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TEXAS COMMISSION ON ENVIRONMENTAL QUALITY

Protecting Texas by Reducing and Preventing Pollution

March 10, 2014

Dr. H. Christopher Frey
Chair, Clean Air Scientific Advisory Committee
Science Advisory Board
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Dear Dr. Frey:

The Texas Commission on Environmental Quality (TCEQ) has reviewed the 2014 *Health Risk and Exposure Assessment for Ozone Second External Review Draft* (REA, US EPA, 2014) (HREA). In this draft, the Environmental Protection Agency (EPA) presents its analysis of 12 urban areas, including Houston. However, EPA highlighted modeled results without a sufficient discussion of the uncertainty surrounding these estimates.

The TCEQ appreciates EPA's efforts to improve the science used to quantify the effects of modifying the ozone (O₃) National Ambient Air Quality Standards (NAAQS), specifically the replacement of the quadratic rollback with a model-based approach that more realistically portrays the anticipated resulting ozone concentrations through use of the Higher-order Direct Decoupled Method (HDDM). However, a number of issues must be emphasized. For example, the 2008 National Emissions Inventory (NEI) has been shown to have nitrogen dioxide (NO₂) biases in Texas and other states, perhaps due to non-road, area, and off-road sources. The modeling shows higher mean normalized bias in many areas of the U.S. which may be attributable to the model's relatively coarse resolution of 12 km. To properly assess the effects of a new ozone standard in these regions, high resolution simulation (4 km or smaller) is necessary. In addition, treating emission reductions uniformly across all sectors and geographic areas is clearly unrealistic. And finally, the results of EPA's analysis wherein both nitrogen oxides (NO_x) and volatile organic compounds (VOC) emissions were reduced simultaneously seem contradictory, because for many cities including Houston it appears that reducing both pollutants simultaneously would be less effective than reducing NO_x alone.

Indeed, in part because of these modeling decisions, the HREA indicates a lower standard may result in additional premature mortality for some areas of the country, including Houston. In fact, the last line of the HREA states that: "[m]ortality from short-term and long-term O₃ exposures and respiratory hospitalization risk is not greatly affected by meeting lower standards..." This observation does not support the necessity of a lower standard. EPA's own modeling shows either adverse or little to no public health benefit from lowering the current standard, therefore TCEQ urges EPA to retain the existing standard.

The draft HREA presents hypothetical health effects that are based on one or two 8-hour exposures above the various benchmarks. Based on modeling presented in the HREA, it appears that the only significant potential exposures would be to 60 ppb ozone. At this concentration we would expect only

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mild, reversible, transient effects on lung function that are not of clinical importance. Furthermore, based on the confidence intervals presented in the document, no significant 8-hour exposure to 70 or 80 ppb would be expected even if the current standard were to be retained. This information does not support a more stringent NAAQS. Moreover, the ozone standard is based on the 4th highest 8-hour exposure averaged over 3 years. This analysis does not support a lower standard that attempts to capture a single exposure over a given benchmark.

For mortality attributable to long-term exposure to ozone, EPA chose to use the same concentration-response function from Jerrett, *et al.*, 2009 for all 12 urban case study areas despite mentioning regional heterogeneity many times throughout the draft. TCEQ would like to emphasize that in light of the substantial regional heterogeneity, it is unclear how to interpret pooled estimates, particularly given the inconsistencies across studies. Moreover, this appears to be the first time a significant association between ozone and mortality has been reported for this cohort and results for six other cohorts¹ have not reported relationships between ozone and mortality. Taken together with the fact that mortality was considered to be "suggestive" of a causal association in the Integrated Science Assessment, the long-term mortality endpoint should not be included in the HREA or subsequent analyses.

Personal exposure is not considered in any of the epidemiology studies. As EPA clearly demonstrates in Figure 5-15, the vast majority of the U.S. population is not exposed to 8-hour ozone concentrations greater than 20 ppb. Using ambient ozone concentrations without consideration of personal exposure greatly overestimates risk and is inappropriate.

The TCEQ agrees with EPA that the NAAQS for ozone should protect public health. We would like to emphasize that modeling presented in the HREA indicates a lower standard may result in additional premature mortality for some areas of the country, including Houston. In addition, we would like to emphasize that when considering alternative O₃ standards, the lower end of the proposed range is not well-supported. In fact, EPA states that at lower concentrations "...the likelihood and magnitude of a response becomes increasingly uncertain..." and elsewhere that "...the relative importance of background O₃ would increase ...with a lower level of the O₃ NAAQS". In summary, EPA has not made the case that a lower standard will improve public health, and TCEQ urges EPA to retain the current standard. Measures designed to achieve a lower ozone standard could actually lead to increased health risk, e.g., decreased electric reliability resulting in summertime blackouts.

Sincerely,

Richard A. Hyde, P.E.
Executive Director

1. Three previous analyses with ACS cohort were negative (Pope et al. 1995, and 2002, Krewski et al. 2000, Smith et al. 2009); a study with the Harvard Six Cities Study cohort reports no association (Dockery et al. 1993); 3 updates with the AHSMOG cohort were negative (Beeson et al. 1998, Abbey et al. 1999, Chen et al. 2005); a study using the Women's Health Initiative cohort reports no association (Miller et al. 2007); two updates from the Veterans Affairs cohort report no association for mean ozone levels (Lipfert et al. 2000 and 2006); a study in Brisbane Australia cohort reports no association (Wang et al. 2009); and a study in Los Angeles reports no association (Jerrett et al. 2005).