

Comments by the Texas Commission on Environmental Quality Regarding the Draft Public Health Assessment for Corpus Christi Refinery Row

Background

The Texas Commission on Environmental Quality (TCEQ) is the lead environmental agency for the State of Texas. Each year, the TCEQ invests millions of dollars and numerous staff hours to, among other things, monitor air quality, evaluate potential emission sources, investigate existing emission sources, and study the risks of environmental pollutants in areas across the state so that it can continue to protect the state's public health and natural resources. In Corpus Christi, in particular, the TCEQ conducted a broad, multimedia environmental investigation in 2010 and 2011 to determine the environmental impact from volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons, and total petroleum hydrocarbons in soil and groundwater in the Hillcrest community. The investigation found that all measured pollutant levels were below a level of health concern (TCEQ 2011a,b). Moreover, TCEQ continues to invest in extensive ambient air monitoring in the Corpus Christi area, which is in addition to the monitoring conducted by other entities. The TCEQ's health effect evaluations of these data are available online at:

<https://www.tceq.texas.gov/toxicology>.

On August 29, 2016, the Agency for Toxic Substances and Disease Registry (ATSDR) released its draft Public Health Assessment (PHA), Summary Brochure, and Odors Fact Sheet for the Corpus Christi Refinery Row area for a 60-day public comment period. The draft PHA provides an evaluation of air monitoring data collected by TCEQ from 1980-2010, monitoring data collected by industry from 1996-2010, and monitoring data collected under the Corpus Christi Air Quality Project from 2005-2010. All three ATSDR documents provide a set of conclusions about the public health risk to monitored concentrations of air pollutants and the draft PHA lists seven recommendations as a result of their evaluation.

General Comments

ATSDR uses incorrect risk assessment methods and the draft PHA and brochure provide incorrect conclusions. ATSDR should use the correct methods and provide a more balanced discussion on potential risks of air quality in Corpus Christi.

ATSDR's PHA violates common risk assessment principles and procedures, including appropriately evaluating and taking into account both exposure and toxicity. An elementary step in assessing the potential for adverse health effects in the public is determining if and how the public would be exposed to pollutants and how certain sample results are representative of public exposure. That is, the assessment of actual public health risk/hazard (as opposed to mere hypotheticals not based in reality) requires that the public was actually exposed to the sample concentrations being evaluated. However, ATSDR did not appear to have fully considered the potential for

public exposure in the draft PHA. ATSDR's most egregious error in this context is the evaluation of reported **on-site** facility concentrations as indicative of public exposure. On-site facility measurements cannot be representative of *public* exposure because no member of the public would ever have been in that location and in such proximity to the emission source. Further, ATSDR's consideration of facility employee exposure is inappropriate for a *public* health assessment. The exposure scenarios of these two groups of people are entirely different and requires entirely different analyses.

In addition to potential for exposure, ATSDR failed to consider several important factors regarding sampling duration. In order to be useful for risk assessment, a sample's duration needs to be considered in terms of how well it represents public exposure and how relevant it is to available toxicity metrics or comparison values (CVs). This is important because toxic effects to a given pollutant concentration can vary dramatically under different exposure durations (i.e., a low chemical concentration delivered over a short period may not be toxic, but the same amount delivered every day over a lifetime may be toxic). However, throughout the draft PHA, ATSDR compares samples collected over very short durations (e.g., 20-seconds or 1-hour) to CVs designed around much longer exposure durations (e.g., 36-hours or 14 days). This comparison method is wrong and disregards basic toxicological and risk assessment principles. Data are available to calculate air concentrations for the time duration appropriate for the CV. Therefore, if ATSDR intends to use an acute Minimal Risk Level (MRL) that is based on 36 hours to 14 days of exposure, ATSDR should calculate rolling 36 hour and rolling 14 day exposure concentrations of the chemical from continuous automated gas chromatograph (autoGC) data, rather than just using the maximum measured instantaneous, 5-minute, or hourly sample. Further, in its chemical mixture analysis, ATSDR discounts the millions of ambient monitoring data points collected over the 30-year study period in favor of the impossible hypothetical scenario that the public was exposed to the maximum concentration of each of the air pollutants at one location, simultaneously, and for the same duration. ATSDR admits that this scenario did not happen, but continued to provide conclusions anyway. Again, if the exposure could not have occurred, neither the analysis nor its conclusions have a place in the PHA.

ATSDR also needs to re-evaluate the CVs that are used in the draft PHA. Many of the TCEQ values represented are incorrect. Tables 1 and 2 in these comments provide corrected values that ATSDR should use in the final PHA.

In addition to these basic methodological problems, ATSDR's conclusions in the Executive Summary and brochure are unnecessarily inflammatory and do not reflect the caveats, limitations, and uncertainties provided in the actual analysis. ATSDR highlighted any instance of an exceedance of a CV, even if such a comparison was inappropriate, and concluded that breathing the air could potential harm people's health. However, these conclusions lack any context and even lack ATSDR's own noted limitations, which include the limited nature of exceedances (in some cases, only 2 single exceedances in 30 years of monitoring), when these exceedances may have occurred (many were over 10 years ago), and the fact that the CVs themselves do not represent a level at which adverse health effects are expected to occur. Specific details of these and other serious flaws are discussed more fully in the Technical Comments below.

In the final PHA, ATSDR needs to reconsider the accuracy and appropriateness of the CVs it uses, both with respect to the reliability of the studies on which they are based and the sampling durations that they represent. Specific attention should also be paid to identifying the representativeness of air samples collected in Corpus Christi before those samples are evaluated for potential public health concerns. Finally, ATSDR should provide the public with the necessary context to understand any conclusions and recommendations provided in the PHA.

ATSDR should better articulate which state agencies were provided an initial review of the PHA.

ATSDR states that “the affected state” was provided an opportunity to review and provide comment prior to the public release of the PHA; however, only the Texas Department of State Health Services was actually afforded this review. The TCEQ was not provided an opportunity to review this document prior to its public release, even though ATSDR relied so heavily on TCEQ data and CVs. Had TCEQ been given an opportunity to review an initial draft, many of the basic methodological problems with this assessment and corrections to CVs could have been addressed at that time.

Technical Comments

Chemical Risk Assessment

Benzene

Short-Term Exposure

Careful consideration of the studies that are the basis for health conclusions is critical in risk assessment but lacking in the draft PHA. In the case of benzene and potential neurological effects, the draft PHA largely relies on the findings of Midzenski et al. (1992), as cited in ATSDR (2007). Among other problems, Midzenski et al. (1992) failed to accurately measure the actual benzene exposure concentrations producing effects in their study. The authors indicate that the statistically significant increase ($p \leq 0.04$) in neurological effects (e.g., dizziness and nausea) occurred in workers with more than 2 days (16 hours) of exposure to benzene concentrations ranging from “greater than 60 ppm [personal communication with marine chemist]” to 653 parts per million (ppm) and could have been even higher due to sampling breakthrough. Consistent with this, the study authors estimated that the air concentration corresponding to the detection limit for benzene in the fuel would be 987 ppm in fuel tank air. Actual benzene concentrations were not measured during exposure, but only after exposure had ended. Moreover, workers were co-exposed to a plethora of marine diesel fuel compounds, whose concentrations were admittedly not characterized but produce similar symptoms (e.g., n-hexane, xylenes, toluene). Consideration of these factors precludes identification of any reliable LOAEL for benzene from this study and, moreover, any use of it in drawing conclusions about potential neurological effects in a human health risk assessment of ambient air sampling results.

In addition to using this unreliable LOAEL to draw health conclusions, the draft PHA fails to consider the importance of the study’s exposure duration. Workers in the Midzenski et al. (1992) study that experienced a statistical increase in reported

neurological effects (e.g., dizziness, nausea) were exposed for more than 2 days (16 hours). This exposure scenario/duration is completely different than that represented by the instantaneous air sample reviewed in the draft PHA using Midzenski et al. (1992). More specifically, the one sample cited to exceed the unreliable Midzenski et al. (1992) LOAEL was collected on a facility's property (an instantaneous sample with a benzene concentration of 370 ppm that was collected downwind of an air intake on a tank's nonoperational thermal oxidizer in July 2000). This sample is clearly not representative of any public exposure, much less a public exposure that would match the greater than two-day worker exposure associated with effects in the Midzenski et al. (1992) study. Furthermore, it was an instantaneous (i.e., less than 30-second) on-site sample (TCEQ 2000a), not a 30-minute sample as the draft PHA indicates, and comparison to the Midzenski et al. LOAEL was inappropriate. The same critical considerations of representativeness and duration would apply to using the 30-minute, 300 ppm LOAEL for similar neurological effects from Flury (1928) to evaluate this on-site facility sampling result. Thus, the draft document first fails to consider that the public was not exposed to the on-site concentration reported to be above these cited LOAELs, and then compounds this error by not appropriately considering sample duration (instantaneous, less than 30 second) relative to the exposure durations associated with the LOAELs. ATSDR attempts to provide a hypothetical transient public exposure scenario (e.g., members of the public passing near the property boundary where the higher benzene level was sampled on site), but fails to address the likelihood of this exposure (e.g., the area is entirely industrial and there are no bike paths, parks, or recreational areas encouraging this activity near the facilities), dispersion with distance, or the issues with exposure duration (e.g., exposure would not be long enough for appropriate comparison to the provided LOAELs). No public exposure occurred to these on-site concentrations; therefore, there is no potential for adverse health effects.

Similar problems occur in the evaluation of potential hematological (e.g., blood cell count) effects. First, the cited short-term samples were not representative of public exposure. The draft PHA agrees, stating, "All of the samples with benzene concentrations above the human equivalent LOAEL (2,550 ppb [parts per billion]) were collected during mobile monitoring events on facility properties located outside of any neighborhood." As mentioned above, the highest grab sample, which was instantaneous (i.e., less than 30 second) and not 30 minutes (TCEQ 2000a), as indicated in the draft PHA, was collected on a facility's property. Additionally, the second (18 ppm) and third (3.3 ppm) highest samples cited in the draft were also instantaneous (i.e., less than 30-second) grab samples collected on facility property (at a frac tank with an open manhole cover and at a benzene seep, respectively) (TCEQ 2000b,c). No public exposure occurred or could possibly occur to these on-site concentrations.

