**COMMENTS BY THE TEXAS COMMISSION ON ENVIRONMENTAL QUALITY REGARDING THE RISK AND EXPOSURE ASSESSMENT PLANNING DOCUMENT FOR THE REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY STANDARDS FOR SULFUR OXIDES**

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## I. Summary of Proposed Action

On February 22, 2017, the United States Environmental Protection Agency (EPA) published a notice in the *Federal Register* (82 FR 11356) that the draft *Review of the Primary National Ambient Air Quality Standards for Sulfur Oxides: Risk and Exposure Assessment Planning Document* (REA Planning Document) is available for public review and comment.

The REA Planning Document presents the EPA's proposed approach for conducting quantitative analyses of sulfur dioxide ($SO_2$) exposures and health risks as part of its current review of the National Ambient Air Quality Standard (NAAQS). The final REA, anticipated to be released in Spring 2018, will inform decisions made in the subsequent Policy Assessment and Proposed Rule. Under a proposed consent decree (82 FR 4866), the EPA will issue its Proposed Rule no later than May 25, 2018, and will finalize the review of the primary $SO_2$ NAAQS no later than January 28, 2019. The EPA last revised the primary $SO_2$ NAAQS based on the available scientific literature supporting that standard in 2010.

## II. Comments

A. General Comments

### The EPA should continue to consider the stability and robustness of the duration of the $SO_2$ standard and maintain the 1-hour duration.

In the last review, the EPA promulgated a 1-hour $SO_2$ standard that was considered to be protective of peak 5-minute exposures. Among other reasons, the EPA found its choice of a 1-hour standard reasonable because, in combination with the form of the standard, it presented a "stable and robust regulatory target" (p. 2-4, USEPA 2017). Because there are no new controlled human studies that provide compelling evidence that the averaging time of the current 1-hour standard is inadequate to protect public health, the TCEQ strongly encourages the EPA to continue to consider the stability and robustness of their chosen standard and maintain regulatory standard continuity with the 1-hour $SO_2$ NAAQS.

### The EPA should include uncertainty bounds in its presentation of risk assessment results to allow for more meaningful communication of risk.

The EPA habitually fails to present more than a point estimate in its presentation of risk assessment results, for example in the 2015 ozone NAAQS review (USEPA 2015). Presenting a point estimate suggests a certainty in the results that is unfounded, as evidenced by the considerable uncertainty presented in Section 4.3 of this planning document. Further, this simplification of results does not allow the appropriate and important communication of uncertainties. It is crucially important that the EPA present results from published literature and its own risk analysis results with both point estimates *and* uncertainty bounds in the REA in order to allow for more accurate and meaningful communication of risk.

B. Technical Comments Related to the $SO_2$ Exposure-Response Function

**The EPA should clearly articulate the shape of the exposure-response (E-R) curve for $SO_2$-mediated decrements in specific airway resistance (sRaw).**

In its discussion of the 2009 $SO_2$ REA, the EPA states, without further justification, that its "risk assessment approach assumes no threshold" (p. 2-22, USEPA 2017). Without other clarification, the no-threshold assumption for the E-R function is presumably being retained for the current REA. However, neither the TCEQ's review of the scientific literature, nor the EPA's assessment documents for the current NAAQS review, provide a mechanistic reason to expect that the response of asthmatics to $SO_2$ would lack a threshold or that the lack of data would necessitate the conservative use of a no threshold model. Indeed, two papers cited by the EPA in the REA Planning Document [Linn et al. (1987) and Linn et al. (1990)] investigated the effects of $SO_2$ exposure in moderate-severe asthmatics. Neither paper showed a relationship between $SO_2$ exposure and clinical severity of asthma. Linn et al. (1987) showed an apparent threshold effect in both minimal-mild asthmatics and moderate-severe asthmatics and noted that responses were similar between the two groups.

The EPA has used thresholds to evaluate risk with other chemicals that elicit similar responses. For example, the EPA's Integrated Risk Information System (IRIS) has produced inhalation reference concentrations (RfCs) to be used in risk assessments. Of the 121 chemicals evaluated, several have similar modes of action (MOAs) to $SO_2$ (e.g., local absorption of an irritant leading to irritation of lung tissue, resulting in respiratory inflammation and decreased lung function). In all of these assessments, whether the data originated from controlled human studies, controlled animal studies, or epidemiological studies, the EPA assumed these MOAs to have a threshold for these critical effects.

