the option for states to submit infrastructure SIP revisions for a distinct secondary NAAQS separately and at a later date than infrastructure SIP revisions for the primary NAAQS.

If both a revised primary NAAQS and a distinct secondary NAAQS are finalized, the EPA is proposing to allow states the option of submitting separate infrastructure SIP revisions for the primary and secondary NAAQS with different deadlines. The EPA is proposing to extend the deadline for infrastructure SIP submittals for a distinct secondary standard by 18 months beyond the initial three-year statutory deadline that would apply to an infrastructure SIP

submittal for the primary NAAQS. The TCEQ appreciates that the EPA is providing flexibility in establishing deadlines for infrastructure SIP submittals provided that such deadlines would adequately meet statutory requirements under the FCAA. As such, the TCEQ requests that the EPA include the deadline for submittal of infrastructure SIP revisions in rulemaking. This would allow states more confidence that the EPA would not face legal challenge on the submittal dates for the SIP revisions.

When promulgating final area designations for a revised ozone NAAQS, the EPA should ensure that the effective date of the designations aligns appropriately with the end of ozone season for all areas.

As indicated in the proposed rule, the EPA plans to finalize area designations by October 1, 2017. It is expected that the effective date of these designations will ultimately determine the deadline for a nonattainment area to comply with the NAAQS based on the area's nonattainment classification. Ozone season in Texas varies and is currently monitored during periods from March through October and January through December depending on the area. If the effective date of designations does not align with the end of ozone season, states may face challenges associated with demonstrating compliance with the revised ozone NAAQS for all potential nonattainment areas.

In the initial rule implementing the 2008 ozone NAAQS, published on May 21, 2012 (77 FR 30160), the EPA recognized the challenge of demonstrating compliance with the NAAQS based on a July 20 effective date. The EPA explained in this initial rule that where a designation is effective late in the ozone season, basing the attainment date on the effective date of the designation had the effect of providing one less complete ozone season for areas to improve their air quality than was accorded areas under the FCAA. A marginal area, for example, would have only two full ozone seasons following the effective date of designation to improve its air quality in order to attain by its attainment date. This is because attainment is based on three *full* ozone seasons of air quality data; thus in order to attain "by" its attainment date, the area could not consider air quality for an ozone season during which the attainment date falls. As a result, all nonattainment areas would ultimately be required to demonstrate compliance with the NAAQS one full ozone season earlier than the year of its attainment date.

This presumably unintended potential timing conflict limits not only the monitoring data used for compliance but also the lead time available for implementing controls to achieve any emissions reductions necessary for attaining the NAAQS by the attainment deadline. In the initial implementation rule, the EPA established that the attainment date for the 2008 ozone NAAQS would be specified as a certain number of years from the end of the calendar year (December 31) in which an area's nonattainment designation is effective. Following years of state SIP development based on a December 31 attainment date, however, the D.C. Circuit Court of Appeals published an opinion on December 23, 2014 vacating the provision of the EPA's initial rule relating to attainment deadlines and noting that the appropriate attainment date should be based on the effective date of designations rather than the end of the calendar year. In light of this recent court ruling, and the implementation challenges that states may face if designations are effective prior to the end of ozone season, the TCEQ urges the EPA to time the effective date of designations appropriately so that it aligns with the end of ozone season for all areas.

H. Implementation For Permitting Programs

The TCEQ strongly supports the EPA's proposed Prevention of Significant Deterioration (PSD) grandfathering provisions.

The EPA generally requires that a project demonstrate compliance with any revised NAAQS that are in effect when a permit is issued. However, the EPA is proposing to allow PSD permit

applications to be "grandfathered" from this requirement for the revised ozone NAAQS, as long as either of the following conditions apply:

- For permitting agencies that make a determination of technical completeness, the formal determination that the application is complete has been made on or before the signature date of the final rule; or
- The public notice for a draft permit or preliminary determination has been published prior to the date the revised ozone standards become effective.

The TCEQ strongly supports the proposed grandfathering provisions. The EPA adopted similar grandfathering rules for the 2012 NAAQS for particulate matter of 2.5 microns or less in diameter, which provided a reasonable transition for the permitting process. The grandfathering rules mitigated an additional resource burden on both permit applicants and reviewers, minimized delays for pending PSD permit applications, and protected air quality.

III. Appendices

A. References for March 17, 2015 Comments on the Primary Standard

B. Paper: Shaw, BW; Lange, SS; Honeycutt, ME. 2015. "Lowering the ozone standard will not measurably improve public health." Environ Manage. In Press.

C. Paper: Goodman, JE; Sax, SN; Lange, S; Rhomberg, LR. 2015. "Are the elements of the proposed National Ambient Air Quality Standards based on the best available science?" Reg Tox Pharm. In Press.

D. Paper: Goodman, JE; Prueitt, RL; Sax, SN; Pizzuro, DM; Lynch, HN; Zu, KE; Venditti, FJ. 2015. "Ozone exposure and systematic biomarkers: evaluation of evidence for adverse cardiovascular health impacts." Crit Rev Tox. In Press.

E. Poster: How dose-response curves derived from clinical ozone exposures can inform public policy

F. Comments by the Texas Commission on Environmental Quality Regarding the National Ambient Air Quality Standards for Ozone EPA Docket ID No. EPA-HQ-OAR-2005-0172

G. Additional References for March 22, 2010 Comments