Second, ATSDR failed to appropriately consider the differences in exposure duration between the health effects study used as the basis of the MRL and the samples in the draft PHA. The draft assessment cites a human equivalent concentration LOAEL of 2.55 ppm for decreased lymphocytes in mice from the Rozen et al. (1984) study, which is actually the study LOAEL of 10.2 ppm adjusted from a single 6-hour exposure to 24 hours. However, ATSDR fails to consider that the mouse exposure that produced effects was 6 hours/day for 6 days for a total of 36 hours. Consequently, there is an inappropriate miss-match of durations in ATSDR's evaluation. A LOAEL based on 36

hours of exposure that produced effects cannot simply be compared to less than 30-second grab sample results or other such short-term sampling duration results (e.g., 15-minutes, 30-minutes, or 1-hour), nor can an acute MRL designed to evaluate exposure up to 14 days (up to 336 hours) be used to reliably assess the risk/hazard associated with such short-term sampling results. Given that 36 hours of exposure produced the effects, it would have been more appropriate to compare the LOAEL of 10.2 ppm to 24-hour concentrations representative of public exposure. Further, the decreased lymphocyte count in Rozen et al. (1984) at 10.2 ppm appeared to be within the normal range of variability (Jackson Laboratory 2007), raising questions as to biological significance and adversity of the reported effect and whether the concentration is most accurately described as a LOAEL.

In regard to the highest benzene value cited as measured in continuously collected samples from a stationary air monitor (1,014.02 ppb), the draft states, “Although this 1,014.02 ppb value is below the human equivalent LOAEL of 2,550 ppb from animal studies, the studies did not document a NOAEL, and it is possible for health effects to occur at lower concentrations than the LOAEL.” However, the cited concentration from the Huisache monitor (Table 11B) is for only a 15-minute duration, and to compare a 15-minute concentration to a LOAEL due to 6-hour exposure/day for 6 days (a total of 36 exposure hours) is scientifically indefensible. Furthermore, while the Rozen et al. (1984) mouse study did not provide a NOAEL, subchronic data from a reliable human study can provide a conservative NOAEL-like value to better understand any potential for hematological effects following shorter-term exposure. More specifically, using benchmark modeling, USEPA determined a subchronic point of departure of 7.2 ppm for an 8-hour work day. According to USEPA, the decreased absolute lymphocyte count (ALC) noted in the study is a very sensitive endpoint and there is no evidence that a decrease in ALC is related to any functional impairment at benzene levels near the benchmark concentration level (USEPA 2002). Note that under current guidance, USEPA would not divide this point of departure further by a factor to approximate a NOAEL because of the use of benchmark modeling. Rather, the point of departure is treated as a NOAEL-like value. This 8-hour value (7.2 ppm) and even the value extrapolated to continuous subchronic environmental exposure ($7.2 \text{ ppm} \times (10/20) \times (5/7) = 2.6 \text{ ppm}$) are higher than the highest benzene value cited as measured in continuously collected samples from Corpus Christi area stationary air monitors, indicating that ATSDR’s conclusion that harmful effects could have occurred is incorrect. Similar to the 15-minute stationary monitor concentration evaluated in the draft PHA, the hypothetical transitory exposure assumed in the draft document and brochure (p. 3) for a member of the public walking or biking by a property boundary site where a higher benzene level was sampled somewhere on site would be too short in duration for comparison to a study LOAEL where effects were produced by a much longer exposure duration (e.g., comparison of a 15-minute concentration to a LOAEL resulting from 36 hours of exposure).

Because of these significant scientific shortcomings, the numerous statements in the draft PHA and brochure indicating that breathing measured benzene concentrations in the past and present could potentially harm people’s health are misguided. These conclusions are purely the result of the unreasonable assumption of hypothetical prolonged public exposure to instantaneous (i.e., less than 30-second) on-site sample

concentrations and/or grossly inappropriate comparisons to available toxicity factors and/or their underlying LOAELs (e.g., indefensible comparison of a 15-minute concentration to a LOAEL due to 36 hours of exposure). Although not mentioned in the conclusions, ATSDR's own exposure investigation (EI) agrees, stating that "Benzene blood levels measured during the EI were compared with NHANES benzene blood level data. Results suggested that compared with a sampling of the United States general population, no apparent, unusual benzene exposure occurred in the Refinery Row area" (p. 370 of the draft PHA). Therefore, ATSDR should revise its analysis and, at a minimum, include important caveats in both the draft PHA and the brochure to transparently put conclusions into proper context.

Long-Term Exposure

ATSDR's calculation of excess cancer risk goes against standard risk assessment principle. Specifically, basic principles in risk assessment dictate that lifetime excess cancer risk can be calculated by multiplying an inhalation unit risk factor (URF) by the lifetime average exposure concentration. As excess risk can only truly be appropriately estimated based on the *lifetime* average exposure concentration, an estimate based on a short duration or even an annual mean is not truly appropriate. However, the draft PHA (p. 35) chooses to: (1) calculate lifetime excess cancer risk based on the "highest Refinery Row mean," which apparently is only a 7-year mean based on pre-2005 data from the Huisache monitor (Table 12B); and (2) ignore 2005-2010 data from the same site (Table 13B), the addition of which would provide a longer and more representative average (albeit less than lifetime) for risk estimation. Long-term monitoring trends are provided in the Ambient Monitoring Data section below. The excess risk estimate (5.5E-05) cited in the draft PHA and referred to in the draft brochure (p. 3, 5) is simply inappropriately calculated and incorrect, contributing to the inaccurate and overestimated 2E-04 excess risk cited in the brochure (p. 3, 5). Moreover, the USEPA URF used by the draft PHA is outdated (USEPA 1998) and a much more recent and definitive dose-response assessment developed under peer-reviewed guidelines is available (TCEQ 2007, 2015a).

Lastly, while the draft PHA properly alludes to the fact that increased leukemia risk has been shown to be causally related to worker exposure to very high long-term benzene concentrations (e.g., USEPA (1998) states that the agency is fairly confident that the risk of leukemia increases at 40 ppm-years of occupational benzene exposure, which equates to a lifetime (i.e., 24 hours/day for 76 years) environmental exposure level \approx 120 ppb), the draft PHA (p. 7, 57, 63) and brochure (p. 5) improperly imply to the lay reader that inhalation exposure to benzene is causally related to liver cancer in humans. The human database is robust for identifying the cancers causally related to chronic inhalation exposure to benzene by humans and there is an extensive, long-term human experience with very high, long-term inhalation exposure to benzene in the workplace. Examination of Table 3-1 in ATSDR (2007) shows that leukemia is the only cancer type shown to be causally related to human benzene exposure. More recently, Section 3.2.1.7 of ATSDR (2015) does not even mention liver cancer as a potential benzene-induced cancer in humans.

Total Chromium

Short-Term and Long-Term Non-Carcinogenic Risk

The draft PHA provides incorrect TCEQ CVs. The correct TCEQ health-protective values for short-term (24-hour) and chronic (non-carcinogenic) effects are 1.3 and 0.22 $\mu\text{g}/\text{m}^3$, respectively (TCEQ 2014). Table 1 details all corrections to TCEQ health-based CVs originally represented by ATSDR in the draft PHA.

In order to conduct the most conservative assessment possible, the draft PHA assumes that total chromium measured in ambient samples is entirely composed of hexavalent chromium (CrVI). The draft PHA correctly notes that this assumption likely results in the overestimation of risk. However, the purpose of a public health risk assessment should be to estimate plausible, reasonably predictive estimates of risk; therefore, the assumption that CrVI is equal to total chromium is unreasonable. For example, based on a year's worth of sampling conducted in Midlothian, Texas, only about 1% of total chromium was in the hexavalent form. Similarly, USEPA (2015) estimated a CrVI to total chromium (measured in total suspended particles (TSP)) ratio of 1.25% for the 2011 National Air Toxics Assessment (see Appendix B of USEPA 2015). Because both monitored and modeled data indicate such a small percentage (1-1.25%) of total chromium is CrVI, ATSDR's comparison of total chromium to CrVI CVs is unjustified. Either of these estimates would provide a more defensible evaluation of likely CrVI concentrations in the Corpus Christi PHA, particularly since ATSDR did not note the presence of an emission source that would likely emit greater CrVI in close proximity to monitoring sites. Based on these data-informed results, CrVI risk/hazard would be 80-100 times lower than the estimates ATSDR provided in the draft PHA.

Even if ATSDR is intent on making the technically incorrect comparisons under the pretext of merely being as conservative as possible, monitoring data would still indicate that there is no elevated risk of potential adverse health effects. The maximum 24-hour, total chromium concentrations cited in the draft PHA are below TCEQ's short-term (24-hour) CrVI CV of 1.3 $\mu\text{g}/\text{m}^3$ (TCEQ 2014). Additionally, in its long-term public health evaluation, ATSDR fails to use mean concentrations that adequately represent chronic exposure (e.g., means are calculated based on less than one year to three years of sampling, which is significantly less than the duration of 7-years or 10% of a lifetime that is typically used by regulatory agencies such as USEPA and TCEQ as a cut-off between subchronic and chronic exposure). Any comparison to concentrations that represent less than a lifetime of exposure should only be used for screening, not for establishing public health risk. Further, even the highest total chromium means in the draft PHA are well below TCEQ's more current 2014 chronic (non-carcinogenic) CrVI CV of 0.22 $\mu\text{g}/\text{m}^3$ (TCEQ 2014).

Lastly, ATSDR should remove the discussion of Lindberg and Hedenstierna (1983) from its short-term exposure section. The workers in the study were exposed subchronically for an average of 2.5 years; therefore, the CrVI LOAEL from this study is not relevant in regard to evaluating effect levels due to short-term exposure. The draft PHA seems to recognize this (p. 37), so it is unclear why this discussion is included in the short-term exposure section.

Cancer Risk

As stated in the benzene cancer assessment comments above, excess risk can only truly be appropriately estimated based on the *lifetime* average exposure concentration; an estimate based on a short duration or even an annual mean is not truly appropriate. However, ATSDR calculates lifetime excess cancer risk (p. 38) based on a 3-year total chromium mean for the Dona Park monitor (Table 16B). Further compounding the issues with this method of excess cancer risk calculation is the issue of such a small percentage of total chromium actually being in the more toxic CrVI form. ATSDR must provide adequate justification for using this extreme assumption, which should include an evaluation of the emission source types in close proximity to monitoring sites. Because of the incorrect calculation method and incorrect assumption of chromium state, the chromium excess risk estimate (2.0E-05) cited in the draft PHA and referred to in the draft brochure (p. 3, 5) is incorrect. This risk estimate then contributes to the inaccurate and overestimated combined excess cancer risk of 2E-04 cited in the brochure (p. 3, 5). Finally, the USEPA URF used by the draft PHA is outdated (USEPA 1984) and based on a very outdated occupational study (Mancuso 1975). A much more recent and scientifically-defensible dose-response assessment and URF (2.3E-03 per $\mu\text{g}/\text{m}^3$) developed under peer-reviewed guidelines is available (TCEQ 2014, 2015), which was published in a peer-reviewed journal (Haney et al. 2014) and presented by invitation at the Risk Assessment of Metals Platform Session of the 2014 Society of Toxicology conference.