The MOA for $SO_2$-induced respiratory effects similarly indicate the presence of a 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, is irritating to the epithelial lining of the lung. This epithelial irritation, presumably due to the bisulfite interacting with sulfur-containing bonds in cellular proteins of the epithelial lining of the lung, produces the observed bronchoconstriction effects (Sheppard, 1988). Such reflex biological responses like bronchoconstriction and cough are fundamentally understood to have a threshold, including a threshold to activate neural tissues controlling the smooth muscle tone of the conducting airways of the lung, or to orchestrate the muscular responses to produce a cough (Kubin et al., 2006). A threshold is presumed even among individuals living with asthma or chronic obstructive pulmonary disease (COPD), although the threshold for activating these responses is likely lower than the average population due to airway remodeling, higher baseline inflammation in the lung, and/or intrinsic genetic and subsequent differences in phenotypic responses (Holgate et al., 2015; Wong and Morice, 1999). As noted by the National Academies of Science (2010), "the body of experimental data suggests that 0.25 ppm may be a threshold for bronchoconstriction in asthmatics" and "0.2 ppm may be a NOEL [No Observed Effect Level] for bronchoconstriction in exercising asthmatics." Therefore, the incorporation of a threshold into the E-R model should be a component of the main model, rather than just part of the uncertainty analysis. Accurate characterization of the E-R curve is particularly important because of the conclusions that will be made based on the model output.

**The EPA should accurately portray the uncertainties of exposures to $SO_2$ at concentrations less than 200 ppb and provide a clearer justification for the use of benchmarks below 200 ppb in its analysis.**

The $SO_2$ REA Planning Document identifies its lowest benchmark level as 100 ppb, consistent with the last review (p. 4-33, USEPA 2017). The EPA states that this decision was based on the need for a value lower than 200 ppb (at which some participants exposed to $SO_2$ in a free-breathing chamber showed an effect, though not statistically significant), as well as "very limited data indicating some potential for $SO_2$-induced lung function decrements in individuals... exposed to 100 ppb $SO_2$ administered via mouthpiece" (p. 4-33, USEPA 2017). In Chapter 2 of the REA Planning Document, however, the EPA states that "there is strong controlled human exposure study evidence for $SO_2$ eliciting lung function responses within the range of tested 5-10 minute exposures (i.e., ≥200 ppb) and as low as 100 ppb" (p. 2-22, USEPA 2017). This disparity in the presentation of human controlled exposure results is misleading to the reader. The evidence of lung function responses below 200 ppb is, in fact, very limited. Linn et al. (1997) conducted the only free-breathing chamber study cited in the 2016 draft $SO_2$ ISA that delivered 100 ppb $SO_2$, and it was present in a mixture with ozone and sulfuric acid that, in comparison with filtered air, "did not result in statistically significant changes in lung function or respiratory symptoms" (p. 32, USEPA 2009). Two studies (Koenig et al. (1990) and Sheppard et al. (1981)) "observed very small changes in $FEV_1$ or sRaw" following exposure to 100 ppb via mouthpiece (p. 55, USEPA 2009); however, the differences in delivery (i.e., free-breathing chamber vs. mouthpiece) preclude their direct comparison. The EPA itself states that mouthpiece studies "cannot be directly compared to studies involving freely breathing subjects, as nasal absorption of $SO_2$ is bypassed during oral breathing, thus allowing a greater fraction of inhaled $SO_2$ to reach the tracheobronchial airways. As a result, individuals exposed to $SO_2$ through a mouthpiece are likely to experience greater respiratory effects from a given $SO_2$ exposure" (p. 2-5, USEPA 2017). The TCEQ agrees with the EPA's decision to not consider exposure via a mouthpiece to be relevant for assessment of ambient $SO_2$ exposure, which will unequivocally occur under a free-breathing scenario. The EPA should clarify the purpose of the 100 ppb benchmark. If it intends to pick an arbitrary number below the concentration shown to have an effect, then it should consistently represent it as such and provide the necessary caveats to explain the high level of uncertainty associated with the level.

