Texas Pipeline Association
Attachment 2

EXECUTIVE SUMMARY
OZONE NAAQS PROPOSED RULE – EVALUATION OF THE SCIENCE
FOR
TEXAS PIPELINE ASSOCIATION
AUSTIN, TEXAS

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EXECUTIVE SUMMARY

Zephyr Environmental Corporation has been asked by the Texas Pipeline Association to perform an analysis of the health-based studies EPA relied upon to justify the setting of a lower ozone National Ambient Air Quality Standard (NAAQS). EPA set the current ozone NAAQS at 75 ppb in March 2008. Based on a thorough review of scientific evidence concerning the impact of ozone exposure on human health and welfare, EPA concluded at that time that a 75 ppb standard was protective. In the current rulemaking, EPA proposes to dramatically lower the NAAQS beyond the 75 ppb level, yet EPA has failed to point to any persuasive body of scientific evidence supporting its proposal. Indeed, EPA has failed to identify any study showing a clear causal relationship between ozone concentrations less than 88 ppb and clinically relevant adverse human health impacts.

Taken together, Zephyr concludes that the evidence and studies relied upon by EPA indicate that the current ozone NAAQS of 75 ppb continues to be protective of public health and welfare, **with an adequate margin of safety**. That is, EPA was correct in 2008 when it declined to set the NAAQS below 75 ppb. This conclusion is supported by EPA's own risk assessment as well as the scientific studies that EPA relied upon in that assessment.

Human health impacts studies do not support lowering the NAAQS.

There are only five published laboratory studies that measure the impact of short-term ozone exposure on lung function and that evaluate ozone concentrations in the range of the proposed ozone NAAQS alternatives (65 ppb–70 ppb). Not one of those studies supports the conclusion that human health would be improved by lowering the NAAQS below the current 75 ppb level.

In fact, those studies show that significant impairment in lung function does not occur until concentrations of 88 ppb or higher are reached. The current NAAQS of 75 ppb is well below the level at which these effects occur. Therefore, evidence from these controlled human exposure studies suggests that the current NAAQS is protective of public health with an adequate margin of safety.

The studies replied upon by EPA show that impairments in lung function observed at concentrations below 75 ppb are not consistently statistically significant (only 2 of 6 exposure groups reached statistical significance), are not usually accompanied by respiratory symptoms (only observed in 1 of 6 exposure groups), and do not reach the threshold that EPA has identified as being clinically meaningful (0 of 6 exposure groups).

Accordingly, Zephyr concludes that the controlled human exposure studies relied on by EPA show the following:

- Evidence of impairment in lung function has not materially changed since EPA made its prior decision to set the NAAQS at 75 ppb. At that time, EPA decided not to establish a NAAQS lower than 75 ppb because of the uncertainty about the extent to which lung

function impairments occur at concentrations below 80 ppb, and that uncertainty continues to exist; and

- The current NAAQS level of 75 ppb is protective of human health.

Population (epidemiology) studies do not support lowering the NAAQS.

Review of the population studies that EPA relied upon clearly indicates that the associations between ozone and respiratory health effects and mortality are inconsistent and uniformly weak and that the confidence in the associations is generally low:

- For example, despite EPA's claims, impairments in lung function do not consistently occur in any population (outdoor workers, adults or children exercising outdoors, asthmatics) in response to increased ozone levels, and neither asthmatic children nor adults consistently respond to ambient ozone levels with an increase in symptoms, medication use or activity limitation.

- In addition, multi-city hospital studies report both positive and negative associations with ozone concentration depending on latency periods, the particular model used to perform the analysis, and whether the results are adjusted for other co-pollutants. In other words, some studies show that human health actually improves as ozone levels go up. Such a counter-intuitive result casts doubt on the reliability of the scientific evidence that EPA is relying upon.

- Moreover, the uniformly small effects reported in epidemiology studies for all ozone-related health effects suggest that the associations are weak between ozone concentrations and short-term respiratory effects, hospital admissions and emergency room visits, respiratory symptoms and medication use in asthmatics, and both short- and long-term mortality.

