# Oral Statement to CASAC Review Panel on the Draft Risk and Exposure Assessment Planning Document for Sulfur Dioxide
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Good afternoon, my name is Lindsey Jones, and I am a Senior Toxicologist with the Texas Commission on Environmental Quality (TCEQ). I appreciate the opportunity to speak to you about the Environmental Protection Agency's (EPA's) draft Risk and Exposure Assessment (REA) Planning Document for Sulfur Dioxide ($SO_2$). The TCEQ has submitted written comments to the EPA on both drafts of the Integrated Science Assessment and will soon be submitting written comments on the REA Planning Document that include detailed information and supporting citations. Rather than reiterate the points raised in those documents, my comments today are focused on the discussion of the exposure-response function in the REA Planning Document.

The mode of action (MOA) for $SO_2$-induced lung irritation is straight forward and exhibits a clear threshold. $SO_2$ is highly water soluble and, once inhaled, rapidly dissolves into the lung producing sulfite, bisulfite, and hydrogen ions (Gunnison et al., 1987). The local absorption of these sulfurous compounds, particularly bisulfite, irritates the epithelial lining of the lung, producing the observed bronchoconstriction effects (Sheppard 1988).

Reflex biological responses like bronchoconstriction and cough are fundamentally understood to have a threshold to activate neural tissues controlling the smooth muscles of the conducting airways of the lung, or to orchestrate the muscular responses to produce a cough (Kubin et al., 2006). Individuals living with asthma or chronic obstructive pulmonary disease (COPD) presumably have a lower threshold for activating these responses than the average person due to elevated baseline airway inflammation, airway remodeling, and/or intrinsic genetic differences causing phenotypic responses (Holgate et al., 2015; Wong and Morice, 1999), but there is a threshold nonetheless. When key studies, like those identified in the current $SO_2$ review, are conducted in these potentially sensitive populations, uncertainty is decreased such that it negates the need for ultra-conservative assumptions like the lack of a threshold in the low-dose range.

The Committee on Acute Exposure Guideline Levels (AEGLs) corroborated this thinking in its 2010 Technical Support Document, which states that "the body of experimental data suggests that 0.25 ppm may be a threshold for bronchoconstriction in asthmatics" (NAS 2010) and that "0.2 ppm may be a NOEL [No Observed Effect Level] for bronchoconstriction in exercising asthmatics." The National Academies of Science published these conclusions in the same year that the EPA finalized the last $SO_2$ national ambient air quality standard (NAAQS). No substantively new MOA data have been

produced by the scientific community since the last review. Likewise, there are no free-breathing controlled human exposure data below 200 ppb to provide additional data, or to inform the shape of the exposure-response curve. Therefore, there is no scientific indication that adverse health effects occur below 200 ppb that would warrant protection under the NAAQS.

Nevertheless, the draft REA Planning Document is noticeably deficient in its consideration of a threshold for $SO_2$-induced bronchoconstriction. In its discussion of the 2009 REA, the EPA states that their "risk assessment approach assumes no threshold" (page 2-22). Not seeing any clarification in the 2017 REA Planning Document, the logical assumption is that the exposure-response function also does not have a threshold in the current analysis. This approach is counter to the biological principles I have laid out and the wealth of evidence available in this review.

Despite the lack of data supporting its decision, the EPA is basing very important conclusions on modeling results below 200 ppb. In its 2009 REA, the EPA itself states that, although there is uncertainty in risk estimates below 100 ppb, their model estimates that exposures at or below 100 ppb contributed substantially to risk estimates. Even in the current planning document, risk estimates in Table 4-7 identify risk at 0 ppb $SO_2$. This table seems to indicate that the probit exposure-response function is assuming a risk from $SO_2$ exposure in the absence of $SO_2$.

Therefore, we urge this committee to recommend that the EPA more thoroughly discuss its exposure-response assumptions and incorporate a threshold into the main exposure-response model, not just in the uncertainty analysis, as is currently done. Further, we strongly urge this committee to recommend the inclusion of uncertainty bounds in the REA and underlying analyses. As stated in my oral comments today and in EPA's analysis, the exposure-response data below 200 ppb are highly uncertain. Presenting a point estimate suggests a certainty in the results that is unfounded and misleading to the reader.

As you know, the EPA will likely be pushing the REA, Policy Assessment, and Proposed Rule out very quickly to meet a consent decree deadline. Per the proposed consent decree published in the January 17th edition of the Federal Register, the EPA plans on releasing all three draft documents in the next 14 months (by May 25, 2018). This is a highly ambitious goal, as it took them 25 months to do the same when the ozone standard was under review. Therefore, it is important to get any major modifications, such as the shape of the exposure-response function, incorporated now so that subsequent assessment documents will not have to be reworked and upcoming policy decisions can be made based on sound science.

Thank you, again, for the opportunity to provide these comments.