The issue of the 100 ppb benchmark is further complicated by its importance in the subsequent risk analysis. The EPA notes in its review of the 2009 $SO_2$ REA that exposures to less than 100 ppb (where there is no evidence of adversity) contribute substantially to the risk estimates (USEPA 2009, 2017). The TCEQ urges the EPA to make it clear when the 100 ppb benchmark is used in the REA or related documents that there is no data showing that adverse health effects occur at 100 ppb $SO_2$. A similar caveat could also be applied to the 200 ppb benchmark because the mean response was not statistically significant at this level and could arguably fail to meet the criteria for an adverse effect.

**The EPA should reconsider using logit and probit E-R functions that estimate risk of $SO_2$ exposure at 0 ppb $SO_2$ concentrations.**

Table 4-7 provides risk estimates for population exposure to $SO_2$ based on different E-R functions. It is unclear what the risk presented at 0 ppb $SO_2$ is intended to represent. Is this the percent of the population (presumably the asthmatic population) that would be expected to experience an sRaw ≥ 100% in the absence of any $SO_2$ (i.e., the known population background incidence)? It seems likely that the background incidence of asthmatics experiencing an airway obstruction is more than $2 \times 10^{-11}$ % and, if this is the correct interpretation, this background incidence should be clarified and supported with pertinent literature citations. Alternatively, is this table showing that the probit E-R function is assuming a risk from $SO_2$ in the absence of $SO_2$? If so, this indicates that there is a flaw in this model and it needs to be reworked, such that there is a concentration of $SO_2$ where no $SO_2$-attributable effects would be expected to occur, even if that is a concentration of 0 ppb.

C. Technical Comments Related to Modeling

**Modeling data should be verified against monitored data (5-minute ambient concentrations) or experimental data (microenvironment data) to improve confidence in the model. The results of these comparisons should be presented in the REA.**

It is clear from the $SO_2$ REA Planning Document that modeling (not monitoring) will be used to assign concentrations to the simulated individuals in the APEX model. The EPA states that it did not conduct a model-to-monitor evaluation in the 2009 $SO_2$ REA because there was limited 5-minute monitoring data available for comparison. However, as the EPA notes in the current REA Planning Document, there is substantially more monitoring data available for this review. The TCEQ, therefore, encourages the EPA to use available monitored data to confirm the accuracy of the modeled exposure concentrations. Where all 5-minute $SO_2$ concentrations have not been reported to the EPA's Air Quality System, the TCEQ encourages the EPA to reach out to the quality assurance organizations of those monitors to obtain all of the 5-minute data instead of interpolating the data with yet another model.

Similarly, the EPA states that it will be relying on APEX to model microenvironmental concentrations, such as indoor air, as well as infiltration rates, reaction rates, etc. The TCEQ strongly suggests that the results of these modeling exercises be compared to the results of experiments that measured indoor or personal concentrations of $SO_2$, to confirm (or not) the modeling results. The results of both of these verification exercises should be provided in the REA to ensure confidence in the model and subsequent conclusions. If these model-monitor comparisons are not conducted, the EPA should include this omission as an uncertainty in the REA.

**The EPA should finalize which analyses will be included in the REA in the REA Planning Document.**

Although the purpose of the $SO_2$ REA Planning Document is to outline its approach for conducting exposure and health risk analyses, the EPA is still not clear on certain analyses. Specifically, the EPA needs to clarify which version of APEX will be used (the current version or the one expected to be released in Spring 2017, p. 4-2, USEPA 2017) and what new approach will be used to estimate a simulated individual's activity-specific ventilation rate (Section 3.4.3, USEPA 2017). These should be clearly articulated. If the EPA does use updated analyses, the EPA should provide a clear description of the analysis and an extended public comment period for the REA. If an updated version of APEX is used, the EPA should provide a guidance document, in addition to the extended review period, so that members of the scientific community have time to review both the model and the REA.