There is enormous uncertainty regarding EPA's "likely causal" and "suggestive of a likely causal" relationship classifications for short-term and long-term mortality, respectively. Neither classification is supported by the available evidence. Notably, the mortality relationships vary across studies and cities and appear to be skewed by the presence of particulate matter. In addition, EPA's questionable practice of averaging individual city mortality coefficients dilutes the high and low values and produces overall averaged mortality coefficients that do not accurately characterize the true relationship for any of the cities.

The uncertainty about associations below 75 ppb from population studies in the last ozone NAAQS review precluded EPA from establishing the NAAQS at a lower level. Despite many new studies, that uncertainty remains today because of the inconsistent and weak associations reported, not to mention the prevalent methodological problems that continue to plague the population studies.

EPA's Health Risk and Exposure Assessment fails to consider all of the evidence and appears to be an attempt to justify a pre-determined decision to lower the NAAQS.

The goal of EPA's Health Risk and Exposure Assessment is to provide information that is helpful in answering questions about the adequacy of the existing ozone NAAQS at 75 ppb. However, EPA's practice of mischaracterizing the studies relied upon suggests that, in this case, EPA's risk assessment was not intended to uncover the real facts but instead was a means to justify a pre-determined end. In addition, EPA completely fails to account for the fact that the relationships between ozone and health effects reported in the population studies are uniformly weak and that the laboratory studies also fail to document clinically relevant adverse effects below 75 ppb.

EPA also overestimates exposure in the risk assessment by:

- Using models known to overestimate personal exposure indoors;
- Assuming unreasonably high activity levels in estimating impairments in lung function; and
- Substituting higher outdoor air concentrations for lower personal exposure levels in estimating the risk of hospital admissions and mortality.

EPA over-predicts risk by selectively relying upon some studies while not relying on others that might lead to a different result. For example, EPA selectively relies on studies that suggest that increased ozone concentrations lead to increased health effects. EPA also cherry-picks positive findings (ignoring negative results) from studies reporting mixed results and estimates risks associated with all ozone concentrations (down to zero), despite evidence that there is a threshold below which adverse health effects do not occur.

The net result of EPA's risk assessment is that, in some of the cities studied, increased ozone would actually improve human health. This is a finding that certainly does not support a lowering of the ozone NAAQS.

Conclusion.

Major concerns exist as to the scientific rigor of EPA's evaluation of the scientific literature, as well as EPA's risk assessment process, which adds to the already substantial uncertainty associated with the scientific studies detailed above and in our report. Recent laboratory studies that evaluate the association between ozone and lung function at exposures below the current NAAQS of 75 ppb indicate that there are no statistically significant adverse effects with clinical relevance to human health below 88 ppb. Nonetheless, EPA summarizes the information as providing positive evidence for adverse effects at levels below the current NAAQS by ignoring widely recognized definitions of what constitutes an "adverse" effect and the criterion for judging the clinical relevance of effects that EPA developed.

EPA does not reveal or appear to adequately consider factors that biased the population study results. Instead, EPA repeatedly provides summaries of the available scientific literature that emphasizes only positive associations between ozone and human health impairments (i.e., cherry-picking the data). The few positive and statistically significant associations reported in mortality studies are very weak and likely completely swamped by the large error introduced by not adequately adjusting the estimates for confounding variables (especially particulate matter) and regional differences.

While uncertainty about the evidence for ozone-associated health effects at levels in the range of the proposed lower NAAQS is already high, EPA's risk assessment process substantially adds to that uncertainty by ignoring evidence, using average mortality coefficients derived from highly variable city-specific estimates that are rarely statistically significant, and extrapolating the results from the studies to broader populations.

Our conclusion, which follows an exhaustive review of the evidence that EPA has provided in support of its recommendation, is that the evidence fails in every way to support a lowering of the NAAQS below its current level of 75 ppb.