Oral Testimony Provided to EPA's CASAC
Regarding the Second Review of the Integrated Science Assessment (ISA)

Good Morning. I am Dr. Neera Erraguntla, Senior Toxicologist in the Toxicology Division of the Texas Commission on Environmental Quality and I will be making the following oral comments on behalf of the TCEQ. I would like to thank EPA for this opportunity. I have four main points to discuss today. However, more extensive comments are provided in our previously submitted written comments.

1) The EPA should be transparent in its process of choosing key studies and provide clear documentation of how it arrived at its conclusions.

In order to determine that there is "sufficient evidence of an association," there must be an observed relationship between the exposure and health outcome in studies in which chance, bias, and confounding variables could be ruled out with reasonable confidence. However, many studies heavily relied by the EPA in the ISA are inadequate to demonstrate sufficient evidence of an association between short and long-term exposures to ozone and cardiovascular and central nervous system effects and mortality.

One key concern of the National Academy of Sciences was EPA's consistent failure to document how studies are selected for review.

- For example, the exclusion by EPA of well-conducted studies if their results showed no adverse health effects and the EPA's policy of discounting or ignoring studies showing no association between ozone exposure and asthma exacerbation.

2) The roles of uncertainty and bias in EPA's assessments have been severely downplayed. It is essential that EPA clearly discuss and characterize the uncertainties.

For ozone, EPA relies on studies that estimate personal exposure by using ambient monitoring data as a surrogate for personal exposure. However, it is very unlikely that people would ever be exposed to those pollutants at concentrations measured at outdoor monitors for very long periods of time. The average American spends a large amount (e.g., 82%) of time indoors, especially during the heat of the summer when ozone

concentrations tend to be at their highest. This assumption that the personal exposure of an average American is similar to ambient ozone concentrations oversimplifies and incorrectly assumes that ambient monitoring data accurately reflects personal exposure.

3) It is imperative that EPA distinguish true risk from just association because associations are not causations.

While it may be possible to find an association during statistical manipulation of data, this does not necessarily demonstrate causation. Application of the modified Hill criteria is necessary to start to prove causality.

- Ecological epidemiology studies are hypothesis-generating studies, not scientifically rigorous enough to be used as a basis for policy decisions.
- Ecological studies have used patient medical records instead of patient histories to monitor exposure and assess health effects. Such records are inadequate indices to associate ozone exposure and health effects.
- Further, meta- analyses of ecological epidemiology studies may give the appearance of hard data, but they have an extremely poor exposure component and studies conducted to date show only slight associations.

4) EPA should rely on biological, not just statistical, significance in identifying an adverse health effect in clinical studies.

The definition of "adverse effect" with regard to exposure to air pollution remains ambiguous. Clinical studies evaluating health effects due to ozone exposure have mainly focused on decreases in lung function as measured by forced expiratory volume in one second (FEV_1) and other similar measures.

- However, daily normal activities, exercise, and diurnal variations can themselves cause changes in the FEV_1 can range from 5 - 17.6%.
- Many agencies have indicated 20% decrease in FEV_1 as an adverse effect.
- Similar to the Adams (2006) study many recent clinical studies (Kim et al. 2011, Schelegle et al. 2009) of ozone exposure at 0.06 ppm have reported ***subtle*** statistically significant effects of FEV_1at 0.06 ppm that are not only within the

range of intra-individual variability but are also substantially less than the 20% decrease in $FEV_{1.}$

In summary, despite lack of compelling evidence, the ISA concludes causal associations with long-term exposure to ambient ozone and mortality. Further, the ISA includes conclusions regarding a wide-range of health effects including stronger causal associations for both short-term and long-term exposures to ozone and newer end points such as the effects to the central nervous system, cardiovascular system, as well as reproductive and developmental effects.