Cadmium

The draft PA cites incorrect TCEQ CVs. The correct TCEQ health-protective values for short-term (24-hour) and chronic (non-carcinogenic) effects are 0.55 and 0.011 $\mu\text{g}/\text{m}^3$, respectively. Additionally, the TCEQ has recently derived an updated URF for cadmium of 4.9E-04 per $\mu\text{g}/\text{m}^3$ and (TCEQ 2016), which has been published in a peer-reviewed scientific journal (Haney 2016).

For the evaluation of short-term exposure, the draft PHA incorrectly uses an acute MRL (0.03 $\mu\text{g}/\text{m}^3$) designed to evaluate exposure up to 14 days (up to 336 hours). As explained above, the duration of this CV means that it cannot be used to reliably assess the risk/hazard associated with a 24-hour sampling duration. The TCEQ 24-hour CV of 0.55 $\mu\text{g}/\text{m}^3$ is appropriate for such a comparison. Because of the differences in durations (i.e., the acute MRL is designed to be applied up to a duration that is 14-fold greater than that of TCEQ's 24-hour CV), the difference between the acute MRL and the 24-hour TCEQ CV is reasonable. The maximum concentrations cited (0.175 and 0.06 $\mu\text{g}/\text{m}^3$) were well below the 24-hour TCEQ CV.

In regard to long-term exposure, ATSDR fails to justify how any conclusions can be made on a chemical that is rarely detected. Because cadmium was detected in less than 20% of the samples, ATSDR chose to evaluate long-term exposure risk using the highest 95th percentile value (p. 36, 52). The draft PHA is unclear on exactly how these values were calculated or why ATSDR believes this calculation would be representative of long-term concentrations for a chemical that is mostly not detected in the air. Additional discussion of ATSDR's data censoring method are provided in the Ambient Monitoring Data section below.

As with the chromium cancer assessment, ATSDR failed to follow basic risk assessment principle and chose to calculate cancer risk using a concentration not representative of even chronic, much less lifetime, exposure. However, unlike the other cancer assessments, ATSDR used a concentration that was not even representative of a single year of public exposure. Consequently, the excess risk estimate (1.1E-05) cited in the draft PHA and referred to in the draft brochure (p. 3, 5) is grossly incorrect, contributing to the inaccurate and overestimated combined excess cancer risk of 2E-04 cited in the brochure (p. 3, 5). Lastly, the draft PHA uses an outdated USEPA cadmium URF derived in 1985 (1.8E-03 per $\mu\text{g}/\text{m}^3$; USEPA 1985) for environmental exposure using lung cancer data from a now outdated occupational study (Thun et al. 1985 was first updated in Stayner et al. 1992 and later in Park et al. 2012). Although USEPA conducted a draft assessment in 1999 (USEPA 1999), the URF on USEPA's Integrated Risk Information System has not been updated in three decades (i.e., since 1985). Not disregarding the comments above on representative chronic (e.g., lifetime) exposure concentrations, ATSDR should use the TCEQ updated URF for cadmium of 4.9E-04 per $\mu\text{g}/\text{m}^3$ (TCEQ 2016), which was been published in a peer-reviewed scientific journal (Haney 2016) and is based on the latest data (Park et al. 2012) for the same cohort used by USEPA.

1,2-Dibromoethane

As with cadmium, ATSDR chose the suspect method of calculating a 95th percentile value to represent long-term concentrations because less than 20% of the TCEQ canister samplers had 1,2-dibromoethane concentrations above the minimum reporting limits. Additional discussion of ATSDR's data censoring method are provided in the Ambient Monitoring Data section below. Because 1,2-dibromoethane is so infrequently measured near Refinery Row, this calculated value cannot be considered representative of ambient conditions or public exposure. Further evaluation of excess risk of long-term adverse health impacts, either cancerous or noncancerous, is therefore inappropriate and ATSDR's conclusion that there was a potential for concern prior to 2005 (p. 180, Table12B) is unsupported. ATSDR needs to better justify their use of this method and properly account for the vast uncertainty associated with chemicals that are below analytical detection limits over 80 percent of the time.

Naphthalene

The draft PHA should provide a better representation of the monitoring data used in the analysis. Specifically, the text does not discuss which locations measured naphthalene, over what duration, and at what frequency. ATSDR needs to clarify which locations had available data and, if means are provided, exactly what time frame the mean represents.

As with its other cancer assessments, ATSDR inappropriately calculates the lifetime excess risk of cancer due to naphthalene exposure. The draft PHA admits that naphthalene was rarely detected in the Refinery Row area over the study period and that only one site had enough detections to calculate an annual average. ATSDR's use of the maximum mean for an unknown time period before 2005 to calculate *lifetime* excess cancer risk is inappropriate and likely does not represent risk to the entire area. The draft PHA should provide a more complete representation of air monitoring trends in the Refinery Row area and refrain from calculating excess risk if there are not enough long-term data to do so correctly.

Even with this incorrect calculation method, the excess cancer risk due to naphthalene (1.0 E-5) is well within the acceptable excess risk range of numerous regulatory organizations, including USEPA and TCEQ. This important context, however, was not provided in ATSDR's presentation of the risks in the draft PHA or the brochure.

Particulate Matter

The ATSDR's discussion on the potential health impacts due to particulate matter (PM) exposure is perhaps one of the most confusing in the draft PHA. Oddly, ATSDR spends significant effort explaining air quality guideline levels established by the World Health Organization, though those values are not used in the actual evaluation. This discussion should be eliminated or truncated so that more attention is placed on the more relevant CV for the analysis.

When the draft PHA does discuss the CV used in the analysis, it incorrectly portrays key details of the particulate matter NAAQS. First, there was no NAAQS for particulate matter of 2.5 micrometers or less in diameter (PM_{2.5}) prior to 2006, so the draft PHA's comparison of sampling data collected prior to 2005 to the 2012 NAAQS is inappropriate. Second, the draft PHA states that the NAAQS are "based on technological feasibility and economic considerations in addition to public health priorities" (p. 42). In fact, the federal Clean Air Act, as upheld by court rulings, is clear that economic and technical feasibility of attaining the standards are *not* considerations in setting or revising a NAAQS. The primary NAAQS are health-based standards. Third, the USEPA Administrator, upon advice from staff and the Clean Air Scientific Advisory Committee, uses three elements (e.g., the averaging time, level, and form) to set the NAAQS at a level that is protective of public health with an adequate margin of safety; therefore, all three pieces of the NAAQS are important. ATSDR disregards the form of the NAAQS (e.g., the 98th percentile averaged over three years) in its evaluation and instead only evaluated whether 24-hour samples were numerically higher than the level of the NAAQS. This method is completely inappropriate. The draft PHA goes on to highlight two individual samples that exceeded the level of the current NAAQS out of the over 2,000 samples collected during the study period. Beyond this inappropriate comparison, the assertion that these two individual samples collected prior to 2005 justify the conclusion that there has been an increased likelihood of respiratory and cardiopulmonary symptoms in sensitive individuals is unfounded. The truth of the matter is that the Corpus Christi area is now and has always attained the primary NAAQS for PM, meaning that, according to EPA, exposures to ambient concentrations do not result in the potential for adverse health effects in the public. More information about monitored trends is available in the Ambient Monitoring Data section below.

Finally, with regard to the long-term health evaluation, the draft PHA over-simplifies the results of the studies that were considered in the World Health Organization air quality guideline (e.g., Pope et al (1993), Pope et al. (2002), Dockery et al. (1993), HEI (2000), and Jerrett (2005)). All of these studies exhibited significant heterogeneity of responses between study cities (i.e., in many cities, increasing PM_{2.5} concentrations was associated with *decreased* premature mortality) and a large degree of uncertainty due to study design. Therefore, ATSDR's statement that "when annual mean concentrations are in the range of 11-15 µg/m³, health effects can be expected" is incorrect.

Sulfur Compounds

To date Corpus Christi has four active ambient air monitors along refinery row that report sulfur compounds (e.g., two hydrogen sulfide monitors and four sulfur dioxide monitors). Five of the seven ambient air monitors that ATSDR included in the draft PHA have been decommissioned, partly because monitored concentrations were consistently below levels of health concern and suggested minimal public risk to sulfur compound-related health effects. Of the four remaining monitoring sites, the J.I. Hailey site is inappropriate for use in a *public* health assessment because it is in area where the general public has limited to no access and Corpus Christi Huisache is no longer representative of public exposure because the nearby area is completely industrial. ATSDR itself agrees, stating that both sites are in sparsely populated areas of Refinery Row and not near any homes (Section 10 pg. 59). Therefore, ATSDR should remove these sites from the analysis in its final PHA and refocus its evaluation on those monitors that actually represent the potential for public exposure.

Hydrogen Sulfide

Conclusion 1 of the draft PHA, which states that “Two stationary monitors (Huisache and JI Hailey) and several mobile monitors found maximum hydrogen sulfide levels that are at and approaching health effect levels” is wrong and unsupported by the data. In the past, peak hydrogen sulfide concentrations at stationary monitors have infrequently exceeded the state standard and the ATSDR’s MRL, but even the maximum concentration monitored in the area was only 25% of the LOAEL of 2,000 ppb referenced in the draft PHA. However, even minor exceedances of the state standard and MRL have been infrequent and there have been no exceedances of the 30-minute state standard for hydrogen sulfide, even at monitors in the industrial area, in over four years. Further, ATSDR needs to ensure that only 30-minute to 1-hour samples collected during mobile monitoring trips are compared to the CV and provide justification for their insinuation that maximum air concentrations measured from singular samples during these trips between 1993 and 2002 would represent current conditions. Finally, as stated previously, these maximum concentrations were measured in locations that were not representative of public exposure and ATSDR’s hypothetical transient public exposure scenario over 30-minutes to 1-hour is unrealistic, making the usefulness of these comparisons questionable in this *public* health assessment. Therefore, this misleading language in the draft PHA needs to be revised to better represent the data.