**While APEX is being updated, the TCEQ encourages the EPA to develop a graphic user interface for the program.**

APEX is a highly complex, intricate model that is an underpinning to several NAAQS. Currently, APEX is very difficult to operate, even for those experienced with such models. A graphic user interface would make it easier for those external to the EPA and researchers of varying levels of expertise to run the program. Greater use by peers and the public will only lead to greater, more meaningful discourse on key modeling parameters and the ultimate policy decisions that use them as their basis.

D. Technical Comments Related to Uncertainties

**If a quantitative evaluation of uncertainty is truly impossible, the TCEQ urges the EPA to use a balanced, science-grounded approach to its qualitative evaluation, and to present important uncertainties together with risk estimates.**

The EPA describes a number of uncertainties from the 2009 $SO_2$ REA (pp. 2-10 to 2-12, and pp. 2-20 to 2-23, USEPA 2017). Whenever possible, the TCEQ encourages the EPA to quantitatively consider all of these uncertainties in the upcoming $SO_2$ REA. The TCEQ understands that there are certain instances when a qualitative evaluation must be conducted. The EPA notes that it will qualitatively rate the various sources of uncertainty it identifies by subjectively "considering the relationship between the source of uncertainty and the exposure concentrations ... and the direction of influence ... (e.g., the uncertainty could lead to over- or under-estimates)" (p. 4-42, USEPA 2017). The TCEQ urges the EPA to provide clear justification for its ratings of uncertainty using information from the available scientific literature and provide equal consideration to the possibilities of over- and under-estimation of risk due to identified uncertainties. In addition, the EPA should present any qualitative uncertainty that it thinks will either have a large impact on the risk results, or that has a high knowledge-based uncertainty (where a change in the understanding of the phenomenon is highly likely to influence the EPA's interpretation of risk), whenever risk estimates are presented.

**The EPA should provide greater discussion on the adversity and variability of sRaw.**

As stated in the 2009 $SO_2$ REA and reiterated in the 2017 $SO_2$ REA PD, the EPA determined that a 100-200% change in sRaw is adverse. Intra-individual variability of sRaw is reported as being quite high and can be influenced by age, gender, disease status, concomitant health issues (e.g., infections or allergies), lung anatomy, and likely additional unknown factors (Mahut et al., 2009; Pekka Malmberg et al., 1999). Likewise, it appears that different laboratories choose to calculate flow rates differently, making inter-laboratory variance for the measure of sRaw a variable that needs to be addressed (Kaminsky, 2012; Strohl, et al., 2012). Further, upon review of available data, particularly the body of data published by pulmonologists, it is unclear if a 100-200% change in sRaw is actually adverse. Although the 2009 REA provides some discussion on the determination that a change of 100-200% in sRaw is adverse, no studies were cited to support this view. The EPA should provide scientifically-supported justification for its determination that changes in sRaw of 100-200% are adverse.

**The EPA needs to justify the representativeness of the four model cities.**

The EPA's analysis in the $SO_2$ REA Planning Document indicates that "among monitors with design values at or below 75 ppb, the number of days with maximum 5-minute concentrations above 200 ppb ranged from zero to 22 (Appendix B)." The data in Appendix B indicates that monitors with design values that are at or below 75 ppb and have 5-minute max concentrations that exceed 200 ppb occur at a frequency of less than approximately 7 days a year. The monitor that exceeded 75 ppb on 22 days appears to be an outlier. It is important to consider and present these outlier results separately, because $SO_2$ may be emitted from a source that does not have relevance to typical $SO_2$ exposure (e.g., volcanic emissions). Therefore, it may be prudent to be selective when choosing the areas to study. Selecting an area that includes a monitor that is an outlier could bias the results of the risk assessment, especially if the results of the risk assessment are to be applied nationally.

Further, it would be helpful if the EPA would provide additional discussion about the purpose of the model cities. It is unclear from the discussion in the REA Planning Document if the model cities are intended to represent average air quality in the nation or more of a worst-case

scenario. Additional contextual details about the selected areas (Brown County, Wisconsin; Cuyahoga County, Ohio; Hillsborough County, Florida; and Marion County, Indiana), such as number and type of emission sources, geographic and meteorological anomalies that impact $SO_2$ fate and transport, and demographic data important to understanding asthma prevalence, should be provided in the REA to provide greater confidence in the applicability of the EPA's assessment to the entire nation.

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