The Texas Commission on Environmental Quality argues that the thoughtful integration of scientific data does not support the assumption that tightening the ozone standard will result in measurable health benefits.

The Texas Commission on Environmental Quality (TCEQ) strives to protect our state’s public health and natural resources consistent with sustainable economic development. In accordance with this mission, the State of Texas alone has spent >$1 billion since 2001 striving to achieve the 1997 0.08 parts per million (ppm) ozone standard. Most of Texas’ air quality areas recorded their lowest ozone values ever in 2014. The Houston and Dallas/Fort Worth areas, for example, have seen ozone levels reduced 29% and 21%, respectively, during the past 15 years, while the population has increased 34% and 29%, respectively. We will continue to expend resources to achieve the 2008 75 parts per billion (ppb) ozone standard, which has yet to be fully implemented by the U.S. Environmental Protection Agency (EPA). However, as the concentration of ambient ozone decreases, it becomes exponentially more difficult, and expensive, to attain further reductions. EPA is poised to lower the standard further. While cost cannot be considered in setting the standard, the high cost of further lowering the standard necessitates that this be a sound policy decision and will result in measurable health benefits.
EPA bases its proposal to lower the ozone standard on three key health-related endpoints: premature mortality, respiratory morbidity (i.e., asthma exacerbation, emergency department visits, and hospital admissions), and lung function (i.e., primarily FEV₁ [Forced Expiratory Volume in 1 second, a measure of lung function] decrements). We agree that respiratory effects can occur at the high ozone concentrations that were measured in the 1980s and 1990s. The pertinent question is whether lowering the ozone standard from 75 ppb to 70 or 65 ppb will result in a measurable reduction in these effects. In this short review, we consider some important concerns with EPA’s conclusions about the health effects of ambient ozone concentrations. We conclude that EPA has not demonstrated that public health will measurably improve by decreasing the level of the ozone standard.

Ecological Epidemiology Studies, Not Adequate for Setting Standard

EPA relies heavily on ecological epidemiology studies for its assessment of premature mortality and respiratory morbidity. These studies have been very inconsistent in their findings, and flaws, biases, and unusual characteristics of the data have made them difficult to interpret. One unusual and as-yet unexplained characteristic of the epidemiological associations between short-term ozone exposure and mortality is regional heterogeneity. This heterogeneity means that different cities have different associations between short-term exposure to ozone and mortality, and very few of those associations are positive.¹⁻⁴

For example, Smith et al.¹ found that only 7 of the 98 cities investigated showed a statistically significant positive association between 8-hr ozone concentrations and mortality (this is very close to the 5% that would be expected purely by chance). Additionally, there was no association between the estimated effect of ozone on mortality for a city and the concentration of ozone in that city (see Figure 1 on page 28). EPA⁵ estimates short-term mortality impacts based on Zanobetti and Schwartz⁴ and the Smith et al. study.¹ However, the concentration response functions (CRFs) vary from negative to positive for the same city, depending on study selection, ozone averaging time, model specifications, and ozone season. In fact, most of these estimates are indistinguishable from zero. EPA uses a pooled nationwide estimate for their risk calculations, but the substantial heterogeneity between cities that ranges from positive to null or even negative (i.e., higher ozone concentrations correlated with reduced mortality) makes this nationwide estimate misleading and overestimates ozone risk.

The relationship between long-term ozone exposure and mortality has been investigated in at least 12 epidemiology studies.⁵⁻¹⁷ When considering other potential causes of mortality, such as other air pollutants, only one of those studies¹⁵ showed a statistically significant (but very small) effect of ozone on respiratory mortality. Interestingly, the effect only occurred at temperatures above 82 °F. It is known that very warm or very cold temperatures are associated with increased mortality.¹⁸ Paradoxically, the increased mortality was not observed in U.S. regions with the highest ozone concentrations (e.g., Southern California) nor in areas with the highest number of respiratory deaths (e.g., the Northeast and industrial Midwest). Therefore, long-term mortality studies also demonstrate unexplained regional heterogeneity and mostly don’t show associations between ozone and long-term mortality.
Evaluating the Current Ozone Standard

Altogether, this means that it is highly unlikely that the measured associations between ozone and respiratory mortality/morbidity are plausible, because the ozone exposures of the people in the population are so low. Were all of the hundreds of thousands of people in the epidemiology studies outside for 8 hours the day immediately before their deaths? In fact, this concern was raised by the Clean Air Scientific Advisory Committee (CASAC) ozone review panel, EPA’s scientific advisors, in a June 5, 2006 letter25 to EPA: “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.” This difference between ambient ozone concentrations and personal exposures is critical for interpreting both epidemiological studies as well as clinical exposure studies.