The TCEQ has noted that historical ambient hydrogen sulfide levels have been measured above published odor thresholds. TCEQ has worked with area facilities and conducted additional air quality evaluations to determine and abate the source of these odors. As a result of numerous efforts, the frequency of odor complaints has decreased dramatically. Residents concerned about odors are encouraged to file an environmental complaint with the TCEQ by using an online form, available at <http://www2.tceq.texas.gov/oce/complaints/index.cfm>, or calling the toll-free complaint line at 1-888-777-3186.

Sulfur Dioxide

The available scientific literature is not supportive of ATSDR’s suggestion that sulfur dioxide exposure causes cardiovascular effects. In April 2016, the USEPA’s Clean Air

Scientific Advisory Committee (CASAC) provided comments on the USEPA's most recent Integrated Science Assessment for Sulfur Oxides, which provides an assessment of the available health effects literature related to sulfur dioxide. CASAC specifically noted that the evidence of short-term exposure causing cardiovascular effects is inadequate due to potential confounding from co-pollutants in the limited studies showing an effect. Therefore, the draft PHA's statement that sulfur dioxide concentrations measured during mobile monitoring trips could potentially cause cardiovascular health effects (p. 46) is unsupported and should be removed.

In addition, the draft PHA should provide better discussion of the representativeness of the monitoring data it states are evidence that sulfur dioxide concentrations could cause respiratory health effects in sensitive populations (p. 46). Specifically, the draft PHA details that maximum 1-hour sulfur dioxide concentrations exceeded the MRL in 1.5% of the stationary ambient samples, but does not provide the important caveat that most of these exceedances occurred at monitors that were not representative of public exposure (e.g., JI Hailey and Port Grain Elevator) or that the highest concentration of 630 ppb was measured prior to 1997 and that subsequent sampling showed maximum concentrations had decreased to 76 ppb for the 1998-2004 period and 19.22 ppb for the 2005-2010 period. Further, the draft PHA highlights 5-minute, peak/instantaneous, 30-minute, and hourly concentrations captured during mobile monitoring events, most of which were reported in locations that would not represent public exposure (e.g., in a heavily industrialized area that the public would not frequent) and it especially would not represent air quality in locations where sensitive members of the public would be exercising. The public health relevancy of rarely measured, maximum values in such industrial areas are questionable.

Finally, the draft PHA and brochure misrepresent the significant limitations of the ATSDR's analysis. Although incorrect, the draft PHA evaluation indicates that maximum sulfur dioxide levels measured in ambient samples would only have affected sensitive members of the population while exercising and maximum levels in mobile monitoring samples would have affected the general population. Page 47 of the draft PHA then includes the important caveat that "after the exposure has ended, these effects will subside." This caveat is not provided in the Executive Summary or the brochure, which the public would be more likely to consult. Further, the CV used in the evaluation is likely very conservative. The ATSDR MRL for sulfur dioxide of 10 ppb is based on a 1998 evaluation of a study with a minimal LOAEL; specifically, slight changes in airway responsiveness in a subset of exercising asthmatics in Sheppard et al. (1981). USEPA's more recent evaluations of the available data indicate that one-hour concentrations of 75 ppb are protective of sensitive members of the population with an adequate margin of safety (USEPA 2010). USEPA determined that this level also offers adequate protection from elevated 5-minute exposures.

ATSDR should revise its conclusions regarding the potential risk of exposure to sulfur dioxide levels to accurately reflect the limitations of its analysis and the potential for public exposure.

Chemical Mixtures

In general, the chemical mixture assessment in the draft PHA is noticeably deficient in context. ATSDR began its evaluation by collecting the maximum sample concentrations and maximum means and, assuming an additive effect, added the toxicities together to calculate the potential risk of short-term and long-term exposures, respectively. This method, however, is flawed. For the calculated risk to be relevant, a person would have to have been exposed to the maximum concentrations simultaneously. ATSDR itself agrees that this exposure did not occur because maximum concentrations occurred in different years and at different locations. For ATSDR to then state, even qualitatively, that “exposure to mixtures of these contaminants could lead to temporary respiratory effects such as nasal and throat irritation, shortness of breath, and neurological effects” is entirely incorrect. Again, basic toxicology principles dictate that if the exposure did not occur (i.e., simultaneous exposure to maximum concentrations), there could never be a resulting effect.

In the long-term non-carcinogenic evaluation, the ATSDR compiles several problems to over-predict the risk/hazard to the respiratory system. In general, ATSDR calculated the chemical-specific hazard quotient by dividing the highest mean concentration by its MRL. However, the MRL for benzene is based on hematological effects, not respiratory effects. ATSDR admits that respiratory effects occur “roughly two orders of magnitude higher than hematological effects” (p. 49), but does not adjust its calculation method nor qualify its resulting respiratory hazard index. Given that the benzene hazard quotient accounts for one-third of the hazard index, this over-prediction is substantial. ATSDR then compounds this problem by repeating mistakes already detailed in the chromium and hydrogen sulfide sections above. Specifically, ATSDR assumed that total chromium measurements were entirely composed of hexavalent chromium. Had ATSDR taken the conservative assumption that CrVI comprises only 1.25% of the total chromium measured (USEPA 2015), the chromium hazard quotient would be reduced from 0.34 to 0.004, which would preclude its inclusion in calculation of the hazard index. Also, the maximum mean hydrogen sulfide concentration used in calculation of the hazard quotient only represented three months (October to December) in 1999 at the Huisache monitoring site. Revising these hazard quotients would certainly drop the respiratory hazard index below 1.0, suggesting there is no added risk to the respiratory system due to this mixture of chemicals.

The long-term non-carcinogenic evaluation for the neurological system suffers from the same mistakes and over-prediction. In fact, none of the chemicals included in the hazard index calculation have a CV related to neurological effects. Hydrogen sulfide (again, the three-month mean from the Huisache monitoring site) accounts for over half of the hazard index, followed by benzene, neither of which are known to cause neurological effects after long-term exposure to ambient levels (e.g., Srbova et al. (1950) provides a free-standing NOAEL of 110 ppm for central nervous system effects for a 2-hour human benzene exposure; no long-term exposure would be this high).

In the evaluation of mixtures of carcinogens, ATSDR again failed to consider the full duration necessary to evaluate excess *lifetime* cancer risk. ATSDR calculated the mean concentration of the putative carcinogen for each year of data, and then used the year with the highest mean concentration to calculate cancer risk. For the risks to be as high

as those quantified in the draft PHA, the population would have to be exposed to the highest concentration measured over the 30-year study period for 70 years. This exposure scenario simply is impossible, as concentrations have varied and in most cases decreased just in the study period alone. A slightly more accurate method would be to use the mean for all of the monitored years, although this would still have to be extrapolated out to 70 years.

Finally, out of ATSDR's list of 19 carcinogenic or possibly carcinogenic chemicals, six chemicals were detected less than 20% of the time. ATSDR likely overestimated the risk from these chemicals by assuming that the 95th percentile of the measured concentration of these chemicals was the actual mean concentration, which again would have to be unreasonably extrapolated out to 70 years. These six chemicals comprised almost 40% of the calculated risk in the cancer chemical mixture risk estimate. Twenty-six percent (26%) of the calculated risk came from 1,2-dibromoethane, a chemical that was detected in less than 1% of samples. Given these unrealistic assumptions and known over-estimates, it is shocking that the conclusion presented in both the draft PHA and the brochure is that air quality contributes to an increased cancer risk in this area. At a minimum, ATSDR needs to fully qualify their statements to fully disclose the underlying wholly unreasonable assumptions and limits of their evaluation to the public.

TCEQ Comparison Values

As stated previously, ATSDR misrepresents many of the TCEQ CVs used in the draft PHA. Tables 1 and 2 below provide the health and odor CVs, respectively, presented in the draft PHA along with the correct value. Please note that the TCEQ's Guidelines to Develop Toxicity Factors were revised in 2015. As part of this revision, TCEQ changed its approach to deriving odor-based CVs (TCEQ 2015b). Many of the revisions in Table 2 are due to changes made to TCEQ's methodology. ATSDR should revise their evaluation to use the correct CVs.

In addition to the published air monitoring comparison values used in this analysis, ATSDR could also consider using TCEQ effect screening levels (ESLs) when another value is not available. Interim ESLs are derived primarily for air permitting purposes, but can also be used as a screening value for air monitoring data. Current interim ESLs are provided in Table 1 for consideration.

Table 1. Corrected health-based TCEQ short- and long-term comparison values.

Chemical	Short-term CV Presented in the Draft PHA	Correct Short-Term CV	Short-term CV Description	Long-term CV Presented in the Draft PHA	Correct Long-term CV	Long-term CV Description
1,2,3-Trimethylbenzene	250 ppb	3,000 ppb	AMCV (final)	25 ppb	37 ppb	AMCV (final)
1,2,4-Trimethylbenzene	250 ppb	3,000 ppb	AMCV (final)	NA	NA	NA
1,2-Dichloroethane	40 ppb	550 ppb	AMCV (final)	NA	NA	NA
1,3,5-Trimethylbenzene	250 ppb	3,000 ppb	AMCV (final)	25 ppb	37 ppb	AMCV (final)
1-Butanol	--	200 ppb	ESL (interim)	--	20 ppb	ESL (interim)
1-Butene	50,000 ppb	27,000 ppb	AMCV (final)	--	2,300 ppb	AMCV (final)
1-Pentene	2,600 ppb	12,000 ppb	AMCV (final)	--	560 ppb	AMCV (final)
2-Methyl-2-Butene	2,600 ppb	12,000 ppb	AMCV (final)	--	560 ppb	AMCV (final)
Butyl Acetate	NA	NA	NA	130 ppb	990 ppb	AMCV (final)
Butyraldehyde	250 ppb	3,800 ppb	AMCV (final)	25 ppb	34 ppb	AMCV (final)
Chloroprene	--	10 ppb	ESL (interim)	NA	NA	NA
Cyclopentane	1,200 ppb	5,900 ppb	AMCV (interim)	120 ppb	590 ppb	AMCV (interim)
Isobutane	NA	NA	NA	2,400 ppb	10,000 ppb	AMCV (final)
Trichlorofluoromethane	5,000 ppb	10,000 ppb	AMCV (interim)	500 ppb	1,000 ppb	AMCV (interim)
c-2-Butene	NA	NA	NA	--	690 ppb	AMCV (final)

Chemical	Short-term CV Presented in the Draft PHA	Correct Short-Term CV	Short-term CV Description	Long-term CV Presented in the Draft PHA	Correct Long-term CV	Long-term CV Description
c-2-Pentene	2,600 ppb	12,000 ppb	AMCV (final)	--	560 ppb	AMCV (final)
n-Butane	NA	NA	NA	2,400 ppb	10,000 ppb	AMCV (final)
Dodecane	--	500 ppb	ESL (interim)	--	50 ppb	ESL (interim)
t-2-Butene	NA	NA	NA	--	690 ppb	AMCV (final)
t-2-Pentene	2,600 ppb	12,000 ppb	AMCV (final)	--	560 ppb	AMCV (final)
Naphthalene	95 ppb	100 ppb	AMCV (interim)	NA	NA	NA
Chlorine	--	15 µg/m ³	ESL (interim)	--	1.5 µg/m ³	ESL (interim)
Chromium (hexavalent)	0.1 µg/m ³	1.3 µg/m ³	AMCV (final)	NA	NA	NA
Vanadium	0.5 µg/m ³	20 µg/m ³	AMCV (interim)	0.05 µg/m ³	2 µg/m ³	AMCV (interim)
Acenaphthene	1,000 ng/m ³	100,000 ng/m ³	AMCV (interim)	100 ng/m ³	10,000 ng/m ³	AMCV (interim)
Acenaphthylene	1,000 ng/m ³	100,000 ng/m ³	AMCV (interim)	100 ng/m ³	10,000 ng/m ³	AMCV (interim)
Anthracene	500 ng/m ³	1,000 ng/m ³	AMCV (interim)	50 ng/m ³	100 ng/m ³	AMCV (interim)
Phenanthrene	500 ng/m ³	8,000 ng/m ³	AMCV (interim)	50 ng/m ³	800 ng/m ³	AMCV (interim)
2,5-Dimethyl-Benzaldehyde	20 ppb	16 ppb	AMCV (interim)	2 ppb	1.6 ppb	AMCV (interim)
Acetone	2,500 ppb	11,000 ppb	AMCV (final)	250 ppb	6,700 ppb	AMCV (final)
Benzaldehyde	20 ppb	21 ppb	AMCV (interim)	2 ppb	2.1 ppb	AMCV (interim)

Chemical	Short-term CV Presented in the Draft PHA	Correct Short-Term CV	Short-term CV Description	Long-term CV Presented in the Draft PHA	Correct Long-term CV	Long-term CV Description
Butyraldehyde	250 ppb	3,800 ppb	AMCV (final)	25 ppb	34 ppb	AMCV (final)
Crotonaldehyde	3 ppb	10 ppb	AMCV (final)	0.3 ppb	2.8 ppb	AMCV (final)
Furfural	--	20 ppb	ESL (interim)	NA	NA	NA
m-Tolualdehyde	21 ppb	18 ppb	AMCV (interim)	2.1 ppb	1.8 ppb	AMCV (interim)
o-Tolualdehyde	21 ppb	18 ppb	AMCV (interim)	2.1 ppb	1.8 ppb	AMCV (interim)
Propionaldehyde	200 ppb	740 ppb	AMCV (final)	NA	NA	NA
p-Tolualdehyde	21 ppb	18 ppb	AMCV (interim)	2.1 ppb	1.8 ppb	AMCV (interim)

-- -- information was left blank in the draft PHA

µg/m³ – micrograms per cubic meter

AMCV – air monitoring comparison value

CV – comparison value

ESL – effects screening level

NA – not applicable; CV in PHA was not a TCEQ value or TCEQ value was correct

PHA – public health assessment

ppb – parts per billion

TCEQ – Texas Commission on Environmental Quality

Table 2. Corrected TCEQ odor comparison values.

Chemical	TCEQ Odor Value Presented in the Draft PHA (ppb)	Correct TCEQ Odor Value (ppb)
1,1,1-Trichloroethane	380,000	(no value)
1,1,2,2-Tetrachloroethane	7,300	(no value)
1,2,4-Trimethylbenzene	140	(no value)
1,2-Dichloroethane	6,000	(no value)
1,2-Dichloropropane	250	(no value)
1-Butanol	--	300
1-Butene	360	(no value)
1-Hexene and 2-Methyl-1-Pentene	140	(no value)
2,2,4-Trimethylpentane	670	(no value)
2,3-Dimethylbutane	420	(no value)
2,3-Dimethylpentane	4,500	(no value)
2,4-Dimethylpentane	940	(no value)
2,-Butanone	440	(no value)
2-Methyl-1-Pentene	140	(no value)
2-Methylheptane	110	(no value)
2-Methylhexane	420	(no value)
2-Methylpentane	7,000	(no value)
3-Methyl-1-Butene	250	100
3-Methylheptane	1,500	(no value)
3-Methylhexane	840	(no value)
3-Methylpentane	8,900	(no value)
3-Pentanone	850	(no value)
4-Methyl-1-Pentene	140	(no value)
Benzene	2,700	(no value)

Chemical	TCEQ Odor Value Presented in the Draft PHA (ppb)	Correct TCEQ Odor Value (ppb)
Butyl Acetate	45	(no value)
Butyraldehyde	4.7	9.2
Carbon Tetrachloride	4,600	(no value)
Chlorobenzene	1,300	(no value)
Chloroform	3,800	(no value)
Cyclohexane	2,500	(no value)
Ethyl Acetate	390	870
Ethylbenzene	170	(no value)
Ethylene	270,000	(no value)
Isopentane	1,300	(no value)
Isopropylbenzene	48	130
Methyl Butyl Ketone	24	(no value)
Methyl Isobutyl Ketone	170	(no value)
Methyl t-Butyl Ether	130	170
Methylcyclohexane	150	(no value)
Methylcyclopentane	1,700	(no value)
Methylene Chloride	160,000	(no value)
Propane	1,500,000	(no value)
Propylene	13,000	(no value)
Tetrachloroethylene	770	(no value)
Toluene	920	(no value)
Trichloroethylene	3,900	(no value)
Trichlorofluoromethane	5,000	(no value)
a-Pinene	18	(no value)
b-Pinene	33	(no value)
c-2-Butene	2,100	(no value)

Chemical	TCEQ Odor Value Presented in the Draft PHA (ppb)	Correct TCEQ Odor Value (ppb)
c-2-Hexene	140	(no value)
Isobutyraldehyde	47	140
m-Diethylbenzene	70	(no value)
m-Ethyltoluene	18	(no value)
n-Butane	1,200,000	(no value)
n-Decane	620	(no value)
n-Heptane	670	(no value)
n-Hexane	1,500	(no value)
n-Octane	1,700	(no value)
n-Pentane	1,400	(no value)
n-Propyl Acetate	240	(no value)
n-Propylbenzene	48	(no value)
n-Undecane	870	(no value)
o-Ethyltoluene	74	(no value)
o-Xylene	380	(no value)
p-Diethylbenzene	70	(no value)
p-Ethyltoluene	8.1	(no value)
p-Xylene and m-Xylene	80	(no value)
t-2-Butene	2,100	(no value)
t-2-Hexene	140	(no value)
Naphthalene	38	85
Acetaldehyde	8.5	67
Acetone	42,000	(no value)
Acrolein	3.6	23
Crotonaldehyde	23	63
Heptaldehyde	50	30

Chemical	TCEQ Odor Value Presented in the Draft PHA (ppb)	Correct TCEQ Odor Value (ppb)
Hexaldehyde	20	81
Methacrolein	5.7	8.5
Methyl ethyl ketone	440	(no value)
Propionaldehyde	9	40
Valeraldehyde	30	28

-- -- information was left blank in the draft PHA
 PHA – public health assessment
 ppb – parts per billion
 TCEQ – Texas Commission on Environmental Quality

Health Outcome Data Evaluation

ATSDR did not represent or consider all health outcome data in its evaluation. Instead, ATSDR only discussed outcome data exhibiting an increasing illness trend. The omission of the full context of the data is misleading to the public.

Respiratory Outcomes – Asthma

While the draft PHA (e.g., p. 53) indicates that ATSDR reviewed available, relevant health outcome data for indications of increased illness in the defined study area, the document fails to capture decreasing trends and tends to only exemplify yearly rates that are higher than others thus exacerbating public health conclusions. In addition, although the draft ATSDR document is dated August 2016, ATSDR relied on 2008 and 2009 asthma hospitalization rates (p. 374). Had more recent, available data been analyzed, the assessment could have captured the decreasing asthma hospitalization trends in both Nueces and San Patricio Counties (Figure 1).

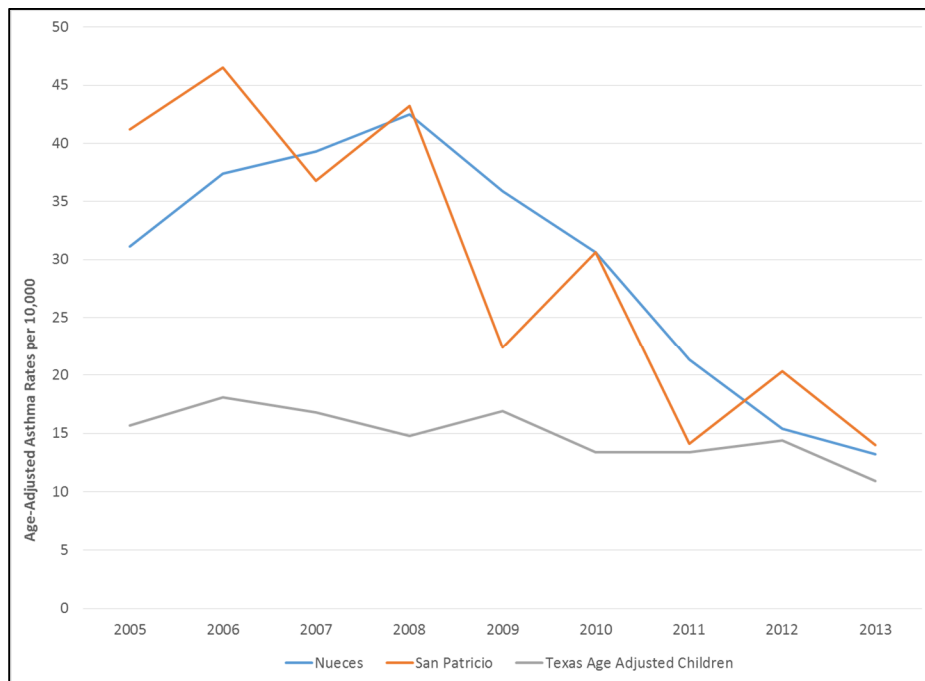


Figure 1. Asthma hospital discharge rates for children ages 0-17 years, 2005-2013. (Source: Texas Department of State Health Services, Texas Health Care Information Collection, Inpatient Hospital Discharge Public Use Data)

Use of Hospitalizations to Quantify Asthma Burden

Asthma is a complex disease that is difficult to measure. As reported by the Texas Asthma Control Program (TACP) for the Texas Department of State Health Services, the burden of asthma disproportionately affects people with certain demographic characteristics, socioeconomic status, and in particular geographic locations (TACP 2014). To understand the entire burden of asthma in a community, all asthma indicators should be considered. Hospitalization is one of the most severe outcomes of asthma;

thus, asthma hospitalization data represents the more severe cases of asthma in a given population. In addition, hospitalization rates are directly affected by demographic characteristics of the population (TACP 2014). Further, as reported by the Center for Disease Control and Prevention (CDC), the economy and the health care system also greatly impact this asthma indicator. Using asthma hospitalization data as a population based-measure has limitations—the data can be misleading, as they are affected by changes in prevalence. Because one person can have multiple hospitalizations for asthma in a specified time period, this indicator of asthma “describes rate of events, not rate of persons hospitalized” (CDC 2015). None of these limitations were discussed in presentation of conclusions in the draft PHA or the brochure.