Lung Function Decrements
Unlikely to Be Adverse Below Current Standard

The TCEQ agrees with EPA that the ozone clinical data are best for setting the ozone standard. The American Thoracic Society (ATS) defines adversity as a significant decrease in FEV$_1$ with a significant increase in symptoms.26 The ATS notes that FEV$_1$ decrements can vary by as much as 5% in healthy adults within a single day and by 15% or more from year to year. EPA defines a 10% FEV$_1$ decrement in a sensitive population as an appropriate adverse effect to protect against because it is mild and reversible. EPA asserts that two clinical studies, by Kim et al.27 and Schelegle et al.,28 justify lowering the current 75-ppb standard.

The Kim study reported statistically significant FEV$_1$ decrements (1.71%) in healthy young adults after 6.6 hours of 60-ppb ozone exposure while exercising heavily for 50 minutes out of every hour. However, these decrements are within normal variation and are not adverse by either the ATS criteria (i.e., because they were not statistically
associated with symptoms), or by EPA’s criteria (i.e., because they were less than 10%).

The Schelegle study reported statistically significant FEV₁ decrements—5.34%, 7.23%, and 11.42%, respectively—associated with symptoms in healthy young adults after 6.6 hours exposure to 72-, 81-, and 88-ppb ozone, but not 63-ppb ozone, while exercising heavily for 50 minutes out of every hour. For 72-, 81-, and 88-ppb ozone, this exposure meets the ATS criteria for adversity, but at 72- and 81-ppb, it does not meet EPA’s criteria of adversity until 88-ppb, which is above the current standard.

To claim that the lung effects at 60- and 72-ppb from the Kim study and the Schelegle study are adverse, even though the group mean FEV₁ decrements were not adverse, EPA notes that at 60-ppb, 3 of 59 study subjects had FEV₁ decrements greater than 10%, and at 72-ppb 5 of 31 individual participants had FEV₁ decrements greater than 10%. EPA is essentially basing its assertion of adverse effects occurring at concentrations lower than the current standard on these eight individual measurements.

On the other hand, 5 of 31 individual participants had increases in FEV₁ after 72-ppb exposure. The remaining participants showed little, if any, change in FEV₁, altogether confirming the known large inter-individual variability in lung function responses. Lung function returned to baseline for all of the participants within 1–4 hours after cessation of exposure. As noted by Folinsbee et al. and McDonnell et al., the exposure regimens used in the Kim and Schelegle studies simulate work performed during a day of heavy manual labor in outdoor workers. This is an unrealistic exposure scenario for sensitive subpopulations, such as asthmatic children and elderly chronic obstructive pulmonary disease patients. In addition, these lung function decrements would be transient, reversible, would not interfere with normal activity, and would not result in permanent injury or respiratory dysfunction.

Further, EPA evaluated these effects based on exposure concentration, not dose (i.e., a function of exposure concentration, time, and ventilation rate). The healthy young study participants exercised vigorously for the majority of their 6.6 hour

References

exposure, dramatically increasing their dose, and therefore response, as compared to a resting or moderate exercise ventilation rate for the same exposure concentration. Given these facts, EPA has not demonstrated that lowering the ozone standard from 75-ppb to 70–65-ppb will result in a decrease in adverse lung function effects in the population.

**Evidence for Ozone Exacerbation of Asthma Is Insufficient**

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, 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 EPA shows that there is no statistically significant decrease in asthma exacerbations with a decreasing level of the ozone standard. EPA also states that emergency department visits and hospital admissions are robust to co-pollutant confounders, but does not mention investigation of confounding by pollen, which is a known, strong inducer of asthma. Also, 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. Therefore, EPA should not have drawn the conclusion that ozone enhances asthma morbidity at ambient concentrations based on these data.

In conclusion, the TCEQ thinks the thoughtful integration of the scientific data does not support the assumption that lowering the ozone standard from 75 ppb to 70–65 ppb will result in measurable health benefits. The ecological epidemiology studies are critically flawed due to severe exposure misclassification because personal exposure to ozone is approximately 10% of ambient levels, dramatically reducing the ozone dose people actually receive. The clinical studies do not indicate anything beyond mild, reversible effects below 75 ppb. It is biologically implausible that 8-hr ambient ozone concentrations below 75 ppb would cause mortality when they do not cause mild effects.

**References**


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