As reported by the CDC, reducing hospitalizations due to asthma is theoretically a function of better care and self-management knowledge. The majority of the problems associated with asthma are preventable if asthma is managed (CDC 2015). Quality improvement initiatives, such as the Coastal Bend Asthma Initiative (CBAI), can target subgroups that experience disparities to aid in reducing asthma hospitalizations (AHRQ 2014). As mentioned on page 376 in the draft PHA, in 2011, “TACP provided asthma-initiative funding to Driscoll Children’s Health Plan (DCHP). This financial assistance helped fund a series of meetings that stimulated interest and participation in Corpus Christi’s previously dormant CBAI. The meetings resulted in establishment of a plan for coordinated presentations and community events throughout Asthma Awareness Month (May 2011). Since Asthma Awareness Month, interest in participating with CBAI has increased dramatically.”

A plausible reason for higher asthma hospitalizations rates among children in Nueces and San Patricio counties than the overall Texas rate from 2005-2008 was not necessarily the alluded issue of living near Refinery Row, but more so a function of the lack of self-management knowledge. As shown in Figure 1, asthma hospital discharge rates have decreased dramatically since 2005-2009, the reported time frame in the draft PHA. Failure to include this information in the ATSDR assessment is inappropriate and misleading to public.

National Ambient Air Quality Standards

In evaluating the potential for public exposure to outdoor air quality, the draft PHA is also misleading when stating,

“To decrease preventable asthma and reduce the severity of asthma symptoms, TACP focuses on both indoor and outdoor air quality. The U.S. Environmental Protection Agency (U.S. EPA) set NAAQs for six “criteria pollutants” considered harmful to public health. The criteria pollutants include ground-level ozone, particulate matter, lead, nitrogen dioxide, sulfur dioxide, and carbon monoxide. Some of these outdoor, criteria pollutants are known asthma triggers. **As recently as March 2006, the Corpus Christi metropolitan area was close to noncompliance with federal standards for at least one such outdoor criteria pollutant.** Successful asthma management requires that people identify outdoor and indoor asthma triggers in the environment so they can control, avoid, or eliminate them.”

The NAAQS standards developed by the USEPA are set at levels that are adequate to protect public health, including protecting the health of "sensitive" populations such as asthmatics, children, and the elderly. The Corpus Christi metropolitan area has always attained all applicable NAAQS, which USEPA would say provides adequate protection for even sensitive members of the population. Suggesting that the Corpus Christi metropolitan area's proximity to the NAAQS may trigger asthma is presumptuous and inappropriate for this assessment.

Birth Defects

The TCEQ agrees that the ATSDR birth defects analysis is limited and may be misleading to the general public since the assessment is based off of residential proximity and not maternal exposure. In addition to the limitations listed on pages 379 and 380 of the draft PHA, another limitation of the analysis is the lack of spatial specificity. It is unclear how ATSDR's Geographic Research, Analysis, and Services Program "determined geographic proximity areas surrounding Refinery Row." Further, maternal residential history is not considered and it is assumed that maternal address at time of delivery was the same as maternal address for the duration of the pregnancy. This limitation is particularly important considering that the critical window of exposure for formation of birth defects is during the first trimester. Again, the significant limitations of this very limited analysis were not included in ATSDR's presentation of the conclusions in the draft PHA or the brochure, which is grossly misleading to the public.

Ambient Monitoring Data

Substitutions for Non-Detects

ATSDR needs to reconsider their methods for dealing with data reported as non-detect. The draft PHA indicates that the reporting limit was substituted for chemicals that were not detected, which introduces an unknown bias into summary statistics of the data. Particularly with pollutants that are rarely detected, such as 1,2-dibromoethane, it is not possible to know how much the estimated mean based non-detect substitutions deviates from the actual mean. Therefore, because the frequency of detected concentrations is so low, there can be no confidence that any mean or other calculation is representative of the actual distribution of the data. Any subsequent conclusions based on these summary statistics are highly suspect. If ATSDR is determined to evaluate the data anyway, it should provide scientific justification supporting the censoring method and better qualify the results of their evaluation of annual averages based on censored data to clearly articulate the limited confidence in the values.

ATSDR also wrongly quotes the TCEQ's typical substitution method. Appendix G indicates that ATSDR used 0.01 ppb as the reporting limit for all chemicals and that data reported as ND were substituted with 0.01 ppb, which is claimed to be most consistent with TCEQ's method. However, this is a misrepresentation of both the reporting limit and TCEQ's method. The reporting limit changes based on the analytical method, so the standard use of 0.01 ppb for all chemicals is inappropriate. In addition, the TCEQ's standard process is to substitute 1/2 the method reporting limit when concentrations are reported as ND and at least 75 percent of possible samples had

concentrations above the reporting limit. In some cases, a method reporting limit is not available, in which case TCEQ uses 1/2 the method detection limit.

Method Detection and Reporting Limits

ATSDR should correct the description of the conversion of parts per billion carbon (ppb-C) to parts per billion by volume (ppbv). On page 310 of the health assessment, ATSDR indicates that detection limits provided in ppb-C were multiplied by the number of carbon atoms in each VOC molecule to derive a ppbv concentration. This method is incorrect. The ppb-C values should be *divided* by the number of carbon atoms.

Monitoring Trends

ATSDR should make a greater effort to put any elevated pollutant concentrations into context with trends over time, rather than providing the general statements it makes about the risk of exposure to the maximum concentrations of air pollutants. Monitored concentrations fluctuate over time and space and, in the vast majority of cases, have decreased since the beginning of the ATSDR's study period. Therefore, potential exposures in the 1990s are not anything like the potential exposures of 2010 or even 2016. ATSDR's failure to account for these differences has contributed to the unnecessarily inflammatory and incorrect conclusions in the draft PHA. Trends specific to benzene, PM, and sulfur dioxide are provided below as examples.

Benzene

The timeframe covered by the draft PHA also includes the period the Refinery Row area was on the TCEQ's Air Pollutant Watch List (APWL) because of elevated annual benzene concentrations at the Corpus Christi Huisache monitor. TCEQ's resources in a number of program areas were gathered to prioritize the reduction of benzene in this area. Due to this work and collaborative efforts with area industrial facilities, benzene concentrations decreased 71% from 1997 to 2010, when the area was removed from the APWL (TCEQ 2010). Annual average benzene concentrations have continued to decline at stationary monitors across Corpus Christi since that time. As highlighted in Figure 2, ATSDR's method of using the maximum mean for the area fails to recognize the significant reductions in ambient benzene concentrations that have occurred over their study period and misrepresents true risk to the public.

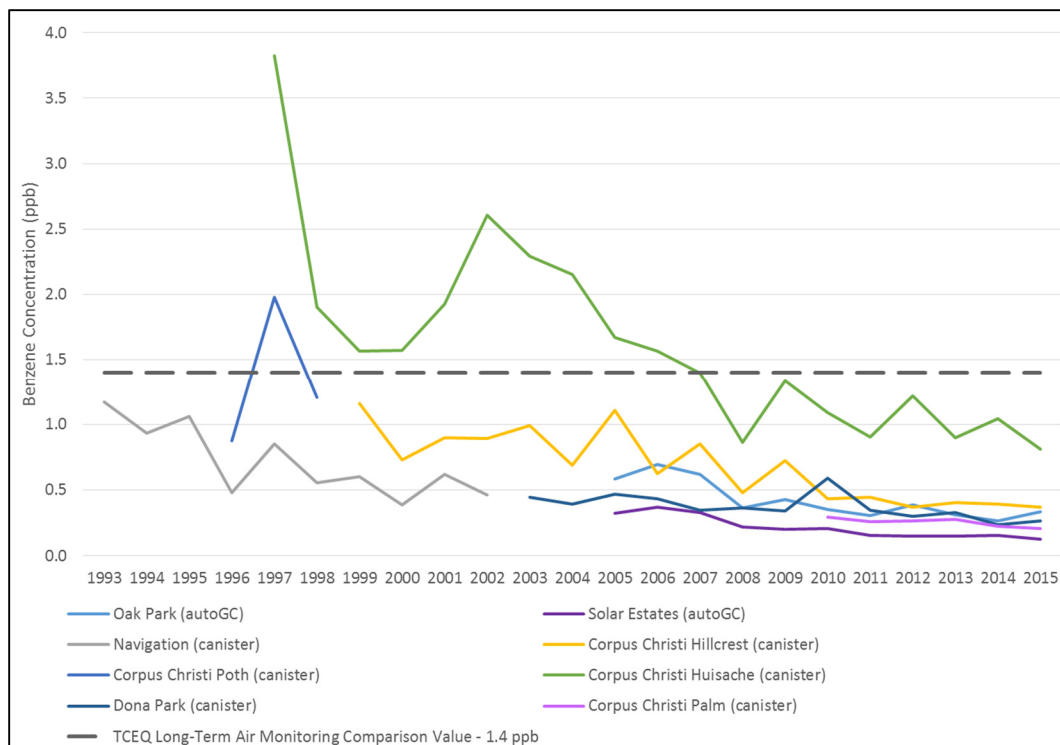


Figure 2. Annual average benzene concentrations at Corpus Christi area monitors, 1993-2015. (Source: Texas Air Monitoring Information System (TAMIS))

Particulate Matter

As stated previously, the Corpus Christi area has always been in attainment of the applicable PM NAAQS. Figures 3 and 4 show design value trends for the two regulatory PM monitors in the Corpus Christi area. Note that the design values in these figures represent the true comparison to the NAAQS and that the PM NAAQS has changed over the draft PHA study period. Prior to 2006, there was not a PM_{2.5}NAAQS. In 2006, the USEPA promulgated 24-hour primary PM_{2.5} NAAQS of 35 µg/m³ (calculated as the 98th percentile averaged over three years). Also in 2006, the annual PM_{2.5} NAAQS of 15.0 µg/m³ was promulgated (calculated as the annual arithmetic mean averaged over three years), though this was decreased in 2012 to an annual NAAQS of 12.0 µg/m³ (also calculated as the annual arithmetic mean averaged over three years).

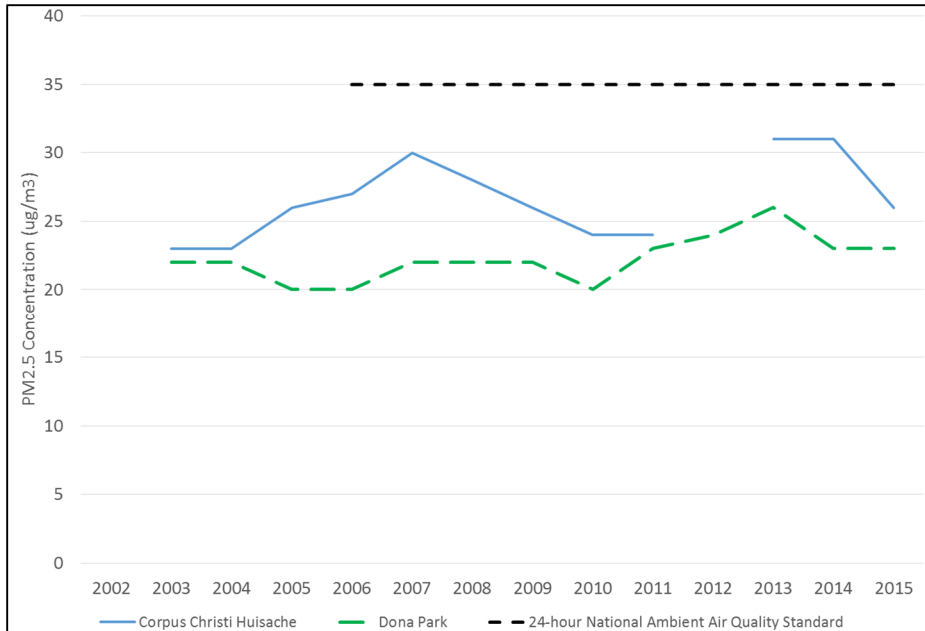


Figure 3. 24-hour particulate matter of 2.5 micrometers or less in diameter (PM_{2.5}) design value trends for Corpus Christi area monitors, 2002-2015. (Source: USEPA Air Quality System)

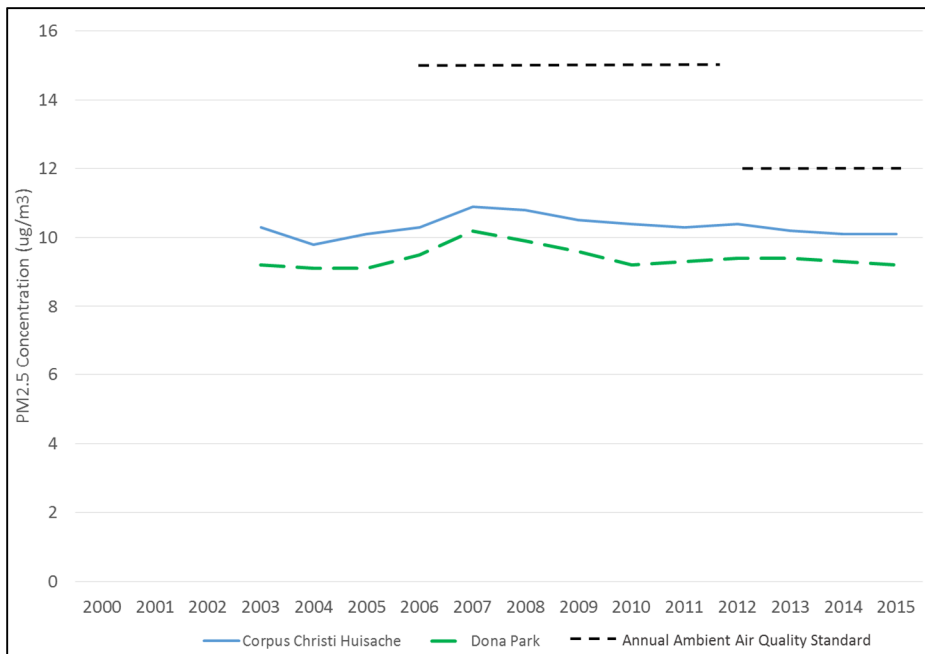


Figure 4. Annual particulate matter of 2.5 micrometers or less in diameter (PM_{2.5}) design values for monitors in the Corpus Christi area, 2000-2015. (Source: USEPA Air Quality System)

Sulfur Dioxide

Monitors in the Corpus Christi area have always attained the applicable sulfur dioxide NAAQS. Prior to 2010, the primary NAAQS consisted of a 24-hour standard of 0.14 ppm

(140 ppb) and an annual standard of 0.03 ppm (30 ppb). On June 2, 2010, USEPA revised the primary sulfur dioxide standard by establishing a new 1-hour standard at a level of 75 ppb. The 24-hour and annual standards were revoked. As shown in Figure 5, one-hour sulfur dioxide design values have steadily decreased since 2003 and are well below the current NAAQS.

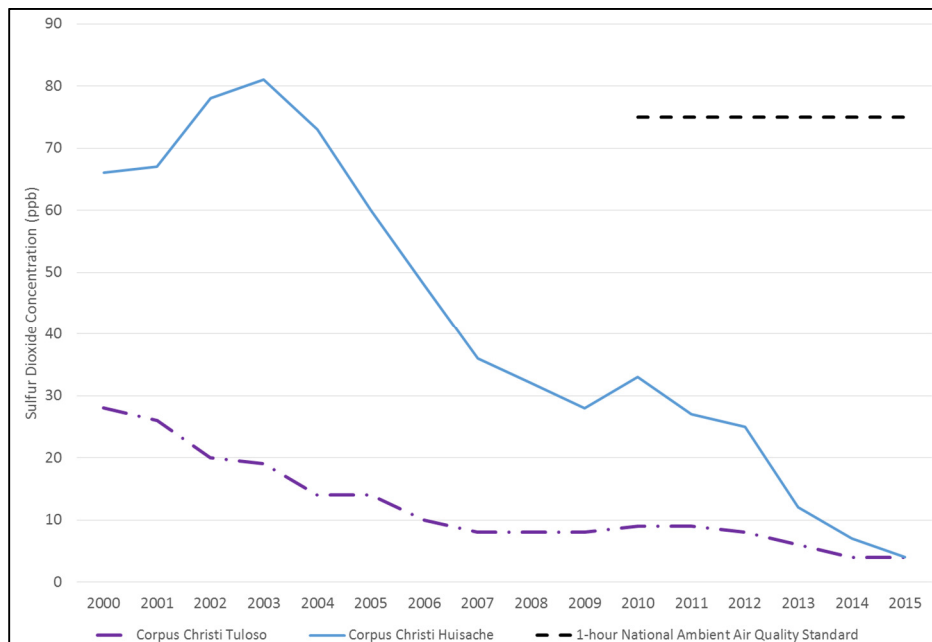


Figure 5. 1-hour sulfur dioxide design values in the Corpus Christi area, 2000-2015. (Source: USEPA Air Quality System)

In addition to the regulatory monitors, five of the six non-regulatory monitors have measured sulfur dioxide concentrations below the 1-hour NAAQS. The J.I. Hailey site, a non-regulatory site operated by the University of Texas (UT), did measure levels above the new NAAQS. As stated previously, this site is located north of the ship channel and more than 1.5 miles from the nearest residential area; therefore, it is not expected to provide data representative of public exposure.

Over a three year period from 2010 through 2013 the TCEQ investigated the potential sources of sulfur dioxide emissions impacting the J.I. Hailey site. This included collaboration with the Port of Corpus Christi and port industries to collect and report field observations and operational information to the TCEQ in response to elevated sulfur dioxide measurements. Other project participants included USEPA Enforcement, UT and its Air Quality Project Advisory Board, as well as other local stakeholders to include the Regional Health Awareness Board (RHAB).

These investigative efforts included the installation of an additional TCEQ sulfur dioxide monitor located to the south of J.I. Hailey and the ship channel but north of port industries for a 2-year period. The use of a TCEQ developed email based alert system triggered by near real-time monitoring allowed for rapid response and reporting. Ultimately, the installation of this monitor, combined with the information gathered and reported by the port and port industries, led to the finding that emissions from international ships operating and docked in the Corpus Christi ship channel were

the primary contributors to elevated sulfur dioxide concentrations at the J.I. Hailey site. Furthermore, the monitoring data demonstrated that declines in sulfur dioxide values directly coincided with the implementation of fuel regulations that significantly lowered the sulfur content of fuels in large ocean-going vessels. Finally, the sulfur dioxide design value at the J.I. Hailey site has significantly improved and measured levels below the sulfur dioxide NAAQS from 2013 to present.

Data Quality

ATSDR should clarify what the results of their comparison study actually represent in the final PHA. ATSDR's data quality evaluation does not actually assess data quality; it assesses data comparability between monitors. TCEQ uses a variety of automated and manual quality control (QC) checks to evaluate data quality (e.g., accuracy, bias, and precision). These QC measurements are performed annually, quarterly, bi-quarterly, monthly, weekly, daily, and even per analysis depending on the complexity of the check and method requirements. Data and the corresponding QC data are reviewed, along with relevant sampling information, to ensure that the data meets the outlined measurement quality objectives. Data that does not meet the quality objectives are invalidated or qualified.

Monitor Name

It would be helpful to the public if a footnote were added to the Old Galveston Road monitor clarifying that the monitor was located in Corpus Christi near the current Old Robstown Road.

Emissions Inventory

ATSDR needs to provide important clarifications on the use of Toxics Release Inventory data related to national rankings, emissions, and reporting sources. Specifically, on page 29, the report cites TRI trend data indicating Nueces County "steadily increas[ed] from 9th to 4th" in the nation in terms of total benzene air releases from 2000 to 2010. While the ranking data is accurate, it leads the reader to believe benzene air releases have also increased during this time period, which is inaccurate. Nueces County benzene air releases decreased by 24% from 2000 to 2010, and have continued to decrease.

This bullet point (and related statistics) should be rephrased to clarify air releases of benzene are decreasing in Nueces County, e.g., "Although benzene air releases decreased by 24% from 2000 to 2010 in Nueces County, the county's ranking in terms of total benzene air releases moved from 9th to 4th in the US during this same time period."

Second, although the report acknowledges the limitations of TRI data, including the fact that only certain industries report to TRI, the report does not clearly acknowledge the potential impact these omissions have on the data. Specifically, bulk storage terminals that store specialty liquids and natural gas processing plants are two potentially significant sources of benzene air emissions that are not required to report to TRI. For example, in 2005, the largest point source of benzene emissions (reporting to the TCEQ point source emissions inventory) in Texas was a bulk storage terminal for specialty liquids, an industry not required to report to TRI. Similarly, in 2010, two of the five

largest point sources of benzene (reporting to the TCEQ point source emissions inventory) in Texas were natural gas processing plants, an industry not currently required to report to TRI. The report needs to clarify that the statistics omit potentially significant sources of benzene air emissions.

Air Pollution Control

The TCEQ manages air quality in the state of Texas by regulating the release of air contaminants under authority in the Texas Clean Air Act (TCAA), codified in Chapter 382 of the Texas Health and Safety Code (THSC). This is implemented through adoption of rules and issuance of permits. The TCEQ also implements the requirements of the Federal Clean Air Act (FCAA) and Code of Federal Regulations (CFR) through rules and permits issued in Texas.

In its recommendations, ATSDR erroneously assumed that air authorizations for Corpus Christi area facilities do not include a Best Available Control Technology (BACT) review. BACT review is a statutory requirement and has been a part of permit application review for major sources subject to New Source Review (NSR) for decades.

The TCAA and TCEQ rules require an evaluation of air quality permit applications to determine whether adverse effects to public health, general welfare, or physical property are expected to result from a facility's proposed emissions. Applications for projects subject to a BACT and health effects review are those with new and modified facilities that may emit air contaminants. The burden is on the applicant to demonstrate that the draft permit, if issued, would be protective of human health and the environment. Therefore, the applicant must fully document the BACT and health effects review. As part of the technical review of applications for new or amended NSR permits, the permit reviewer audits the sources of air contaminants at the proposed facility and evaluates air pollution control requirements to assure that the facility will be using BACT for the sources and types of contaminants emitted.

BACT is based on control measures that are designed to minimize the level of emissions from specific sources at a facility. Specifically, BACT is defined as "an air pollution control method for a new or modified facility that through experience and research, has proven to be operational, obtainable, and capable of reducing or eliminating emissions from the facility, and is considered technically practical and economically reasonable for the facility. The emissions reduction can be achieved through technology such as the use of add-on control equipment or by enforceable changes in production processes, systems, methods, or work practice" (30 TAC § 116.10(1)). BACT may be expressed in terms of an emissions limit or as a performance criteria. Applying BACT results in requiring technology that best controls air emissions with consideration given to the technical practicability and economic reasonableness of reducing or eliminating emissions.

In addition to BACT evaluation, applicants are required to provide the necessary documentation to show that emissions from all new facilities and modifications will be protective of potential health and environmental impacts through compliance with the NAAQS and TCEQ effects screening levels. The TCEQ permit reviewer's BACT evaluation and final recommendation provide a record that demonstrates that the operation of a proposed facility or related source will not cause or contribute to a

nuisance, will be protective of human health and the environment, and will comply with all applicable state and federal statutes and regulations and the intent of the TCAA.

References

- Agency for Healthcare Research and Quality (AHRQ). 2014. Asthma Care Quality Improvement. Available at: <http://www.ahrq.gov/professionals/quality-patient-safety/quality-resources/tools/asthmaqual/asthmacare/module4.html>
- Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for benzene.
- ATSDR. 2015. Addendum to the toxicological profile for benzene. Agency for Toxic Substances and Disease Registry (ATSDR).
- Centers for Disease Control and Prevention (CDC). 2015. Indicator Definitions – Asthma. Available at: <http://www.cdc.gov/edi/definitions/asthma.html>
- Flury F. 1928. [II. Toxicities in modern industry. IIa. Pharmacological-toxicological aspects of intoxicants in modern industry.] *Arch Exp Pathol Pharmacol* 138:65-82.
- Haney J, Erraguntla N, Sielken R, et al. 2014. Development of an Inhalation Unit Risk Factor for Hexavalent Chromium. *Regul. Toxicol. Pharmacol.* 68:201-211.
- Haney J. 2016. Development of an inhalation unit risk factor for cadmium. *Regul. Toxicol. Pharmacol.* 77: 175-183.
- Jackson Laboratory. 2007. Hematology measurements involving C57BL/6J mice.
- Mancuso T. 1975. Consideration of chromium as an industrial carcinogen. In: Hutchinson, T.C. (Ed.), *Proceedings of the international conference on heavy metals in the environment*, Toronto, Canada, Toronto Institute for Environmental Studies, Toronto.
- Midzenski MA, McDiarmid MA, Rithman N. 1992. Acute high dose exposure to benzene in shipyard workers. *Am J Ind Med* 22:553-565.
- Park R, Stayner L, Petersen M, et al. 2012. Cadmium and lung mortality accounting for simultaneous arsenic exposure. *Occup Environ Med* 69:303-309.
- Rozen MG, Snyder CA, Albert RE. 1984. Depression in B- and T-lymphocyte mitogen-induced blastogenesis in mice exposed to low concentrations of benzene. *Toxicol Lett* 20:343-349.
- Sheppard D, Saisho A, Nadel JA, et al. 1981. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. *Am Rev Respir Dis* 123:486-491.
- Srbova J, Teisinger J, Skramovsky S. 1950. Absorption and elimination of inhaled benzene in man. *Arch Ind Hyg Occup Med* 2:1-8.
- Stayner L, Smith R, Thun M, et al. 1992. A dose-response analysis and quantitative assessment of lung cancer risk and occupational cadmium exposure. *Ann Epidemiol* 2(3):177-194.

- Texas Asthma Control Program (TACP). 2014. 2014 Texas Asthma Burden Report. Available at: <https://www.dshs.texas.gov/asthma/>
- Texas Commission on Environmental Quality (TCEQ). 2000a. November 9, 2000 Interoffice Memorandum from Vincent A. Leopold to Buddy Stanley, “Toxicological Evaluation of Air Sampling Results, Benzene and Other Volatile Organic Compounds (VOCs), Hydrocarbon Seep and Remediation Activities at Elementis Chromium Recovery Wells, Recovery Wells at Coastal Refining and Marketing-East and West Plants, Corpus Christi, Nueces County, July 31, 2000.”
- TCEQ. 2000b. September 7, 2000 Interoffice Memorandum from Vincent A. Leopold to Buddy Stanley, “Toxicological Evaluation of Air Sampling Results, Benzene and Other Volatile Organic Compounds (VOCs), Hydrocarbon Seep at Elementis Chromium, and Amerada Hess Recovery Wells in Oak Park Neighborhood (Cenizo Street), Corpus Christi, Nueces County, May 31, 2000.”
- TCEQ. 2000c. March 31, 2000 Interoffice Memorandum from Vincent A. Leopold to Buddy Stanley, “Toxicological Evaluation of Ambient Air Sampling for Benzene and Other Volatile Organic Compounds (VOCs), Petroleum Hydrocarbon Seep, Elementis Chromium, Corpus Christi, Nueces County, March 1, 2000.”
- TCEQ. 2007. Development support document for benzene. TCEQ.
- TCEQ. 2010. Region 14 Air Pollutant Watch List removal document. Available at: <https://www.tceq.texas.gov/assets/public/implementation/tox/apwl/1402-removed-benzene-corpus.pdf>
- TCEQ. 2011a. Health effects evaluation of Part 1 of Phase II data for the Hillcrest Community Environmental Investigation.
- TCEQ. 2011b. Health effects evaluation of Part 2 of Phase II data for the Hillcrest Community Environmental Investigation.
- TCEQ. 2014. Development support document for hexavalent chromium (particulate compounds). TCEQ.
- TCEQ. 2015a. Guidelines to develop toxicity factors. RG-442: Texas Commission on Environmental Quality (TCEQ). Available at: <https://www.tceq.texas.gov/toxicology/esl/guidelines/about.html/#pp>
- TCEQ. 2015b. Approaches to derive odor-based values. Position paper: TCEQ. Available at: <https://www.tceq.texas.gov/assets/public/implementation/tox/dsd/positionpaper/odor.pdf>.
- TCEQ. 2016. Development support document for cadmium and cadmium compounds. TCEQ.
- Thun M, Schnorr T, Smith A, et al. 1985. Mortality among a cohort of U.S. cadmium production workers-an update. J Natl Cancer Inst 74:325-333.
- U.S. Environmental Protection Agency (USEPA). 1984. Health assessment document for chromium. Washington, DC: USEPA. EPA-600/8-83-014F.

- USEPA. 1985. Updated mutagenicity and carcinogenicity assessment of cadmium (addendum to the health assessment document for cadmium of May 1981). EPA-600/8-83-025F. USEPA, Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC.
- USEPA. 1998. Carcinogenic effects of benzene: an update. Washington, DC: USEPA. EPA600P97001F. PB99101420.
- USEPA. 1999. Toxicological review of cadmium and compounds in support of summary information on the integrated risk information system (IRIS), external review draft. USEPA, Office of Research and Development, National Center for Environmental Assessment, Washington, DC. USEPA. 1998. Carcinogenic effects of benzene: An update. Washington, DC: USEPA. EPA600P97001F.
- USEPA. 2002. Toxicological review of benzene (noncancer effects). Washington, DC: USEPA. EPA/635/R-02/001F.
- USEPA. 2010. Primary National Ambient Air Quality Standard for Sulfur Dioxide. 75 Federal Register 119 (June 22, 2010).
- USEPA. 2015. Technical Support Document, EPA's 2011 National-Scale Air Toxics Assessment. USEPA.